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Le développement de la dépression chez l'adolescent : état actuel des connaissances et analyse du rôle du vécu scolaire à titre de facteur de risque potentiel

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SOMMAIRE

Si les connaissances portant sur le développement de la dépression ont évolué au cours des dernières années, deux obstacles en limitent encore la portée : le manque d'intégration des connaissances et la méconnaissance du rôle du vécu scolaire. Cette thèse tente de repousser ces deux limites.

En regard de la première limite, une recension des études publiées au cours des 12 dernières années, et portant sur les antécédents psychosociaux du développement de la dépression chez l'enfant et l'adolescent, a été effectuée. Malgré le grand nombre d'études publiées au cours de cette période, seules 91 études présentaient des caractéristiques méthodologiques leur assurant des résultats valides. Pour chacun des antécédents psychologiques ou sociaux considérés, la présentation des bases théoriques est suivie d'une description des résultats des études retenues. Les principales conclusions de ces études sont ensuite exposées. À la suite de cette démarche, une synthèse des résultats obtenus, décrivant la dépression comme un problème biopsychosocial d'adaptation résultant de l'action combinée de multiples antécédents individuels et environnementaux en interaction les uns avec les autres, est proposée. En conclusion, trois défis majeurs sont identifiés en vue de guider les études futures dans ce domaine.

En rapport avec la seconde limite, une étude empirique a été complétée afin d'évaluer le rôle potentiel de différents aspects du vécu scolaire adolescent, à titre de facteurs de risque pour le développement de la dépression. À cette fin, les données du Projet Montréalais sur le Développement de la Dépression Adolescente ont été utilisées. Ce projet repose sur un suivi longitudinal en trois temps de 1167 adolescents, élèves en de première année du niveau secondaire. D'une part, les résultats obtenus ont indiqué que la majorité des variables considérées représentaient des facteurs de risque pour la

dépression, et que cet effet était indépendant du niveau antérieur de dépression des sujets. D'autre part, la prise en compte simultanée de ces variables dans des analyses multivariées a entraîné la disparition de la majorité des effets observés. Ces dernières analyses ont révélé que, parmi les principaux prédicteurs associés au développement de la dépression, la violence en milieu scolaire occupait une place importante. Enfin, des analyses additionnelles ont montré que plusieurs des variables exerçaient un effet plus important chez les filles que chez les garçons, et que l'effet de certaines variables variait en fonction du niveau antérieur de dépression manifesté par les sujets. La convergence de ces résultats avec les conclusions des études antérieures et leur utilité clinique ou préventive sont ensuite discutées.

Mots-clés: dépression, développement, recension, facteurs de risque et de protection, antécédents, vécu scolaire, longitudinal.

SUMMARY

Although scientific knowledge on depression development has evolved over the past decade, its impact is still limited by two obstacles: the lack of knowledge integration and the lack of attention devoted to the role of school life. This thesis attempts to attenuate these two limitations.

Concerning the first limitation, studies published over the past 12 years on the subject of psychosocial antecedents involved in child and adolescent depression development were reviewed. Although many studies were published during this period, only 91 of them presented methodological characteristics ensuring valid results and were included in this review. For each psychological or social antecedent considered, the theoretical bases are presented first, followed by a description of the results from the retained studies. Next, the main conclusions from these studies are reported, and an integrated synthesis of the results, in which depression is described as a biopsychological adaptation problem resulting from the combined action of multiple individual and environmental antecedents framework, is proposed. In conclusion, three major challenges are identified as a guide for future studies in this field.

Regarding the second limitation, an empirical study was conducted with the goal of evaluating the potential role of various aspects of adolescent school life as risk factors for depression development. To this end, data from the Montréal Adolescent Depression Development Project were used. This project was based on a three-measurement-point longitudinal follow-up of 1167 adolescents in seventh grade. The results obtained indicated that the majority of the included variables represented risk factors for depression and that their effects were independent of the subjects' previous levels of depression. However, the simultaneous consideration of these variables in multivariate analyses resulted in the disappearance of the majority of the observed

effects. These last analyses revealed that, among the main predictors associated with depression development, violence within the school environment was a major source of influence. Finally, additional analyses revealed that several of the variables exerted stronger effects in girls than in boys and that the effect of certain variables varied according to the subjects' previous levels of depression. The convergence of these results with the conclusions from previous studies and their clinical or preventive usefulness are then discussed.

Key words: depression, development, review, risk and protective factors, antecedents, school life, longitudinal.

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Chapitre I
Introduction

LA DÉPRESSION :
AMPLEUR ET DÉFINITION DU PHÉNOMÈNE

Dans le *Global Burden of Diseases Study*, l'Organisation Mondiale de la Santé révèle que la dépression représente actuellement la quatrième cause mondiale d'incapacité (Murray & Lopez, 1996 a, 1996 b). Des projections issues de cette étude indiquent aussi que la dépression devrait occuper le second rang de ce palmarès en 2020. Des analyses similaires ont d'ailleurs révélé que les coûts annuels de la dépression pour la société pouvaient atteindre 30.4 à 43.7 milliards de dollars aux Etats-Unis, et 3.39 milliards de livres sterling en Angleterre (Chisholm, 2001; Greenberg, Stiglin, Finkelstein, & Berndt, 1993). De surcroît, plusieurs auteurs mentionnent que la situation est probablement encore plus sérieuse que ne le laissent présager les conclusions des ces études, celles-ci s'appuyant sur des estimés prudents des taux réels de prévalence de la dépression et ne considérant pas toujours l'entièreté des conséquences associées (Üstün & Chatterji, 2001).

L'ampleur du phénomène

Plusieurs raisons permettent d'expliquer ces observations. Premièrement, la dépression représente l'un des problèmes de santé mentale les plus prévalents. Ainsi, diverses études révèlent que, chaque année, 1 % des enfants d'âge préscolaire, 1 à 4 % des enfants d'âge scolaire, 3 à 10 % des adolescents et 3 à 15 % des adultes souffriront de symptômes de dépression de sévérité suffisante pour mériter un diagnostic psychiatrique (Angold & Costello, 2001; Baron, 1993; Bebbington *et al.*, 1998; Fleming & Offord, 1990; Garber & Horowitz, 2002; Kashani, Holcomb, & Orvaschel, 1986; Kessler, 2002; Lewinsohn, Hops, Roberts, Seeley, & Andrews,

1993; Offord *et al.*, 1996; Weissman *et al.*, 1996). Au cours de leur vie, près de 21.3 % des femmes et 12.7 % des hommes seront atteints de dépression (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). Cette situation serait d'ailleurs en train de s'envenimer puisque de nombreuses études ont noté que les taux de prévalence semblent augmenter chez les individus nés entre 1935 et 1940 (Cross National Collaborative Group, 1992; Kessler *et al.*, 1994; Klerman & Weissman, 1989). Des résultats similaires ont été observés plus récemment chez les adolescents (Fombonne, 1998; Lewinsohn, Rohde, Seeley, & Fisher, 1993). D'ailleurs, les taux de prévalence à vie de dépression, observés dans des échantillons récents d'adolescents, sont généralement comparables à ceux observés chez l'adulte et reflètent les mêmes différences intersexes (Lewinsohn, Hops *et al.*, 1993; Hankin *et al.*, 1998).

Deuxièmement, il est généralement reconnu que la dépression représente un problème de santé mentale récurrent, parfois même chronique, qui tend à se développer au cours de l'adolescence (Angold & Costello, 2001; Boland & Keller, 2002; Fleming & Offord, 1990; Kessler, 2002; Lewinsohn, & Essau, 2002; NIMH/NIH Consensus Development Panel, 1985). En effet, les résultats de nombreuses études convergent pour démontrer que la majorité des adultes souffrant de dépression ont déjà souffert d'épisodes dépressifs au cours de leur adolescence (Bland, 1997; Newman *et al.*, 1996) et que les adolescents souffrant de dépression tendent à faire des rechutes au cours de leur vie adulte (Fombonne, Wostear, Cooper, Harrington, & Rutter, 2001; Harrington & Dubicka, 2001; Lewinsohn, Rohde, Klein, & Seeley, 1999), ce qui n'est pas nécessairement le cas des enfants dépressifs (Harrington, Rutter, & Fombonne, 1996). Il convient ici de préciser que, si les taux de prévalence observés chez les garçons et les filles apparaissent relativement faibles et similaires au cours de l'enfance, l'augmentation des taux de prévalence observés au cours de l'adolescence apparaît particulièrement marquée chez les filles. En effet, dès l'âge de 13 ans, les filles seraient deux fois plus nombreuses que les garçons à souffrir de dépression (e.g., Angold & Costello, 2001; Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Cole et al., 2002; Hankin et al., 1998; Kessler, 2003; Nolen-Hoeksema, & Girgus, 1994). Il est intéressant de noter que ces derniers résultats ont pu être reproduits dans

une variété d'études prospectives longitudinales dans lesquelles des schèmes analytiques variés ont été utilisés.

Troisièmement, en plus de présenter des taux élevés de comorbidité avec d'autres problèmes de santé mentale (Angold, Costello, & Erkanli, 1999; Compas, Connor, & Hinden, 1998), la dépression est souvent associée à un ensemble de conséquences néfastes qui couvrent l'ensemble des sphères de la vie des individus touchés (Bardone, Moffitt, Caspi, Dickson, & Silva, 1996; Glied & Pine, 2002; Ensinck *et al.*, 2002; Kessler, Foster, Saunders, & Stang, 1995; Hodgins, 1996; Quiroga, & Janosz, 2002; Schneider, Müller, & Philipp, 2001). Par exemple, les individus souffrant de dépression tendent à afficher de nombreux problèmes interpersonnels, une diminution du rendement au travail et une scolarisation incomplète ou insuffisante. De même, ces individus présentent aussi un risque plus élevé de devenir d'éventuels bénéficiaires de l'aide sociale ou intinérants et de mourir prématurément.

Que faire face à la dépression ?

Face à ses observations, plusieurs organisations nationales et internationales de santé, telles que l'Organisation Mondiale de la Santé (Dawson & Tylee, 2001) et l'Institut de Médecine Américain (Mrazek & Haggerty, 1994), ont lancé un appel visant à souligner l'urgence et l'importance d'en arriver à une plus grande concertation pour le développement de programmes efficaces de prévention de la dépression. De nombreux chercheurs ont d'ailleurs donné leur appui à cet appel en soulignant l'urgence, pour la recherche développementale, d'accorder une plus grande importance à l'élaboration de programmes de ce type (Harrington & Clark, 1998; Morin & Chalfoun, 2003, Marcotte, 2000; Muñoz, Le, Clarke, & Jaycox, 2002; Muñoz & Ying, 1993). Cependant, pour être efficaces, de tels programmes devraient s'appuyer sur une connaissance intégrée et approfondie des mécanismes responsables du développement de la dépression chez l'enfant et l'adolescent (Coie *et al.*, 1993; Kazdin, 1993; Mrazek & Haggerty, 1994).

Malheureusement, et malgré le fait que les connaissances scientifiques aient considérablement évolué au cours de la dernière décennie, certaines limites importantes font encore obstacle à l'atteinte d'un niveau suffisant de compréhension de ces mécanismes. Avant de présenter ces limites, il convient de clarifier la définition de certains termes utilisés tout au long de cette thèse et de proposer une définition claire et opérationnelle de la dépression.

Clarifications terminologiques

Dans cette thèse, les termes « facteurs de risque », « facteurs de protection », de même que « médiateurs » et « modérateurs » reviendront souvent.

Le terme « *facteur de risque* » fait référence aux « caractéristiques d'un individu ou de son environnement qui sont associées à une plus grande probabilité pour cet individu de développer certains problèmes d'adaptation » (Compas, Hinden, & Gerhardt, 1995, p.273).

Le terme « *facteur de protection* » renvoie à des caractéristiques individuelles ou environnementales qui « interagissent avec certains facteurs de risque de manière à réduire la probabilité que les individus exposés ne développent des problèmes d'adaptation, sans toutefois être reliés au développement de ces mêmes problèmes chez les individus non exposés » (Compas *et al.*, 1995, p. 273).

Un « *modérateur* » est une variable qui « modifie la relation qui existe entre deux variables, de manière à ce que l'impact du prédicteur ne soit pas le même à différents niveaux du modérateur » (Holmbeck, 1997, p. 599). Typiquement, les variables modératrices, de même que les facteurs de protection qui en sont un cas spécifique, sont identifiés par la présence d'effets d'interaction dans le cadre d'analyses statistiques (Baron & Kenny, 1986; Holmbeck, 1997, 2002).

Finalement, un « *médiateur* » est un « mécanisme par lequel une variable indépendante exerce son effet sur une variable dépendante » (Baron & Kenny, 1986, p. 1173). L'identification d'effets médiateurs vise donc à déterminer les mécanismes responsables ou explicatifs de l'action d'un prédicteur sur une variable donnée. Un effet de médiation est démontré lorsque : (a) un prédicteur, P, prédit significativement une variable dépendante, D; (b) P prédit significativement une variable potentiellement médiatrice, M; (c) M prédit significativement D; (d) la relation qui unit P et D est significativement réduite ou disparaît complètement après l'inclusion de M dans le modèle analytique (Baron & Kenny, 1986; Holmbeck, 1997, 2002).

Qu'est-ce que la dépression?

La dépression se définit habituellement comme un ensemble de problèmes dont les caractéristiques principales sont la présence d'une humeur dépressive, ou d'une perte de plaisir généralisée, et de détresse psychologique. Selon cette définition, les problèmes suivants pourraient également, dans certains cas et à différents degrés, faire partie de la dépression : (a) perte ou prise de poids significative; (b) insomnie ou hypersomnie; (c) agitation ou retard psychomoteur; (d) fatigue ou perte d'énergie; (e) sentiment d'indignité ou culpabilité inappropriée ou excessive; (f) diminution de la capacité de concentration ou indécision; (g) idéation suicidaire. Sur la base de cette définition très générale, deux façons très différentes de conceptualiser ce phénomène sont présentes dans la littérature : les conceptions catégorielle et dimensionnelle.

La *conception catégorielle* repose sur un système diagnostique (DSM-IV; APA, 1994) qui tente de regrouper les différents problèmes humains en catégories de comportements « anormaux », sur la base du postulat selon lequel ces catégories sont distinctes les unes des autres sur le plan qualitatif. Dans cette conception, le fait de recevoir un diagnostic de dépression majeure requiert de satisfaire à au moins cinq des critères énumérés précédemment, sur une période d'au moins deux semaines. Un tel diagnostic est alors conceptualisé comme qualitativement distinct des autres catégories diagnostiques (dont la dysthymie) et des niveaux inférieurs de désespoir ou

de démoralisation. La *conception dimensionnelle*, quant à elle, ne repose pas sur des critères de temps et de sévérité, la dépression y étant dépeinte comme un phénomène « normatif », réparti le long d'un continuum de gravité (Akiskal, 2001; Zahn-Waxler, Klimes-Dougan, & Slattery, 2000; Zuckerman, 1999). En fait, ce continuum s'étendrait d'un état de bien-être psychologique total à un état dépressif grave et handicapant pour l'individu.

En ce qui a trait à l'intervention, le choix entre ces deux approches peut avoir des conséquences théoriques et pratiques importantes. En effet, le fait de conceptualiser la dépression d'une manière catégorielle entraîne qu'une intervention sera considérée efficace si elle permet de diminuer le pourcentage d'individus correspondant encore aux critères diagnostiques à la fin du programme. Dans cette optique, une intervention qui atteindrait ce but serait perçue comme efficace, indépendamment des problèmes que les participants pourraient encore présenter. Par ailleurs, l'adhésion à un modèle dimensionnel exige plutôt que l'intervention vise à rapprocher le plus possible l'individu d'un état de bien-être psychologique, social et affectif total.

Pour résoudre cette ambiguïté, Flett, Vredenburg et Krames (1997) ont effectué une recension des écrits, dans laquelle ils ont examiné les résultats d'études portant sur les quatre tests directs du caractère catégoriel ou dimensionnel d'un trouble psychologique : (a) les tests de continuité phénoménologique, qui cherchent à vérifier si les individus qui présentent différents niveaux de dépression se distinguent les uns des autres en ce qui concerne les manifestations, les antécédents et les conséquences; (b) les tests de continuité étiologique, qui tentent d'évaluer si la présence de symptômes sous-cliniques de dépression est un prédicteur du développement d'un épisode diagnostique; (c) les tests de continuité typologique, destinés à évaluer l'existence de sous-types de dépression qualitativement distincts les uns des autres; et (d) les tests de continuité psychométrique, qui tentent de vérifier si les scores obtenus à l'aide d'instruments de mesure de la dépression se distribuent d'une façon continue ou discontinue. Sur la base de ce travail, ils ont conclu que la plupart des résultats empiriques appuyaient une conception dimensionnelle de la dépression. En outre,

dans un numéro spécial du *Journal of Affective Disorders* consacré à la même question, de nombreux experts de la dépression en sont arrivés aux mêmes conclusions (voir Judd, 1997).

D'un point de vue plus pratique, d'autres arguments ont aussi été formulés pour appuyer la validité d'un modèle dimensionnel de la dépression. Ainsi, plusieurs études ont démontré que la présence de seulement deux ou trois symptômes de dépression pouvait être associée à un niveau considérable de dysfonctionnement dans la vie des individus atteints (Akiskal, 2001; Harrington, 1993). Pour cette raison, Judd, Schettler et Akiskal (2002) ajoutent que, de par leur prévalence élevée dans la population, les niveaux sous-cliniques de dépression sont potentiellement responsables d'un plus grand nombre de problèmes sociaux que les niveaux diagnostiques. Les individus présentant des niveaux sous-cliniques de dépression seraient d'ailleurs moins nombreux à bénéficier de traitements efficaces, ce qui pourrait avoir pour effet de prolonger leur souffrance (Wells *et al.*, 1989). Dans ce contexte, le recours à une conception dimensionnelle semble permettre une compréhension beaucoup plus juste et complète de la dépression (Harrington & Clark, 1998; Kazdin, 1993; Muñoz, 1993). De plus, les mesures dimensionnelles sont généralement associées à un plus grand pouvoir statistique dans le cadre d'analyses statistiques. Par conséquent, l'utilisation de mesures continues de la dépression permettrait aux études développementales de détecter des effets beaucoup plus fins et d'utiliser des modèles analytiques plus complexes (Muñoz, 1987).

En ce qui concerne le modèle catégoriel, son seul avantage serait, sauf erreur, « de permettre une classification de la maladie [la dépression] qui ressemble de près aux méthodes de mesures utilisées en épidémiologie psychiatrique » (Roberts, 1987, p. 46). Bien que la standardisation des mesures et des approches représente un objectif louable pour toute démarche scientifique, les connaissances actuelles suggèrent que cette standardisation devrait plutôt reposer sur une conception dimensionnelle de la dépression ou sur une approche permettant la comparaison directe des deux conceptions (Pickles & Angold, 2003; Zahn-Waxler *et al.*, 2000).

La mesure de la dépression

En attendant le développement ou l'adoption plus répandue d'outils de mesure valides reposant sur une conception réellement dimensionnelle des troubles psychologiques (à ce sujet, voir Clark & Watson, 1991; Zuckerman, 1999), trois approches distinctes sont actuellement utilisées pour évaluer la dépression (Compas, Ey, & Grant, 1993; Compas *et al.*, 1998). Premièrement, l'approche *diagnostique* repose sur une évaluation catégorielle de la dépression. Dans ce cadre, des entrevues cliniques structurées ou semi-structurées sont habituellement utilisées afin de déterminer la présence ou l'absence des divers symptômes associés à la dépression (First, Spritzer, Gibbon, & Williams, 1997). Le résultat de cette forme de mesure est une classification des individus évalués en deux groupes : les « normaux » et les « dépressifs ». La principale limite de cette approche, souvent citée comme la plus valide pour évaluer la dépression, est son incompatibilité avec une conception dimensionnelle (Ingram & Siegle, 2002).

Deuxièmement, l'approche par *syndromes* s'appuie habituellement sur l'utilisation de questionnaires visant à évaluer la présence ou l'absence de groupes de symptômes (syndromes) définis empiriquement sur la base d'analyses factorielles (Achenbach & Eldebrock, 1983). Le caractère empirique de ces instruments nécessite que, chez les individus évalués, la présence ou l'absence des syndromes ciblés repose habituellement sur une comparaison, à une norme, des résultats obtenus par ces individus. Ainsi, seuls les individus présentant des résultats extrêmes à l'échelle de dépression de ces instruments seront considérés comme « dépressifs ». Deux limites majeures diminuent l'utilité de cette approche. Premièrement, l'utilisation de normes entraîne qu'un phénomène « normatif » (c.-à-d. répandu), mais tout de même handicapant, tel que la présence de quelques symptômes de dépression, ne sera pas considéré comme problématique (Petersen *et al.*, 1993). Deuxièmement, étant issus des résultats d'analyses factorielles, les syndromes évalués par ces échelles ont souvent un caractère artificiel à cause du niveau élevé de comorbidité observé entre différents problèmes de santé mentale (c.-à-d. anxiété et dépression). En effet,

l'observation d'un niveau élevé de comorbidité entre deux problèmes distincts ne signifie pas que ces problèmes représentent un même construit sous-jacent ou qu'ils se développent sous l'influence de mécanismes identiques (Barnett & Gotlib, 1988).

Finalement, une troisième approche repose sur l'évaluation, souvent par le biais de questionnaires autorévélés, de la *sévérité des symptômes* de dépression manifestés par les individus concernés (Beck, Steer, & Brown, 1996). Habituellement, les résultats obtenus à ces questionnaires sont traités d'une manière continue ou en référence à des points de coupure visant à distinguer différents niveaux de sévérité. Si cette approche semble être celle qui se rapproche le plus d'une conception dimensionnelle de la dépression, elle n'est cependant pas exempte de limites (Coyne & Downey, 1991; Ingram & Siegle, 2002; Kolvin & Sadowski, 2001). Premièrement, un score élevé peut parfois être difficile à interpréter, compte tenu de la nature des questions ou des choix de réponses utilisés. Par exemple, un individu pourrait obtenir un score élevé à l'inventaire de dépression de Beck (Beck *et al.*, 1996) de trois manières différentes, soit en : (a) présentant quelques symptômes sévères de dépression; (b) en présentant plusieurs symptômes peu sévères; (c) en ne présentant qu'un seul symptôme sévère (c.-à-d. culpabilité) qui serait évalué par un grand nombre d'items distincts. Deuxièmement, les résultats obtenus à ces questionnaires étant basés sur l'addition des réponses d'un individu aux items, un individu ne présentant aucun des symptômes principaux de la dépression (humeur dépressive, perte d'intérêt ou de plaisir ou irritabilité) pourrait tout de même obtenir un résultat élevé.

La solution suggérée à ces divers problèmes serait d'utiliser un questionnaire (ou une entrevue clinique structurée) dans lequel un seuil minimal de sévérité serait établi, afin d'évaluer la présence ou l'absence des différents symptômes de dépression. Le résultat global pourrait alors reposer sur le nombre total de symptômes présentés par l'individu évalué, et les individus ne présentant aucun signe d'humeur dépressive, de perte d'intérêt ou de plaisir ou d'irritabilité obtiendraient alors un score total de zéro (Ingram & Siegle, 2002; Kolvin & Sadowski, 2001). À l'heure actuelle, le seul instrument permettant d'effectuer ce type d'évaluation est, à notre connaissance, le

Inventory to Diagnose Depression (Present and Lifetime Version) de Zimmerman et Corryell (1987 a, 1987 b, 1988, 1994).

LE DÉVELOPPEMENT DE LA DÉPRESSION CHEZ L'ENFANT ET L'ADOLESCENT : LIMITES DES CONNAISSANCES ACTUELLES

Bien que la compréhension scientifique des mécanismes intervenant dans le développement de la dépression chez l'enfant et l'adolescent ait beaucoup évolué au cours des dernières décennies, deux obstacles en limitent encore la portée : (a) le manque d'intégration des connaissances actuelles; (b) la méconnaissance du rôle effectif de certaines variables environnementales, telles que le vécu scolaire dans le développement de la dépression chez l'enfant et l'adolescent.

Première limite : le manque d'intégration des connaissances

Compte tenu du très grand nombre d'articles scientifiques portant sur le développement de la dépression publiés au cours des dernières années, le besoin d'une recension complète et intégrée des écrits portant sur les facteurs actifs dans l'émergence de ce trouble est évident. De nombreuses recensions de ce type ont d'ailleurs été réalisées au cours des dernières années. Hélas, la majorité de celles-ci ne considèrent qu'un nombre limité de facteurs de risque et de protection (voir Albright, 1999; Baron, 1993; Barnett & Gotlib, 1988; Birmaher *et al.*, 1996; Garber & Horowitz, 2002; Hammen, 1991; Kovacs & Devlin, 1998; Lewinsohn & Essau, 2002) ou se limitent à une description détaillée des différents mécanismes sous-jacents à l'action de facteurs de risques spécifiques (voir Brooks-Gunn, Auth, Petersen, & Compas, 2001; Cicchetti & Toth, 1995; Joiner & Coyne, 1999; Zuckerman, 1999). D'autres chercheurs ont aussi effectué des recensions plus théoriques, se contentant d'utiliser une partie des connaissances actuelles de manière à soutenir l'ébauche de modèles explicatifs, n'intégrant eux-mêmes qu'une fraction des facteurs en jeu dans le développement de la dépression (voir Cummings, DeArth-

Pendley, Du Rocher Schudlich, & Smith, 2001; Haines, Metalsky, Cardamone, & Joiner, 1999; Hankin & Abramson, 2001; Roberts & Monroe, 1999).

Malgré ces dernières observations, les efforts mentionnés demeurent louables et ne peuvent qu'enrichir notre compréhension des mécanismes du développement de la dépression. Cependant, à plus long terme, le recours répété à cette approche fragmentée de développement des connaissances risque d'obscurcir la compréhension générale du phénomène à l'étude si elle n'est pas combinée à une approche plus englobante.

À notre connaissance, le seul effort tenté pour en arriver à une intégration de ce type a été réalisé par Cicchetti et Toth (1998). La principale limite de cette recension provient du fait que la majorité des études consultées est issue de travaux portant sur le développement d'enfants de parents dépressifs, ce qui diminue la généralisation possible des conclusions. Par contre, cette recension des écrits a tout de même contribué à mettre clairement en lumière le fait que la dépression représente un phénomène biopsychosocial, et que la compréhension de son développement ne sera possible que par la considération simultanée des multiples facteurs en cause.

Deuxième limite : la méconnaissance du rôle du vécu scolaire

Nonobstant l'état parcellaire des connaissances actuelles, certains des facteurs responsables du développement de la dépression chez l'enfant et l'adolescent sont aujourd'hui connus. Comme nous le verrons plus loin, l'effet de différents facteurs sur l'émergence de symptômes dépressifs a pu être démontré dans le cadre d'études rigoureuses et systématiques au cours des dernières années. Généralement, ces facteurs concernent certaines caractéristiques biologiques (héréditaires) et psychologiques des enfants et des adolescents, de même que certaines caractéristiques des expériences de socialisation qu'ils ont pu vivre au sein de leurs familles et de leurs groupes de pairs.

Si l'on se réfère au modèle écologique de Bronfenbrenner (1977), qui décrit les différentes sources possibles d'influence sur le développement humain, ces trois types de facteurs peuvent se regrouper en deux catégories. Premièrement, les différents facteurs de risque biologiques (héréditaires, endocriniennes, etc.) et psychologiques représentent un « ontosystème », ou un système composé de l'ensemble des caractéristiques propres à l'individu (voir aussi Cicchetti & Toth, 1995). Deuxièmement, les différents facteurs faisant référence aux expériences de socialisation des individus, au contact des membres de leur famille et de leurs pairs, représentent différents « microsystèmes ». Un microsystème est défini par Bronfenbrenner (1977) comme un système qui englobe les différentes expériences de socialisation qui résultent des interactions directes entre un individu et divers agents de socialisation (c.-à-d. parents, pairs, enseignants, employeurs, collègues, etc.).

Outre les ontosystèmes et les microsystèmes, Bronfenbrenner (1977) identifie trois autres sources potentielles d'influence sur le développement humain : les méso-, exo- et macro- systèmes. Il définit un mésosystème comme « les interrelations qui existent entre les différents milieux de vie incluant l'individu en développement [microsystèmes] à un moment particulier de son existence ». D'un autre côté, un exosystème serait une

> « extension du mésosystème englobant différents types formels et informels de structures sociales qui, si elles n'incluent pas directement l'individu en développement, exercent tout de même une influence déterminante sur ce qui se produit à l'intérieur des différentes structures sociales auxquelles l'individu participe plus directement. » (Bronfenbrenner, 1977, p. 515)

Finalement, un macrosystème représente

> « les principes institutionnels généraux qui caractérisent ou influencent une culture ou sous-culture spécifique, tels que les systèmes économiques, sociaux, éducatifs, légaux et politiques, et dont les différents micro-, méso- et exo- systèmes sont les manifestations concrètes. » (Bronfenbrenner, 1977, p. 515)

Malheureusement, l'impact sur le développement de la dépression de ces systèmes plus complexes, de même que de plusieurs autres microsystèmes potentiellement importants (tels que les relations maîtres-élèves), semble avoir fait l'objet de très peu d'études systématiques. Il est peu probable que les effets de différents exo- et macro-systèmes sur le développement de la dépression puissent être clarifiés sans que l'aient été préalablement ceux des micro- et méso- systèmes dont ils sont en partie composés. Dans ce contexte, il apparaît clair que l'effet des microsystèmes restants et de différents mésosystèmes représente un défi prioritaire de taille pour les travaux portant sur le développement de la dépression. Compte tenu du fait que l'école est un mésosystème dans lequel interagissent différents microsystèmes centraux au développement humain (relations maîtres-élèves, groupes de pairs, pratiques éducatives « scolaires » des parents), l'étude de l'effet du vécu scolaire sur le développement de la dépression semble représenter une façon privilégiée de faire face à ce défi.

Le milieu scolaire, parce qu'il représente un élément central du vécu des enfants et des adolescents, une source de socialisation déterminante pour l'adaptation sociale et professionnelle future des élèves et un milieu dans lequel interagissent différents microsystèmes importants, occupe en effet une place de choix pour influencer le développement humain (Bronfenbrenner, 1977; Eccles, Lord, & Midgley, 1991; Moos, 1979; Mortimore, 1995; Roeser, Eccles, & Strobel, 1998; Rutter *et al.*, 1997). Certaines hypothèses suggèrent d'ailleurs que le vécu scolaire puisse exercer un impact déterminant sur le développement de la dépression.

Tout d'abord, certaines hypothèses suggèrent indirectement que l'augmentation des taux de prévalence de dépression observée auprès de cohortes récentes d'adolescents (Fombonne, 1998; Lewinsohn, Rohde, Seeley, & Fisher, 1993) puisse être en partie liée aux caractéristiques des écoles modernes. Ainsi, Eccles *et al.* (1991, 1993) ont présenté une série d'éléments de preuve suggérant que les écoles modernes étaient insuffisamment équipées pour répondre aux principaux besoins développementaux des adolescents. Ces besoins ont trait au développement de l'autonomie, à l'intimité,

au développement de relations amicales caractérisées par la confiance et la réciprocité, à la construction de l'identité et à l'atteinte de la pensée opératoire formelle. Or, les écoles secondaires modernes sont souvent caractérisées, en comparaison aux écoles primaires, par un resserrement des pratiques d'encadrement disciplinaire, des niveaux élevés de compétition académique et sociale, une rupture des réseaux sociaux et de plus faibles exigences cognitives. Cet écart entre les besoins développementaux des adolescents et les caractéristiques de leur vie scolaire représente un facteur de risque potentiellement important pour le développement de problèmes psychosociaux, particulièrement chez les élèves présentant déjà un niveau élevé de vulnérabilité parce que généralement moins bien préparés à gérer le déséquilibre résultant de cet écart (Eccles *et al.*, 1991, 1993). Dans le cas plus spécifique de la dépression, il est probable que les élèves exposés à un tel déséquilibre puissent en venir à intérioriser l'idée que leurs besoins personnels sont indignes d'attention et à développer ainsi un sentiment d'impuissance ou de désespoir. De tels sentiments ont souvent été associés à la dépression (Abramson, Metalsky, & Alloy, 1989; Beck, 1967, 1987; Haaga, Dyck, & Ernst, 1991; Hankin & Abramson, 2001).

Ces observations sont d'autant plus alarmantes que des hypothèses additionnelles suggèrent que certains changements importants survenus au cours des dernières décennies ont pu créer des déséquilibres additionnels chez les adolescents. Par exemple, Diekstra (1995) et Robins (1995) indiquent que le vécu des adolescents modernes est souvent caractérisé par un début plus hâtif des changements pubertaires, une exposition prolongée au système scolaire causée par la prolongation de la formation requise pour occuper la majorité des emplois et une désagrégation des sources traditionnelles de soutien social (familles intactes et étendues, cohésion communautaire, appartenance à des groupements religieux, etc.). Dans ce contexte, les adolescents modernes doivent s'adapter plus tôt à un corps et à une physiologie adultes, sans toutefois pouvoir assumer des rôles sociaux adultes. De surcroît, compte tenu de la désagrégation des sources traditionnelles de soutien social, ils se retrouvent souvent seuls pour affronter la crise d'identité qui pourrait résulter d'un tel conflit de

rôle. Finalement, alors que le milieu scolaire occupe une place privilégiée pour offrir aux adolescents les sources complémentaires de soutien dont ils ont besoin, les écoles modernes semblent insuffisamment équipées pour remplir cette fonction (Eccles *et al.*, 1991, 1993). La qualité du soutien social dont dispose un adolescent représente d'ailleurs un facteur de protection connu eu égard au développement de la dépression (Cheng, 1998; Kiesner, 2002; Stein, Newcomb, & Bentler, 1996). Ces différentes hypothèses sont d'autant plus plausibles que la dépression se développe habituellement pour la première fois au cours de l'adolescence, suite à la transition du primaire au secondaire et à l'apparition des premiers changements pubertaires (Cyranowski, Frank, Young, & Shear, 2000; Nolen-Hoeksema, 2002).

SOMMAIRE DES OBJECTIFS DE LA THÈSE

Tout au long de ce chapitre introductif, nous nous sommes efforcé de démontrer l'importance d'en arriver à une compréhension intégrée des facteurs intervenant dans le développement de la dépression chez l'enfant et l'adolescent. Par la suite, nous avons brièvement exposé deux des principales limites des connaissances actuelles dans ce domaine. La première limite concerne un manque d'intégration des connaissances actuelles, et la seconde la méconnaissance du rôle potentiel du vécu scolaire dans le développement de la dépression. L'objectif principal de cette thèse est de repousser ces deux limites.

Dans le chapitre suivant, une synthèse des connaissances actuelles sera proposée par le biais d'une recension des écrits rapportant les travaux relatifs au développement de la dépression chez les enfants et les adolescents parus au cours des 12 dernières années. Compte tenu du très grand nombre d'études publiées au cours de cette période et des limites méthodologiques importantes de plusieurs d'entre elles, seules les études respectant les critères méthodologiques ciblés par Barnett et Gotlib (1988) dans une recension similaire visant à distinguer les antécédents, corrélats et conséquences de la dépression adulte, seront considérées dans ce chapitre. De plus,

compte tenu des enjeux méthodologiques distincts inhérents aux études portant sur l'identification de facteurs biologiques de risque et de protection, seuls les facteurs psychosociaux seront considérés. Plus précisément, ce chapitre sera divisé comme suit. Dans un premier temps, une brève introduction permettra de situer la portée et l'objectif du chapitre; elle sera suivie d'une courte section présentant la définition de la dépression utilisée. Suivra la présentation des critères de sélection des études. Dans le corps du chapitre, la présentation des connaissances actuelles concernant les facteurs individuels actifs dans le développement de la dépression précédera celle des facteurs sociaux. Pour chacun des facteurs, les bases théoriques sous-jacentes à l'étude du facteur seront présentées avant l'exposé des résultats obtenus dans les études retenues. Enfin, après une synthèse des résultats exposés, nous présenterons les principaux défis qui devront être relevés au cours des prochaines années.

Dans le troisième chapitre, la seconde limite précitée sera abordée. Plus précisément, l'impact de différents éléments inhérents au vécu scolaire des adolescents sur leur risque de développer des symptômes de dépression sera examiné plus en profondeur. À cette fin, seront utilisées les données issues de la première année du Projet Montréalais sur le Développement de la Dépression Adolescente (PMDDA, ou MADDP dans sa version anglaise), une étude prospective longitudinale élaborée précisément pour répondre à cette question. Dans un premier temps, nous présenterons un résumé des connaissances actuelles concernant la relation entre la vie scolaire et le développement de la dépression. Par la suite, un exposé succinct de la méthodologie PMDDA sera effectué. La stratégie analytique utilisée dans ce chapitre vise à répondre à la question suivante : Quels aspects de la vie scolaire des adolescents représentent des facteurs de risque pour le développement de symptômes dépressifs? Deux sous-questions spécifiques seront aussi abordées. Les relations observées sont-elles affectées par le sexe des participants? Le sont-elles par leurs niveaux antérieurs de symptômes dépressifs? Cette dernière sous-question servira à distinguer les facteurs de risque selon leur contribution à l'émergence (utilité préventive) ou à l'aggravation (utilité clinique) de symptômes dépressifs. La

conclusion de ce chapitre mettra l'accent sur l'utilité préventive et explicative des résultats obtenus, de même que sur leurs principales limites.

La conclusion de cette thèse effectuera un retour sur les grandes conclusions des deux chapitres centraux. Finalement, certaines recommandations visant à guider les efforts préventifs dans ce domaine seront brièvement formulées.

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Chapitre II

Psychosocial antecedents for child and adolescent depression: A review of the past 12 years[1]

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[1] Soumission probable de l'article/monographie : *Psychological Bulletin* ou *Genetic, Social, and General Psychology Monographs*

ABSTRACT

In the last decade, we have witnessed an explosion of scientific publications on depression development. In this review, we attempt to provide an integrated synthesis of the current knowledge regarding the psychosocial antecedents implicated in depression development in children and adolescents. Following a brief definition of depression, we will present the results of a detailed review of the psychosocial risk and protective factors implicated in children's and adolescents' depression development. Based on five criteria (peer-reviewed, valid measures of depression, prospective longitudinal design, control of previous symptom levels, and adequate statistical power), 91 relevant studies, published between 1990 and 2003, were identified through the MEDLINE, ERIC, and PsycINFO databases. For each potential antecedent of depression considered in this review, theoretical issues will be presented first. Empirical support regarding the role of these factors in depression development will be presented next. Throughout this review, results relevant to each potential risk or protective factor are presented after a brief review of the theoretical bases underlying the study of this specific factor. Finally, we propose an integrated synthesis of the results and pinpoint three key challenges for the future study of depression development (375 references).

Key words: childhood and adolescence, depression, antecedents, literature review, developmental psychopathology.

The World Health Organization's Global Burden of Disease study revealed that depression currently represents the fourth cause world-wide of disability and is expected to reach second rank in 2020 (Murray & Lopez, 1996 a, 1996 b). Many facts explain this phenomenon. First, depression is a highly recurrent, comorbid, and sometimes chronic, early-onset mental health problem (Angold, Costello, & Erkanli, 1999; Baron, 1993; Fleming, & Offord, 1990; Harrington & Dubicka, 2001; Kessler, 2002; Kessler, Avenevoli, & Merikangas, 2001; Lewinsohn, & Essau, 2002; Newman *et al.*, 1996). Second, depression constitutes a highly prevalent mental health problem. Indeed, during their lifetimes, close to 21% of women and 12% of men will suffer from depressive symptoms severe enough to meet diagnostic criteria, and this rate appears to be on the rise in cohorts born since 1935-1940 (Cross National Collaborative Group, 1992; Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Kessler *et al.*, 1994; Klerman & Weissman, 1989). Similar rates and temporal trends have been reported in recent adolescent samples (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Lewinsohn, Rohde, Seeley, & Fisher, 1993). Finally, depression is associated with significant impairment in most domains of the affected person's life: consequences range from impaired interpersonal functioning, work productivity, and educational attainment to homelessness and premature death by suicide (Ensinck *et al.*, 2002; Kessler, 2002; Kessler, Foster, Saunders, & Stang, 1995; Harrington, & Dubicka, 2001; Hodgins, 1996; Schneider, Müller, & Philipp, 2001). In this context, it is not surprising that the annual societal costs of major depression have been estimated to range from 30.4 to 43.7 billion dollars US and to be close to 3.39 billion pounds in the United Kingdom (Chisholm, 2001; Greenberg, Stiglin, Finkelstein, & Berndt, 1993).

These observations have led many national and international health organizations (e.g., Dawson & Tylee, 2001; Mrazek & Haggerty, 1994) and scholars (e.g.,

Harrington & Clark, 1998; Morin & Chalfoun, 2003; Muñoz, Le, Clarke, & Jaycox, 2002; Muñoz, & Ying, 1993) to stress the pressing need to reinforce the research agenda for the prevention of childhood and adolescent depression. Such efforts, however, would not be possible without a clear and integrated knowledge of the different antecedents implicated in depression development (Coie *et al.*, 1993; Kazdin, 1993; Mrazek & Haggerty, 1994). Unfortunately, few reviews have been published on this topic and most of them have either focused on a limited number of risk factors (e.g., Albright, 1999; Baron, 1993; Birmaher *et al.*, 1996; Garber & Horowitz, 2002; Kovacs, & Devlin, 1998) or have used only some of the available studies to support a proposed new theoretical integration (e.g., Joiner & Coyne, 1999; Zuckerman, 1999). Additionally, many reviews were published with the goal of uncovering the mechanisms responsible for the actions of factors purportedly implicated in depression development (e.g., Brooks-Gunn, Auth, Petersen, & Compas, 2001; Cicchetti, & Toth, 1995; Cummings, DeArth-Pendley, Du Rocher Schudlich, & Smith, 2001; Haines, Metalsky, Cardamone, & Joiner, 1999; Roberts & Monroe, 1999). However, this fragmented approach impairs our ability to see the "big picture" of depression development. To our knowledge, the only recent attempt to provide an integrated overview of the mechanisms implicated in depression development was made by Cicchetti and Toth (1998). But most of the evidence presented by these authors relies on studies of children of depressed parents, thus limiting the extent to which their interpretations can be generalized.

Given the current number of available studies, the need to integrate current knowledge is even more pressing. Indeed, studies on depression are now so numerous that it has become very difficult to integrate all of the available knowledge. To illustrate this point, we conducted a quick search on PsycINFO and MEDLINE databases using only "depression" as a key word, for various decades. We then limited this search to studies of "children and/or adolescents." The results of this search are depicted in Figure 1. Looking at these numbers, scholars can only feel overwhelmed by the incredible body of available knowledge.

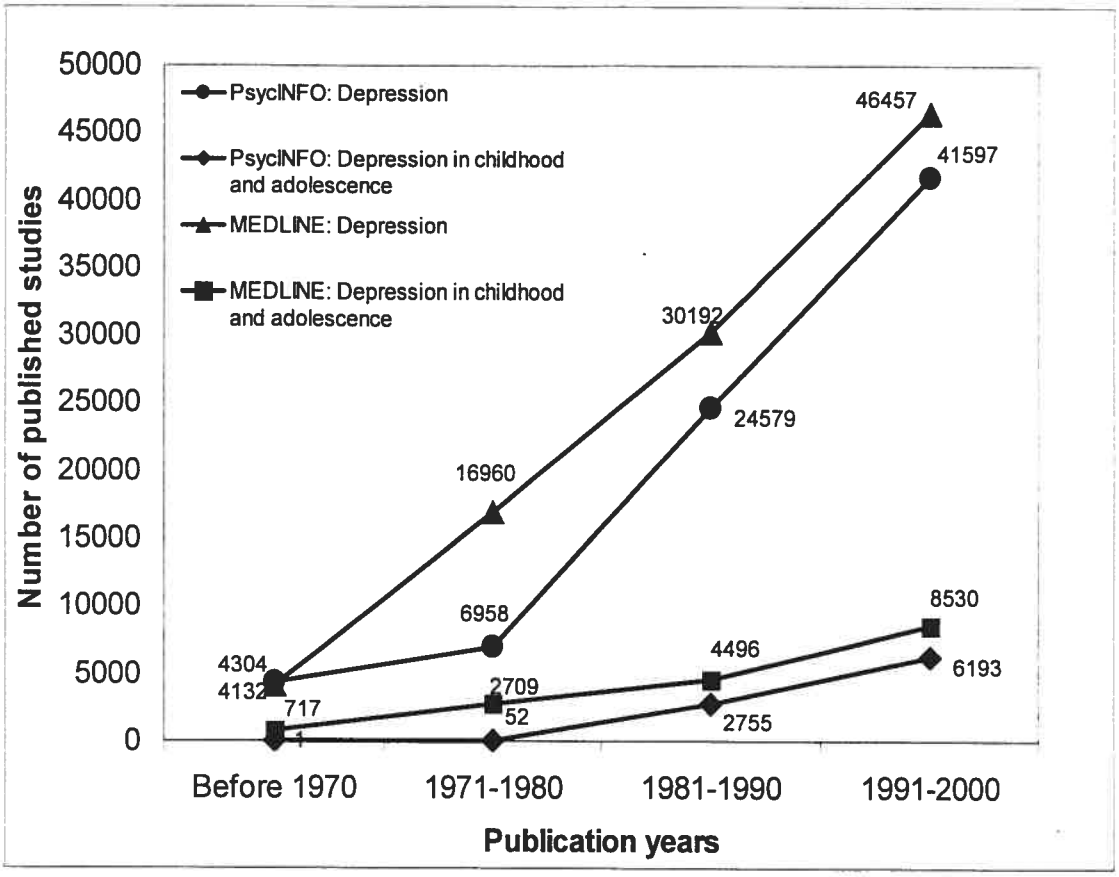

Figure 1: Number of publications on depression and on child and adolescent depression across decades according to the PsycINFO and MEDLINE databases.

THE PRESENT REVIEW

Based on the complementary objectives of guiding preventive efforts and of assisting the development of an integrated etiological model of depression, the present review will attempt to provide a synthesis of the current knowledge regarding the specific role of psychosocial factors in child and adolescent depression development. Following a brief section in which the categorical-dimensional debate on the definition of depression will be addressed, the main part of this paper will present a detailed review of research evidence produced within the last twelve years regarding

the role of psychosocial antecedents in child and adolescent depression development. This section will focus mostly on results from methodologically sound studies published between January 1990 and March 2003. Evidence regarding the role of individual factors in depression development will be presented first (temperament and personality traits, cognitive style, self-schemas and attachment style, self-esteem and perceived competencies, behavioral competencies and coping style, previous disorders, and other individual factors). A presentation of research results concerning the role of social factors will then follow (generic life events, family environment, peer relationships, school life, and neighborhood environment). For each of these potential antecedents, theoretical issues and conclusions from previous reviews will be presented first. A presentation of the empirical support from the studies retained for this review will then follow. As a conclusion, an attempt will be made to integrate the conclusions from the previous sections and to identify the main challenges that should guide future research efforts.

DEFINING DEPRESSION

Depression is usually defined as an array of adaptive problems, mostly characterized by depressed mood, loss of interest/pleasure in activities or irritability, and psychological distress. According to most definitions, the following problems may also be part of the clinical picture of depression: (a) significant weight or appetite changes; (b) insomnia or hypersomnia; (c) psychomotor retardation or agitation; (d) fatigue or loss of energy; (e) inappropriate or excessive feelings of guilt/worthlessness; (f) indecisiveness or diminished ability to think and to stay focused; (g) suicidal ideation. Although many ways of conceptualizing depression emerged from this generic definition (American Psychiatric Association - APA, 1994; Compas, Connor, & Hinden, 1998; Zuckerman, 1999), most of them can be grouped into two categories: dimensional and categorical. According to categorical conceptions, depression is defined according to a diagnostic system (DSM-IV; APA, 1994) based on the hypothesis that psychological disturbances can be grouped into

qualitatively distinct categories of "abnormal" behaviors. Accordingly, someone showing at least five of the aforementioned "symptoms" with sufficient severity (i.e., most of the day and nearly every day for depressed mood) for at least 2 weeks would be diagnosed with "major depression." This diagnosis is seen as being qualitatively distinct from other affective diagnoses, such as dysthymia, as well as from lower levels of hopelessness or demoralization. In contrast, the dimensional conception depicts depression as a "normative" phenomenon and treats persistence and severity as quantitatively distinct points on the same severity continuum (Akiskal, 2001; Zahn-Waxler, Klimes-Dougan, & Slattery, 2000; Zuckerman, 1999). This conceptualization thus hypothesizes that depression could be positioned on a continuum somewhere between a state of complete emotional well-being and a state of severe handicapping depression (Akiskal, 2001; Zahn-Waxler *et al.*, 2000; Zuckerman, 1999).

Clearly, categorical measures of depression are the most widely used in depression research. However, most of the clinical, statistical, and methodological arguments favor the dimensional approach (Akiskal, 2001; Angst, & Merikangas, 2001; Harrington, 1993; Judd, Schettler, & Akiskal, 2002; Kazdin, 1993; Kendler, & Gardner, 1998; Maier, Gänsicke, & Weiffenbach, 1997). In fact, the only argument we located in favor of a categorical conceptualization of depression is that such an approach is far more consistent with a psychiatric epidemiology framework (e.g., Roberts, 1987).

In an attempt to investigate more systematically the scientific evidence favoring both approaches, Flett, Vredenburg and Krames (1997) reviewed studies in which the results allowed for a direct comparison of both conceptualizations. In addition to indirect evidence, Flett *et al.* (1997) based their conclusions on the results from four kinds of direct continuity tests: (a) phenomenological continuity (are individuals with different levels of depression qualitatively different from each other regarding the manifestations, antecedents, and consequences of depression?); (b) etiological continuity (do sub-clinical depressive symptoms significantly predict diagnostic

depression?)[2]; (c) typological continuity (can different sub-types of depression be qualitatively distinguished?)[3]; (d) psychometric continuity (are the patterns of scores obtained on depression measures distributed continuously or are they discontinuous?). Based on these four tests, the authors concluded that most of the current evidence appeared to support a dimensional view of depression. Interestingly, in a special issue of the Journal of Affective Disorders (Judd, 1997), other leading experts in this field reached the same conclusion. Clearly, current evidence advocates a dimensional view of depression. Nevertheless, comparability of results still being a worthy goal, it has previously been suggested that scholars should attempt to rely on comparisons of both forms of conceptualization in future studies (Pickles & Angold, 2003). Accordingly, in a previous draft of this review, we attempted to distinguish risk factors according to the categorical or dimensional nature of the instruments used in the various studies. Since the results were similar, we decided to combine them in the present paper for the sake of brevity (for similar conclusions, see Ackaert & Van den Bergh, 2002; Duggal, Carlson, Sroufe, & Egeland, 2001).

STUDY SELECTION

To find relevant studies, we limited the present review to peer-reviewed studies published or presented between January 1990 and March 2003. Building on Barnett and Gotlib's (1988) suggestions concerning the identification of depression "antecedents," studies were retained if: (1) the outcome was evaluated through a validated categorical or continuous measure of "pure" depression; (2) risk and protective factors were identified in analyses predicting depression from a variable measured earlier (in childhood or adolescence), after controlling statistically for the effects of previous levels of depression; and (3) they had sufficient statistical power.

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[2] For instance, Horwarth, Johnson, Klerman, & Weissman's (1992) study revealed that 55.3% of diagnostically depressed individuals previously exhibited sub-clinical levels of depression.

[3] For example, Merikangas, Wicki, and Angst (1994), in an attempt to distinguish amongst depressive subtypes according to their longitudinal course and associated characteristics, failed to find any evidence of qualitative distinctions across subtypes.

More precisely, limiting ourselves to studies relying on "pure" measures of outcome depression means that studies relying on mixed measures of depression (anxiety-depression, internalized disorders, etc.) were excluded. Indeed, mixed measures of depression are often based on the results of factor analysis in which the high level of correlation (comorbidity) between disorders results in their aggregation in a single factor. However, a high level of comorbidity between disorders does not mean that these disorders represent the same underlying phenomenon or that they are subjected to the same risk factors (Barnett & Gotlib, 1988).

Second, statistical controls of previous levels of depression are necessary to account for the bidirectionality of the observed relationships between depression and purported risk factors. For example, depression represents a known predictor of school adaptation problems (Kessler *et al.*, 1995; Mamostein & Iacono, 2001). Consequently, to conclude that school adaptation problems predict depression development, one must demonstrate that the effects observed are not due to the influence of students' baseline levels of depression. Controlling previous levels of the dependant variable (depression) within longitudinal studies is thus necessary to the unbiased identification of antecedents (Barnett & Gotlib, 1988). However, two exceptions regarding the nature of the controlled variable(s) were tolerated: (a) if sufficient alternative controls (early childhood variables known to be closely related to depression) were included in the analyses, (b) if controls were provided for previous levels of internalizing disorders (or other mixed measures). The first exception was tolerated based on the observation that depression represents a relatively rare phenomenon in early childhood. The second exception was tolerated because, due to their simultaneous consideration of multiple problems, mixed measures may represent more complete controls than "pure" measures.

Third, our focus on childhood and adolescence also meant that we retained only studies in which the predictors were measured in childhood or adolescence and in which the outcomes were evaluated in early adulthood at the latest.

Finally, it is well known that insufficient statistical power (which is determined simultaneously by the analytical strategy, the number of subjects and the number of variables) precludes the identification of otherwise real effects. We used Cohen's (1988) guidelines to evaluate the studies' levels of statistical power.

Using this strategy, we identified 91 longitudinal studies which evaluated the role of psychosocial factors in child and adolescent depression development. These studies are marked by an * in the reference list[4].

EXCLUDED THEMES

In an effort to be brief and given the high methodological quality of these studies, our methodological comments will be kept to a minimum in this review. More precisely, these comments will be limited to cases in which methodological differences may explain discrepant results and to widespread methodological limits (e.g., limits affecting most studies in which the impact of a specific risk factor was studied). It should also be noted that in most of the retained studies, previous levels of depression were controlled for and identified as significant predictors of later levels of depression. As this observation is highly consistent with conclusions from previous reviews regarding the recurrent character of depression and the temporal stability of depressive symptoms, this result will not be further highlighted (Baron, 1993; Fleming, & Offord, 1990; Harrington & Dubicka, 2001; Kessler, 2002; Lewinsohn, & Essau, 2002).

Similarly, gender, age, and ethnic group effects will not be treated in this review, for three reasons. First, these variables represent fixed and unchangeable characteristics of the person which cannot be completely considered as psychological or social in

[4] Methodological characteristics (authors, number of subjects, age, gender composition, follow-up duration, measures: Appendix A) and detailed results (statistical analyses, individual factors, family factors, other social factors, statistically non-significant results, strength of associations: Appendix B) of the 91 selected studies are presented in tables available upon request from the first author.

nature. Therefore, even studies which did not meet our inclusion criteria, such as epidemiological studies, could provide valid evaluations of their effects. Second, in the specific case of age and gender, the fact that females present higher levels of depression than males and that this difference emerges around 13 years of age has repeatedly been called one of the most robust findings in psychiatric epidemiology (e.g., Angold & Costello, 2001; Angold & Worthman, 1993; Hankin *et al.*, 1998; Kessler, 2003; Nolen-Hoeksema, & Girgus, 1994). Accordingly, we did not deem relevant to further address this question. Finally, as these factors cannot be modified in prevention programs, useful guidelines for such programs are more likely to come from studies that attempt to uncover the mechanisms underlying the effects of these factors than from studies in which their main effects are evaluated. Once again, the methodological soundness of such studies could not be evaluated with the present criteria. Moreover, recent reviews and studies on these topics were previously published and should be consulted by interested readers (Bebbington, 1996; Choi, 2002; Cyranowski *et al.*, 2000; Hankin & Abramson, 1999; Hill, Bush, & Roosa, 2003; Lee & Larson, 2000; Nolen-Hoeksema, & Girgus, 1994; Rudolph, 2002; Rumbaut, 1994; Tsai & Chentsova-Dutton, 2002).

Finally, the decision to limit this review to psychosial antecedents to the exclusion of biological or genetic ones is based on three observations. First, whereas many biological factors were found to play a role in depression, such as a dysregulation of neuroendocrine and neurotransmitter systems, sleep architecture irregularities, and brain structure abnormalities, most of the current knowledge about the exact role of these factors is based on cross-sectional adult studies (Ashman & Dawson, 2002; Brooks-Gunn *et al.*, 2001; Davidson, Pizzagalli, & Nitschke, 2002; Garber, & Horowitz, 2002; Gold, Goodwin, & Chrousos, 1988 a, 1988 b; Thase, Jindal, & Howland, 2002; Wallace, Schneider, & McGuffin, 2002). The exact etiological role of biological factors in depression development therefore remains unclear and these factors are, for the moment, best conceptualizsed as correlates (or state-markers) rather than antecedents of depression (Brooks-Gunn *et al.*, 2001; Thase *et al.*, 2002). Moreover, based on an extensive review of studies conducted in child, adolescent and

adult populations, Kaufman, Martin, King and Charney (2001) concluded that generalizing adult results to younger populations was far from evident.

Second, although the etiological role of heredity in depression development received strong support in family, twin, adoption and mixed studies, the exact genes implicated in depression development are still unknown (Kendler, 2001; Rice, Harold, & Thapar, 2002; Silberg, & Rutter, 2002; Souery, Rivelli, & Medlewicz, 2001; Sullivan, Neale, & Kendler, 2002; Wallace *et al.*, 2002). Genes therefore do not yet represent plausible targets for prevention programs.

Third, current knowledge indicates that the effects of biological and hereditary factors in depression development are most likely deeply intertwined with those of psychosocial factors. In fact, most of the biological factors previously found to be associated with depression represent known components of the human stress-response system (Ashman & Dawson, 2002; Brooks-Gunn *et al.*, 2001; Gold *et al.*, 1988 a, 1988 b; Thase *et al.*, 2001; Wallace *et al.*, 2002). Consequently, these factors may not be etiologically implicated in depressive onsets, but may rather represent automated mechanisms through which stressors exert a deleterious impact on the organism. Moreover, previous reviews indicate that these factors may be more relevant to the understanding of depression recurrence, rather than first onsets (Gold *et al.*, 1988 b; Meyer, Chrousos, & Gold, 2001; Post, Rubinow, & Ballenger, 1986). For example, most studies that attempted to explain the rise in depression often associated with pubertal development concluded that the effects of puberty were mostly due to the psychosocial changes (body image, relationships, etc.) which tend to follow physiological maturation, rather than hormonal and physiological changes (e.g., Alsaker, 1995; Angold, & Worthman, 1993; Cyranowski, Frank, Young, & Shear, 2000).

Similarly, genetic factors implicated in depression development were also found to predict other psychological risk factors, such as anxiety, behavioral disorders, or neuroticism, as well as to amplify an individual's exposure and reactivity to stressful

life events and other forms of social risk factors such as low social support and problematic family relations (e.g., Caspi *et al.*, 2003; Kendler, Gardner, & Prescott, 2002; Kendler, & Karkowski-Shulman, 1997; Kendler, Kessler *et al.*, 1995; Kendler, Walters *et al.*, 1995; O'Connor, McGuire, Reiss, Hetherington, & Plomin, 1998; O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998; Rice *et al.*, 2002). Similarly, whereas the heightened prevalence of depression and other psychosocial problems found in children of depressed parents could be due to genetic factors, current hypotheses converge on the fact that a plethora of other factors, such as parental depression-induced parenting impairments, stressful life event exposure, marital disharmony, and perinatal injuries are likely at play (e.g., Cicchetti, & Toth, 1998; Goodman, & Gotlib, 1999, 2002). To explain these interrelations between genetic and psychosocial risk factors, a new consensus rapidly emerging is that most of the effects of heredity on children and adolescent development are likely to be completely due to gene-environment or gene-personality correlations and interactions (e.g., Plomin, 1995; Rutter *et al.*, 1997; Silberg & Rutter, 2002; Zuckerman, 1999)[5].

In summary, our decision to limit the present review to psychological and social factors was based on current knowledge indicating that: (a) the exact etiological role of biological factors in the development of depressive onsets in children and adolescents remains unknown; (b) the precise genes implicated in depression development remain unidentified: and (c) in any case, the effects of biological and genetic factors in depression development appear to be deeply intertwined with those of psychosocial risk factors. Moreover, recent integrative reviews on these topics

[5] Rutter *et al.* (1997, p. 337) define gene-environment correlations as "situations in which variations in genetic liability are systematically associated with variations in specific environmental circumstances." Three types of gene-environment correlations are generally distinguished: (a) passive, which "reflect the fact that parents pass on their genes to their children and also create the environment in which children are raised" (Silberg & Rutter, 2002, p. 21); (b) reactive, which refer to "the experiences of children that derive from reactions of other people to children's genetic propensities" (Plomin, 1995, p.47); (c) evocative, which occurs when "children select, modify, construct, or reconstruct experiences that are correlated with their genetic propensities" (Plomin, 1995, p. 47-48). For their part, gene-environment interactions "describe the situation whereby genetic effects vary according to environmental circumstances" (Silberg & Rutter, 2002, p. 21) as well as a "type of genetic liability that involves differential susceptibility to specific environment features" (Rutter *et al.*, 1997, p. 337).

were previously published (Ashman & Dawson, 2002; Brooks-Gunn *et al.*, 2001; Davidson *et al.*, 2002; Garber, & Horowitz, 2002; Kaufman *et al.*, 2001; Kendler, 2001; Rice *et al.*, 2002; Silberg, & Rutter, 2002; Sullivan *et al.*, 2002; Thase *et al.*, 2002; Wallace *et al.*, 2002).

DEFINITIONS

In the remainder of this review, we refer to depression antecedents as risk factors, protective factors, moderators and mediators. These terms deserve a definition. First, *risk factors* represent "characteristics of the person or the environment that are associated with an increased probability of maladaptive developmental outcomes" (Compas, Hinden, & Gerhardt, 1995, p.273). Second, *protective factors* are characteristics that "interact with sources of risk such that they reduce the probability of negative outcomes under conditions of high risk but do not show an association with outcome under low risk" (Compas *et al.*, 1995, p. 273)[6]. Third, a *moderator* is a variable that "affects the relationship between two variables, so that the nature of the impact of the predictor on the criterion varies according to the level or value of the moderator [...]. A moderator interacts with a predictor variable in such ways as to have an impact on the level of a dependent variable" (Holmbeck, 1997, p. 599). Typically, moderators (and protective factors, which represent a specific case of moderation) are tested in statistical interaction procedures (Baron & Kenny, 1986; Holmbeck, 1997, 2002). Finally, a *mediator* "represents the generative mechanism through which the focal independent variable is able to influence the dependent variable of interest" (Baron & Kenny, 1986, p. 1173) and thus explains how a given predictor affects a dependent variable. The presence of a mediator is demonstrated when (Baron & Kenny, 1986; Holmbeck, 1997, 2002): (a) a hypothetical predictor, P, is significantly related to an outcome, O; (b) P is significantly related to a hypothetical mediator, M; (c) M is significantly related to O; and (d) the relation

[6] See also Kraemer *et al.* (1997) for a more complete discussion of risk and protective factors.

between P and O is significantly reduced (in cases of partial mediation, or disappears altogether in cases of complete mediation) when M is included in the equation.

INDIVIDUAL ANTECEDENTS

Temperament and Personality Traits

Theoretical Issues

The study of the relationships between personality traits and depression has its origins in the old nature-nurture controversy. Indeed, the stability and known genetic basis of personality traits led many theorists to postulate that some of these traits may represent lifelong vulnerability markers for depression (e.g., Eysenk, 1981). Recent theories integrate personality traits in more comprehensive models in which they are purported to play a central mediating role, linking genetic and early family influence to later social problems and stress reactivity, thus influencing depression development (Zuckerman, 1999).

Based on its replication in several independent samples from various countries and cultures, the Five-Factor Model of personality (or Big Five) recently emerged as consensual higher-order taxonomy of personality traits (John & Srivastava, 1999). This taxonomy represents broad dimensions of personality that summarize a number of more specific facets or primary traits: (a) Neuroticism (vs. Emotional Stability) reflects a proneness to experience psychological distress and a tendency to worry and to be insecure about things; (b) Conscientiousness reflects a tendency toward organization, persistence, and dependability; (c) Agreeableness reflects a tendency to show compassion and to seek and elicit cooperation and interpersonal trust; (d) Extraversion (vs. Introversion) reflects a capacity for happiness and a need for social stimulation and assertiveness ; and (e) Openness to Experience reflects a tendency to be creative, and an ability to tolerate and seek novelty and ambiguity.

In depression development research, the most frequently invoked personality trait is Neuroticism. This trait, which is influenced by hereditary factors similar to those involved in depression (Fanous, Gardner, Prescott, Cancro, & Kendler, 2002; Kendler *et al.*, 2002), has often been proposed to represent a preeminent vulnerability marker for depression (Cloninger, 1987; Eysenk, 1981; Zuckerman, 1999). Additionally, Neuroticism has been proposed to play an important moderating role in the stressor-depression relationship, increasing the individual's sensitivity and exposure to environmental stressors (e.g., Ormel & Wohlfart, 1991; Van Os & Jones, 1999; Watson, 2000). Introversion has also been alleged to play a role in depression development. Indeed, it has been suggested that this trait, when combined with a high level of interpersonal dependency (reviewed later in the "schemas" section), may place individuals in a paradoxical situation that increases their risk of developing depression: Introversion decreases their level of social support whereas interpersonal dependency increases their need for social support (Barnett & Gotlib, 1988). The potential role of other personality dimensions in depression development has received very little theoretical attention.

Although they did not always agree on all issues, previous reviews of the personality-depression relationship concluded that there was little support for an effect of Introversion in depression development, while Neuroticism appeared to constitute a clear risk factor (Enns & Cox, 1997; Klein, Durbin, Shankman, & Santiago, 2002; McCauley, Pavlidis, & Kendall, 2001). Some reviews, however, concluded that shy/inhibited temperament in childhood, an introversion precursor, did constitute a risk factor for depression (Dill & Anderson, 1999; Klein *et al.*, 2002). These reviews also unanimously concluded that the lack of methodologically sound studies considerably limited the conclusions that could be drawn.

Empirical Support

Although many studies attempted to understand the personality-depression relationship, very few of them (nine) met our inclusion criteria. Among those studies,

five were based on adolescent samples (Davies & Windle, 2001; Hops, Lewinsohn, Andrews, & Roberts, 1990; Katainen, Räikkönen, & Keltikangas-Järvinen, 1999; Stein, Newcomb, & Bentler, 1996; Windle & Windle, 2001) and four on long-term follow-up samples (Caspi, Moffit, Newman, & Silva, 1996; Jaffe *et al.*, 2002; Kasen, Cohen, Brook, & Hartmark, 1996; Krueger, 1999). It should be noted that none of these studies reported evidence of gender-based variability in the results.

Six of these studies evaluated the role of Neuroticism-related traits in depression development. For instance, Caspi *et al.* (1996) and Jaffe *et al.* (2002) found no relation between undercontrolled temperament and later depression. However, since undercontrolled temperament groups variables related to Conscientiousness and Agreeableness in addition to Neuroticism, these results are hard to interpret. Windle and Windle (2001), however, obtained a negative relationship between flexibility, another form of combination of the same traits, and depression development. Similarly, Davies and Windle (2001) revealed that dysrythmicity and low adaptability, which are clearer components of Neuroticism, represented significant predictors of later depression. For their part, Kasen *et al.* (1996) discovered no evidence of a relation between immaturity and depression. In the only study in which Neuroticism was directly measured, Krueger (1999) found that high levels of this trait and of some of its subcomponents (well-being, stress reaction, alienation, and harm avoidance) did predict depression development.

In five studies, high levels of Extraversion and related traits (sociability, activity, low inhibition, cheerfulness, etc.) were reported to predict lower levels of subsequent depression (Caspi *et al.*, 1996; Davies & Windle, 2001; Jaffe *et al.*, 2002; Kaitainen, Räikkönen, & Keltikangas-Järvinen, 1999; Stein *et al.*, 1996). Only two studies failed to replicate these results (Hops *et al.*, 1990; Krueger, 1999).

Other studies (Caspi *et al.*, 1996; Jaffe *et al.*, 2002) using personality traits based on a combination of Neuroticism and Conscientiousness suggest an absence of relation between this last trait and depression. However, Davies and Windle (2001) obtained a

relation between a temperamental measure of poor task orientation, a conscientiousness precursor, and depression. In the only study in which Conscientiousness was directly measured, Krueger (1999) found that this personality trait did not predict depression development. Finally, none of the studies retained evaluated the role of Agreeableness or Openness to Experience in depression development.

Summary

Current evidence suggests that introverts and neurotics may be at heightened risk of depression. Yet, very little is known about the hypothetical interactions between Neuroticism and stress reactivity and between Extraversion and social support availability that were proposed to explain the personality-depression relationship. Given the amount of theoretical attention devoted to the role of Neuroticism and Extraversion in depression development, it is surpising that so few scholars designed methodologically sound studies to verify these hypotheses in child and adolescent populations. Similarly, few conclusions could be reached regarding the potential role of Openness to Experience, Agreeableness, and Conscientiousness in depression development. Truly, until very recently, childhood personality was a neglected field of inquiry, and this neglect could partly explain the lack of available studies (Shiner & Caspi, 2003). Nevertheless, valid measurement instruments of childhood personality are now accessible and studies on the subject are increasingly available. Hopefully, these developments will allow future research to devote more attention to the potential role of personality in depression development

Cognitive Style

Theoretical Issues

Many classical theories attributed a determining role to cognitive style in the onset and maintenance of depression. Seligman's learned helplessness theory (Seligman,

1975; Abramson, Seligman, & Teasdale, 1979), later reformulated as the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989), hypothesizes that a negative attributional style (attributing negative events to stable, global and internal causes, and positive events to unstable, specific and external causes – the opposite attributional style is generally called optimism) represents a stable risk factor (diathesis) for depression. More specifically, the authors suggest that when individuals are exposed to stressors, those with a negative attributional style will become "hopeless" (i.e., expect that nothing positive will ever happen to them and/or that they are "doomed" to be exposed to uncontrollable negative events). Hopelessness is thus seen as a proximal determinant of depression, acting as a mediator between stress and depression, whereas attributions are seen as moderators of the stress-hopelessness relationship. Although Beck's (1967, 1987) theory does not directly implicate deviant cognitive processes, automatic thoughts, and the cognitive triad (negative view of the self, the world, and the future) in depression development, preferring to describe them as part of the depressive phenomenology, he suggested that exposure to life events congruent with an individual's "vulnerabilities" (reviewed later in the schema section) may give rise to dysfunctional attitudes about the significance of events. These dysfunctional attitudes are then presumed to favor the emergence of a cognitive triad (similar to hopelessness), leading to depression. Overall, both theories suggest that individuals exposed to stressful life events will react more negatively (cognitive triad, hopelessness) when they present a negative cognitive style. These attempts to describe the interrelationships between cognitive vulnerabilities and stressors in the emergence of depression are generally referred to as *diathesis-stress* hypotheses. For detailed discussions of these theories, interested readers should consult Haaga, Dyck, and Ernst (1991) and Abramson *et al.* (2002).

Cognitive theories of depression rapidly became, and still are, very popular among scholars and clinicians. This popularity probably stems from the fact that their initial formulations provided a first attempt to answer the numerous limitations of classical psychodynamic theories and of radical behaviorism, as well as from their successful application to the treatment of depression (see Hollon, Haman, & Brown, 2002).

Accordingly, many scholars conducted studies designed to test the cognitive theories of depression, mostly in adult and college student populations. The overall results, however, were quite disappointing. In fact, many previous reviews concluded that negative cognitive styles mostly represent, across the lifespan, correlates and consequences of depression rather than antecedents (Abramson *et al.*, 2002; Barnett, & Gotlib, 1988; Gladstone, & Kaslow, 1995; Haaga *et al.*, 1991; Joiner & Wagner, 1995; McCauley *et al.*, 2001). Moreover, additional reviews suggest that depressive individuals may even present more realistic and objective perceptions of themselves, the world, and the future than "normal" individuals who may be overly optimistic (Alloy & Abramson, 1988; Alloy, Albright, Abramson, & Dykman, 1990; Mezulis, Abramson, Hyde, & Hankin, 2004; Taylor, 1989; Taylor & Brown, 1988, 1994).

Empirical Support

In the present review, we found 16 studies examining the direct role of cognitive style in depression development (Abela, 2001; Garber, Keiley, & Martin, 2002; Hankin, Abramson, & Siler, 2001; Hilsman & Garber, 1995; Hops *et al.*, 1990; Lewinsohn, Allen, Seeley, & Gotlib, 1999; Lewinsohn, Gotlib, & Seeley, 1995; Lewinsohn, Joiner, & Rhode, 2001; Lewinsohn *et al.*, 1994; Nolen-Hoeksema, Girgus, & Seligman, 1992; Panak & Garber, 1992; Pomerantz, 2001; Robinson, Garber, & Hilsman, 1995; Rudolph, Kurlakowsky, & Conley, 2001; Schwartz & Koening, 1996; Spence, Sheffield, & Donovan, 2002). Only four of these studies did not try to evaluate the diathesis-stress hypothesis (Hops *et al.*, 1990; Lewinsohn *et al.*, 1994, 1995; Rudolph *et al.*, 2001).

Of these 16 studies, 8 found clear support for a direct main effect of cognitive style on depression development (Garber *et al.*, 2002; Hankin *et al.*, 2001; Lewinsohn *et al.*, 1994, 1995; Panak & Garber, 1992; Pomerantz, 2001; Robinson *et al.*, 1995; Spence *et al.*, 2002), one noted that this effect was stronger in adolescents than in children (Nolen-Hoeksema *et al.*, 1992), and 5 reported no evidence of a main effect of cognitive style on depression development (Abela, 2001; Hilsman & Garber, 1995;

Lewinsohn *et al.*, 1999; Lewinsohn *et al.*, 2001; Schwartz & Koenig, 1996). Two other studies attempted to evaluate the differential impact of various dimensions of participants' cognitive style (Hops *et al.*, 1990; Rudolph *et al.*, 2001). Both of these studies revealed that personal beliefs of control were related to lower levels of depression. Hops *et al.* (1990) study also identified irrational beliefs, self-consciousness, and low optimism as significant risk factors for depression. Yet, Rudolph *et al.*'s (2001) study showed that hopelessness did not predict depression, and Hops *et al.* (1990) obtained no relationship between negative attributional styles or self-reinforcing tendencies and depression.

Of the seven studies evaluating the diathesis-stress hypothesis in adolescents, three found that a negative cognitive style did not amplify the deleterious effects of stressful life events on depression development (Lewinsohn *et al.*, 1999; Schwartz & Koenig; 1996; Spence *et al.*, 2002) and two found direct support for the diathesis-stress hypothesis (Garber *et al.*, 2002; Hankin *et al.*, 2001). However, two additional studies yielded more confusing results. First, results from Robinson *et al.*'s (1995) study indicate that a negative cognitive style amplified the deleterious impact of stressful life events on depression, but only among low self-worth individuals and only during high school transition (a known stressful life event), but not 6 months later. Second, Lewinsohn *et al.*'s (2001) results suggest that a negative attributional style may play a role in depression development in the absence of stressors, rather than in their presence.

Two studies on children yielded support to the diathesis-stress hypothesis (Hilsman & Garber, 1995; Pomerantz, 2001) while one study obtained non-significant results (Panak & Garber, 1992). Moreover, both Abela's (2001) and Nolen-Hoeksema *et al.*'s (1992) studies, which evaluated this hypothesis in mixed child and adolescent samples, concluded that the diathesis-stress hypothesis may be more valid in adolescence than in childhood, possibly due to differences in cognitive development – children's cognitive capabilities being far less developed than adolescents'. This

hypothesis, however, should be considered with prudence as this result could also reflect the general inconsistencies of the results from studies on adolescents.

Three studies attempted to verify if the results regarding the diathesis-stress interaction varied according to subjects' gender. Abela (2001) revealed that, in 7th graders, self-blame amplified the effects of life events in girls only. But Hankin *et al.* (2001) identified such an interaction in boys only. Finally, Spence *et al.* (2002) reported no evidence of gender interactions.

It is interesting to note that most studies, although their results vary greatly, revealed some form of relationship (main effects or interactions) between cognitive style and depression development. Among the few exceptions in which no effects whatsoever were found, several were based on child samples (Abela, 2001, sample 1; Hilsman & Garber, 1995; Nolen-Hoeksema *et al.*, 1992, child subsamples). As previously noted, children's cognitive functions may not be sufficiently developed for them to be affected by cognitive factors. Only the Schwartz and Koening (1996) and Lewinsohn *et al.* (1999) studies yielded non-significant results in adolescents. In this latter study, the authors evaluated the potential role of attributional style in first onsets versus recurrences of depression using the Oregon Adolescent Depression Project data set. Whereas previous reports based on this data set suggested that attributional style may represent a significant predictor of depression (Lewinsohn *et al.*, 1994, 1995), these new analyses revealed that attributional style effects on depression development were limited to recurrences. As this is the only study evaluating the moderating role of previous levels of depression on the relationship between cognitive style and depression, these intriguing results deserve further exploration.

Summary

In summary, current evidence suggests that the direct effects of cognitive style on depression development, as well as its effects on stress reactivity, may be more potent in adolescence than in childhood. Results, however, remain inconsistent, and some

issues need to be investigated more thoroughly: (a) Do the results vary according to gender? (b) Is cognitive style a more potent predictor of first onsets or of recurrence? (c) Is attributional style a moderator, a mediator, or both, of the stress-depression relationship? (d) Do the effects of cognitive style vary according to the psychosocial domain (academic, social, etc.) to which such cognitions apply (for a related discussion, see Cole, Martin, Powers, & Truglio, 1996)? And (e) What is the specific role of cognitive style in young children's depression development and how is this role transformed according to developmental transitions?

Given the inconsistency of current results, it is interesting to note that Hankin and Abramson (2001) recently proposed a reformulated hopelessness theory of depression, which they call the "cognitive vulnerability-transactional stress theory." This theory extends the cognitive variables purported to act as depression diatheses to include, in addition to attributional style and dysfunctional attitudes, coping style, perceived physical attractiveness, and body image (reviewed in later sections). Moreover, they hypothesized that this cognitive vulnerability, which is determined by early adverse experiences in addition to preexisting vulnerabilities, interacts with negative life events to increase the normative initial negative affective consequences of stress and further interacts with these negative reactions to produce depression. In this theory, negative affectivity thus replaces the hopelessness component of the original hopelessness theory of depression. Unfortunately, the plausibility of this new proposition has not, to our knowledge, been systematically investigated. Moreover, this proposition also fails to take into account the previously cited results on the greater attributional realism shown by depressive individuals (e.g., Alloy *et al.*, 1990; Mezulis *et al.*, 2004; Taylor & Brown, 1988, 1994).

Self-Schemas and Attachment Styles

Theoretical Issues

Self-schemas

Schmidt, Schmidt, and Young (1999) define self-schemas as "mental templates, which are built up from historical experiences, [and] serve to create cognitive generalizations about the self as well as about social experiences" (pp. 127-128). Self-schemas thus represent "the basic structural components of cognitive organization through which humans come to identify, interpret, categorize, and evaluate their experiences" (Schmidt *et al.*, 1999, p. 129). According to Young (1990), the three defining characteristics of self-schemas are that they: (a) "selectively filter for corroborating evidence"; (b) "are self-perpetuating and highly resistant to change"; and (c) "rest at the core of the individual's self-concept, [and are] familiar, comfortable, and unconditional" (Schmidt *et al.*, 1999, p. 130).

Many theories have attempted to integrate personality and cognitive hypotheses of depression development by emphasizing the potential role of problematic self-schemas as stable, personality-like risk factors or diatheses for depression. For instance, according to Beck's (1983) theory, dysfunctional childhood experiences may give rise to two different kinds of vulnerable self-schemas, sociotropy and autonomy, both of which represent diatheses for depression. Furthermore, these two self-schemas are purported to be responsible for the emergence of the negative cognitive style and cognitive triad implicated in depression development when individuals are exposed to negative life events relevant to their self-schemas. Indeed, Beck (1983) proposes that sociotropic individuals are more likely to become depressed when they are exposed to interpersonal stressors, whereas autonomous individuals would be at risk when facing individual stressors. This theoretical proposal is generally referred to as the *stressor congruency* hypothesis. The main rationale behind this hypothesis is that individuals with problematic self-schemas tend to over-invest their self-esteem in a limited number of life domains (i.e.,

professional achievement or interpersonal relations) and thus present a risk of depression when environmental stressors disrupt these limited sources of self-worth.

Self-schema hypotheses gave rise to a considerable amount of research and scientific interest, in part because of the potential of such factors to bridge cognitive and personality theories of depression development. However, this interest also transformed this field of inquiry into what appears to be the most confusing area in depression research, for three related reasons.

First, several theories were formulated to emphasize the role of vulnerable self-schemas in depression development and few attempts were made to integrate these different views (for efforts in this direction, see Blatt & Zuroff, 1992; Coyne & Whiffen, 1995): sociotropy/autonomy (Beck, 1983), dependency/self-criticism (Blatt, 1990), socially prescribed/other-oriented/self-oriented perfectionisms (Hewitt, & Flett, 1993), interdependent/independent self construals (Cross, & Madson, 1997), communion/agency (Helgeson, 1994), femininity/masculinity (Bem, 1974), achievement/power/affiliation motivations (McClelland, 1987), and allocentrism/autocentrism (Gjerde, 1995). Interestingly, most of these definitions suggest the presence of two vulnerable self-schemas potentially implicated in depression development: an interpersonally oriented self-schema and an independently oriented one. The interpersonally oriented self-schemas emphasize excessive preoccupation with interpersonal relations, overinvestment of self-esteem in the quality of his/her social integration, and recurrent concerns about being disapproved of. Conversely, the independently oriented self-schemas emphasize excessive preoccupations with autonomy and personal gains, overinvestment of self-esteem in personal achievement, and concerns about losing control over one's life. A recent study based on Young's (1990) more detailed self-schema dimensions provides empirical support for both of these generic vulnerable self-schemas (Schmidt *et al.*, 1999).

Studies comparing the various operationalizations of these constructs also add to the confusion by revealing, for instance, a high level of interrelation between sociotropy, dependency, and self-criticism and the relative independence of autonomy (Blatt & Zuroff, 1992; Coyne & Whiffen, 1995). As autonomy and self-criticism are generally thought of as the same underlying construct, these results are intriguing. However, the fact that dependency and self-criticism definitions (Blatt, 1990) are themselves confusing - both constructs putting emphasis on individuals' excessive needs for others' approval - may explain this result.

Second, many studies found that both the interpersonally and the independently oriented self-schemas were closely related to Neuroticism and also presented more specific relations with other Big Five personality traits (Bagby & Rector, 1998; Cappeliez, 1993; Dunkley, Blankstein, & Flett, 1997; Mongrain, 1993; Zuroff, 1994). On the one hand, it is thus possible to argue that the vulnerable self-schemas hypothesis represents an interesting way to parsimoniously integrate at least four of the Big Five personality dimensions (excluding Extraversion, which was proposed to interact with dependency to predict depression: Barnett & Gotlib, 1988). On the other hand, it may also be argued that vulnerable self-schemas are nothing more than another way to look at the same variables (Coyne & Whiffen, 1995; Klein *et al.*, 2002).

The third source of confusion concerns the psychometric conceptualizations of both vulnerable self-schemas. For instance, they have sometimes been defined in a categorical manner (extreme levels are needed to label someone as "sociotropic") and sometimes in a dimensional manner (one can be more or less "sociotropic" and more or less "autonomous") (see Coyne & Whiffen, 1995). Yet, other authors placed these two problematic self-schemas at either ends of the same continuum and defined the mid-point as an adaptive state of equilibrium between self-focus and other-focus and the extreme points as problematic (Helgeson, 1994). Finally, others argued that an individual could also be extreme on both of these vulnerability factors (Coyne & Whiffen, 1995). Current evidence pinpoints a need for equilibrium between one's

interpersonal and independent self-orientations and places both self-schemas on separate continuums (Coyne & Whiffen, 1995).

Attachment

According to Bowlby (1958, 1969/1982), one of infants' main developmental tasks is to form an attachment relation with primary caregivers. This attachment bond is seen as an evolutive mechanism whose function is to encourage the mother-infant proximity (protection) and to provide the infant with a secure base from which to explore his/her environment. The quality of this attachment is further alleged to directly influence the infant's self-concept and to exert a lifelong influence on his/her interpersonal relationships. For this reason, insecure attachment styles have often been proposed as potential risk factors for depression development (Cummings & Davies, 1994; Cummings *et al.*, 2001; Goodman, 2002).

This proposition shares great similarity with self-schema theories, all of which emphasize the preeminent role of the parent-child relations as a key source of influence on vulnerable self-schema development (Blatt & Zuroff, 1992; Cross & Madson, 1997). It is thus interesting to note that two of the insecure attachment styles identified are very similar to the proposed problematic self-schemas (Zuroff, Moskowitz, & Côté, 1999): avoidant attachment resembles the independently oriented self-schema, anxious-ambivalent attachment bears similarity to the interpersonally oriented self-schema, and an insecure-disorganized attachment style would adequately characterize someone presenting a high level on both vulnerable self-schemas. Likewise, a secure attachment style, characterized by a combination of trust and autonomy, bears conceptual similarity to a state of adaptive equilibrium between both vulnerable self-schemas. Consequently, even if doubts can be cast on the real usefulness of self-schema theories, the fact that many classical theories (e.g., Beck, 1983; Bowlby, 1958; McClelland, 1987) came independently to similar conclusions is sufficient justification for further studies.

Empirical Support

Perhaps not so surprisingly, we located quite an explosion of studies designed to evaluate the role of independently and interpersonally oriented self-schemas and of their interactions with congruent stressors in depression development. Again, the main surprise was to find only six studies meeting our inclusion criteria, including those evaluating the role of attachment styles (Allgood-Merten *et al.*, 1990; Fergusson et Woodward, 2000; Hops *et al.*, 1990; Krueger, 1999; Reinherz *et al.*, 1993; Sund & Wichstrøm, 2002).

Among these studies, none evaluated the stressor congruency hypothesis and none was based on a sample of children. Among the studies based on adolescent samples, two found non-significant relations between masculinity/femininity and depression development (Allgood-Merten *et al.*, 1990; Hops *et al.*, 1990), one obtained non-significant relations between agency/communion and depression (Krueger, 1999), and one study showed that childhood levels of dependency were predictive of adolescent depression, but for boys only (Reinherz *et al.*, 1993). Yet, the last two studies, which focused on patterns of attachment (Fergusson & Woodward, 2000; Sund & Wichstrøm, 2002), revealed that attachment insecurity was predictive of later depression development. These highly inconsistent results could be due to the variability of the measures. For instance, whereas masculinity/femininity and agency/communion tests are designed to evaluate normative levels of self-schemas, dependency, and attachment insecurity scales are designed to tap more extreme patterns.

Summary

Research on the impact of problematic self-schemas on depression development has been marked by confusions regarding conceptual and psychometric questions. Emerging as a preliminary consensus are two vulnerable self-schemas, an interpersonally oriented one and an independently oriented one, best represented

along two separate continuums and providing a potential way to integrate many basic concepts such as personality, motivation, and attachment styles. Unfortunately, research results are still lacking and characterized by confusions and contradictions. Given the theoretical relevance of vulnerable self-schema theories and of their stressor congruency hypothesis, further research will need to verify the exact added value of these theories to our understanding of the mechanisms involved in depression development.

Self-esteem and Perceived Competencies

Self-Esteem

Theoretical issues

In accordance with classical psychoanalytical and humanistic theories and with the hypothesized negative cognitive triad of Beck's (1967, 1987) cognitive theory of depression, self-esteem has often been alleged to play a significant role in depression development either as a risk factor (low self-esteem) or as a protective factor against the deleterious effects of stressful life events (Barnett & Gotlib, 1988; Baron, 1993; Roberts & Monroe, 1999). The self-discrepancy theory (Hankin, Roberts, & Gotlib, 1997) proposes a further integration of self-esteem and cognitive theories of depression, indicating that an individual's sense of self subdivides into three dimensions: the *actual* self (what one is), the *ideal* self (what one wants to be), and the *ought* self (what one should be according to one's perception of others). According to this theory, depression results from a perceived divergence between the actual and ideal selves, whereas anxiety would result from an actual-ought selves discrepancy. Finally, whereas it is often viewed as a generic concept, self-esteem may also be conceptualized according to four dimensions (Roberts & Monroe, 1999): (a) self-esteem valence (positive or negative quality of generic self-perceptions); (b) self-esteem stability; (c) self-esteem resiliency to stress; and (d) self-esteem regulation strategies and processes (downward or upward social comparisons, self-esteem sources, etc.). To our knowledge, most of these refinements have not yet received

enough scientific attention aside from the preliminary evidence presented by Hankin *et al.* (1997) and Roberts and Monroe (1999).

Whereas many previous reviews (Albright, 1999; Lewinsohn & Essau, 2002; McCauley *et al.*, 2001; Roberts & Monroe, 1999) appeared to confirm the role of self-esteem as both risk and protective factors for depression development, recent critics are shedding doubt on these previously taken-for-granted results. For instance, some authors questioned the validity of the assumption that low self-esteem, which in itself represents a symptom of depression, could constitute a risk factor for something of which it is part (Coyne, 1999; Roberts & Monroe, 1999). An answer to this question can be obtained only in prospective longitudinal studies controlling for previous levels of depression such as those described in the present review, and thus eliminating the effect of the self-esteem/depression shared variance.

Empirical support

We located 12 studies which evaluated the role of self-esteem in depression development and met our inclusion criteria. Of those, four are based on child populations (Cole, Jacquez, & Maschman, 2001; Hoffman, Cole, Martin, Tram, & Seroczinski, 2000; McGrath & Repetti, 2002; Pomerantz, 2001) and eight on adolescent populations (Algood-Merten, Lewinsohn, & Hops, 1990; DuBois, Felner, Bartels, & Silverman, 1995; Hops *et al.*, 1990; Lewinsohn *et al.*, 1994; Palosaari, Aro, & Laippala, 1996; Robinson *et al.*, 1995; Siegel, 2002; Tram & Cole, 2000). Overall, these studies indicate that low self-esteem significantly predicts depression development in children and adolescents and that this relation is maintained even when previous levels of depression are controlled in the analysis.

Additional results deserve further attention. First, DuBois *et al.*'s (1995) study suggests that self-esteem may be a better predictor of moderate, rather than severe, depression. However, other studies using a categorical definition of depression did found a relation between self-esteem and depression (e.g., Lewinsohn *et al.*, 1994). Second, two studies found that self-esteem represented a protective factor against the

deleterious impact of stressors on depression development in late childhood (Pomerantz, 2001) and during high school transition (Robinson *et al.*, 1995), whereas another study failed to replicate this result in an adolescent population (Tram & Cole, 2000). However, the Tram and Cole (2000) study identified self-esteem as a significant mediator of the life event/depression relationship. These results, which certainly deserve further scientific attention, suggest that the mechanisms responsible for the effects of self-esteem may, like those of cognitive style, vary according to the developmental stage of the subjects. Third, Hoffman *et al.* (2000), in a more complex study, attempted to evaluate the mechanisms underlying self-esteem effects on depression development. To this end, they asked respondents to evaluate their own competencies in multiple domains and compared these evaluations with others' evaluations. Intriguingly, they found that both reflected (coherent with others' evaluations) and discrepant (discordant with others' evaluations) self-evaluations were related to higher levels of depression. The authors attribute the "reflective self-evaluation" results to the fact that subjects may tend to internalize others' negative evaluations better than positive ones. Accordingly, they propose that the main predictor of depression should be negative self-evaluations (low self-esteem) instead of the discrepant or reflective character of such evaluations. Clearly, more studies are needed on this topic. Finally, the only study failing to report a relation between generic self-esteem and depression included, among the predictors, subjects' self-perceptions of their academic and social self-competencies (McGrath & Repetti, 2002). This last result suggests, in conformity with Roberts and Monroe's (1999) proposal, a need to devote more attention to self-esteem sources in depression development research and to the fact that the effects of generic self-esteem may be secondary to those of domain-specific self-evaluations.

Perceived Competencies

Theoretical issues

As mentioned, knowing that negative self-esteem per se predisposes an individual to depression does not answer the basic question of the mechanisms involved in such an

effect. Self-esteem is a very generic concept, representing a *gestalt* of many dimensions and potentially applying to various life domains. Harter's (1985 a, 1985 b) conception of competency dimensions allows a far more detailed view of these questions and provides an answer to one of the questions left unanswered in the cognitive section: Do the effects of self-esteem or self-cognitions vary according to the psychosocial domain to which they apply? Harter (1985 a, 1985 b) proposes that youths' perceived competencies can be grouped into five basic domains of competence: (a) academic; (b) social; (c) body image; (d) sports (or athletic); and (e) behavioral (conduct).

Empirical support

We located 13 studies meeting our inclusion criteria that evaluated the direct role of perceived competencies in depression development (Bandura, Pastorelli, Barbaranelli, & Caprara, 1999; Choi, Patten, Gillin, Kaplan, & Pierce, 1997; Cole, Martin, Peeke, Seroczinski, & Fier, 1999; Cole, Martin, Peeke, Seroczinski, & Hoffman, 1998; Hilsman & Garber, 1995; Holsen, Kraft, & Røysamb, 2001; Lewinsohn *et al.*, 1994; McFarlane, Bellissimo, & Norman, 1995; McGrath & Repetti, 2002; Siegel, 2002; Stice & Bearman, 2001; Stice, Hayward, Cameron, Killen, & Taylor, 2000; Stice, Presnell, & Bearman, 2001). Only one of those also tested potential interactions with stressors in explaining the emergence of depression in youths (Hilsman & Garber, 1995). Most studies assessing the effects of perceived academic (Bandura *et al.*, 1999; Hilsman & Garber, 1995; Lewinsohn *et al.*, 1994) or social competencies (Bandura *et al.*, 1999; McFarlane *et al.*, 1995) noted that low scores on these variables were associated with increased levels of depression. Hilsman and Garber's (1995) results even indicate that perceived academic competence may serve as a protective factor against the negative effects of grade-related stressors (e.g., getting low grades). The only exception is Choi *et al.*'s (1997) study, in which the authors reported no relation between perceived academic competence and later depression. However, this discrepant result could be because this study relied on Kandel and Davies' (1982) limited 6-item depression scale.

McGrath and Repetti's (2002) results, however, indicate that in children, such results could vary according to age. For instance, the authors found that whereas academic self-concept in fourth grade was related to depression in fifth grade, this relation did not hold for sixth grade depression. Nevertheless, they showed that social self-concept in fourth grade did significantly predict sixth grade depression. No relation was noted between self-concept in fifth grade and depression in sixth grade. The authors also verified whether the discrepancy between self-rated competence and extraneous evaluations (teacher rating of social competence and grade point average) was related to depression development. The only significant result was that social self-concept distortions in fourth grade predicted sixth grade depression. Consistent with this result, Cole *et al.* (1999) observed that academic competence overestimation (relative to teachers' evaluations) was predictive of later depression in 3rd, 6th, 7th, and 8th grade, but not in 4th or 5th grade. However, in a previous study in which they used "objective" ratings created from a combination of peers' and teachers' ratings on the five domains of Harter's scale (Harter, 1985 a), Cole *et al.* (1998) found no evidence that distorted self-evaluations of academic, social, and sports competencies were related to later depression. However, they observed significant relations between distorted self-evaluations of behavioral conduct and physical appearance in adolescents (7th and 8th grade) and depression development. One may explain this result by the fact that appearance and behavior are probably the only domains on which peers' and teachers' evaluations could be expected to agree: peers are probably better raters of sports competence and social acceptance, whereas teachers are probably better evaluators of academic performance. Most certainly, more studies are needed on this topic.

Studies estimating the role of body image satisfaction in depression development generally agreed that a negative body image was related to increased levels of depression in adolescent girls (Holsen *et al.*, 2001; Siegel, 2002; Stice & Bearman, 2001, Stice *et al.*, 2000, 2001), but also that these effects were deeply intertwined with those of dieting and thin-ideal internalization. However, with one exception (Stice *et al.*, 2001), body weight was not found to predict depression, suggesting that

body image effects are almost entirely social and cognitive in nature. In an attempt to integrate most of these factors, Stice and Bearman (2001) revealed that perceived pressure to be thin was interrelated with the internalization of a thinness ideal, which was predictive of body image dissatisfaction. Negative body image was then related to dieting and/or bulimic symptoms, which were in turn predictive of depression development. Other results suggest that these two variables may even act as complete mediators of the body image-depression relationship (Stice *et al.*, 2000). Finally, it should be noted that, whereas most scholars concluded that the role of body image in depression development was limited to girls, Holsen *et al.*'s (2001) results suggest otherwise. For instance, they revealed that negative body image at age 13 was related to depression at age 15 in girls only, whereas negative body image at age 15 was related to depression at age 18 for boys only.

Summary

In summary, many studies converge to indicate that generic self-esteem as well as perceived competencies in specific domains are negatively related to depression development. Furthermore, some studies propose that positive self-esteem may even protect youths against the deleterious effects of stressors. In an additional "regression tree" cross-sectional analysis of their sample, Seroczynski, Cole, and Maxwell (1997) even concluded that a positive self-view in some domains may compensate for negative self-evaluation in other domains. Although more studies are needed, these results pinpoint that interventions targeting positive self-concept development in multiple domains may be an efficient way to prevent depression. The specific case of body image has received more attention and may serve to explain part of the gender difference in depression development. In fact, most studies suggest that puberty-related body changes, combined with societal ideals of thinness, may lead to body image dissatisfaction in girls and that this dissatisfaction predicts later increases in girls' depression, in part through dieting behaviors. Known for their general inefficiency, dieting behaviors may lead to a form of "physical hopelessness," which in turn could explain depression development.

Some other results suggest an interesting way to integrate cognitive theories of depression and self-concept research. Indeed, it has been hypothesized that distortions in self-evaluations may be related to depression development. Although more studies are needed on this topic, these results underline that the effects of self-concept on depression development may be mediated through at least three forms of social processes. First, overestimation of one's competence may lead to risky enterprises and to failure accumulation, whereas underestimation may limit someone to small, under-stimulating achievements. Second, Hoffman *et al.*'s (2000) results suggest that it is the internalization of other negative perceptions that leads to depression rather than the adequacy of self-evaluations. A third perspective could be to question the adequacy of teachers' and peers' reports on students' competencies, especially because at-risk individuals have often been found to be socially aversive to others (Joiner & Coyne, 1999). In this context, the over- or under-estimation of competencies could lie within the evaluators, and the risk of depression could be related to the subject's knowledge of others' misperceptions. Future studies should attempt to disentangle these three hypotheses.

Many other questions remain. First, the possibility that the results could be moderated by gender and developmental stage should be more thoroughly investigated. For example, some studies suggest that the role of self-esteem as a protective factor may be stronger in childhood (Pomerantz, 2001; Tram & Cole, 2000), whereas this role could also depend on the individual's cognitive style in adolescence (Robinson *et al.*, 1995). Secondly, Roberts and Monroe's (1999) definitions of self-esteem dimensions should be taken into account in future research. For example, the role of self-esteem stability and resiliency could easily be integrated to previously reported findings on the role of neuroticism (emotional instability) in depression. Similarly, if research on multiple perceived competencies is coherent with Roberts and Monroe's (1999) suggestion that self-esteem regulation strategies should be studied, sources of self-esteem constitutes only one of the possible ways to address this suggestion.

Behavioral Competencies and Coping Style

Behavioral Competencies

Theoretical issues

Knowing that negative evaluations of one's competencies and discepancies between self-evaluation and evaluations by others may lead to depression does not help us to understand the precise mechanisms underlying such effects. For instance, does negative evaluation of one's competencies predict depression through "pure" cognitive pathways or are such evaluations related to real-life behavioral problems? Repeated suggestions of more adequate and less biased self-evaluation in depressed persons lend support to the second hypothesis (e.g., Alloy *et al.*, 1990; Mezulis *et al.*, 2004; Taylor & Brown, 1988, 1994). For intervention and prevention purposes, such questions are of major relevance: Should we increase at-risk persons' real-life competencies or should we restructure their biased evaluations of such competencies?

The interactional perspective of depression (Coyne, 1976; Joiner & Coyne, 1999) lends support to the need to evaluate the effects of behavioral competencies, particularly social skills, in depression development. Indeed, this perspective proposes that depression-prone individuals exhibit a problematic interpersonal style, characterized by a paradoxical combination of reassurance- and negative feedback-seeking and problematic communication styles (i.e., negative speech content, slow pace and long pauses, less vocal tone modulation, etc.) (Coyne, 1976; Joiner & Coyne, 1999). This negative interpersonal style may lead others to develop negative views of the depression-prone person and to modify their interaction with him/her, thus decreasing the individual's level of social support and increasing his/her exposure to interpersonal stressors. As a result, such individuals will be exposed to higher levels of interpersonal stressors, as well as loneliness and alienation, which will in turn affect their ability to cope efficiently with stressors, thus producing depression. In summary, this perspective clearly proposes that social-skill deficits represent the first part of a long chain of mediators eventually leading to depression

development. If the original interpersonal theory attempted to explain depression maintenance, this framework was recently expanded to depression development (Joiner & Coyne, 1999).

Empirical support

We found 11 studies evaluating the prospective relation between behavioral competencies and depression development. It should be noted that only one of those included a child sub-sample and that this study showed no evidence of social or academic competencies playing a role in depression development (Cole *et al.*, 1996). When it comes to the role of academic and social competencies in the development of adolescents' depression, the results are more complex. For instance, among the nine studies evaluating the role of generic academic competencies (or related concepts) in depression development, three found significant negative relations (Chase-Lansdale, Cherlin, & Kiernan, 1995; Fergusson & Woodward, 2000; Roeser & Eccles, 1998), and seven yielded non-significant results (Bandura *et al.*, 1999; Cole *et al.*, 1996; DuBois *et al.*, 1995; Lewinsohn *et al.*, 1994, 1995; Reinherz *et al.*, 1993; Rosenfield, Vertefeuille, & McAlpine, 2000). Moreover, among the studies reporting significant relations, Chase-Lansdale *et al.* (1995) observed such a relation for depressive symptoms only, failing to replicate their results with a categorical diagnosis of depression. Such results are consistent with the conclusions from Flemming and Offord's review (1990) and indicate few relations between academic achievement and depression development. However, these conclusions should be viewed prudently, as Chase-Lansdale *et al.* (1995) did report an association between academic achievements at age 7 and depression in young adulthood, suggesting that a potential chain of mediators may be involved in these effects. Fergusson & Woodward (2000) also identified a long-term relation between academic achievement at age 12 and depression at age 18.

Lewinsohn *et al.*'s (1994, 1995) results deserve further attention as they distinguish the effects of more specific aspects of academic competencies on adolescent depression development. Thus, in their analyses, the authors showed that students

who did not regularly complete their homework presented an increased risk of developing depression. Yet, they noted non-significant relationships between depression and school failures, truancy, and lateness. Clearly, more long-term follow-up studies will be needed on this point.

Finally, among the six studies focusing on the relation between social skills and depression development in adolescents, two obtained evidence of a predictive role of social-skill deficits and related concepts (i.e., shyness) (Cole *et al.*, 1996; Segrin & Flora, 2000) and four yielded non-significant results (Bandura *et al.*, 1999; DuBois *et al.*, 1995; Lewinsohn *et al.*, 1994; Reinherz *et al.*, 1993). Once more the evidence suggests an absence of relation between social skills and depression. However, this conclusion should also be viewed with prudence. Indeed, Segrin and Flora's (2000) results propose that, while the main effects of social skills on depression development are quite small, such skills may act as a protective factor in the presence of adversities, lending partial support to the interpersonal theory of depression. Besides, Bandura *et al.*'s (1999) study suggests yet another hypothesis by demonstrating that the effects of behavioral competencies on depression development tend to disappear once the adolescents' perceived self-competencies are taken into account, indicating that the "active ingredient" could in fact be cognitive self-appraisals. Finally, none of these studies report any evidence of gender-based variability in the results.

Coping Style

Theoretical issues

Coping style represents a more specific form of competency, referring to "effortful or purposeful thoughts and actions undertaken in an attempt to manage or overcome stressful situations and the negative emotions associated with them" (Compas, 1995, p. 255). At least three generic coping styles have been identified (Endler & Parker, 1999): (a) problem-solving or task-focused coping, which refers to purposeful attempts to solve, alter, or cognitively restructure the problem through action and planning; (b) emotion-focused coping, which refers to self-oriented emotional

reactions aiming to reduce the perceived stressfulness of the situation; and (c) distraction coping, which refers to cognitions and activities aimed at avoiding the stressful situation, either through behavioral avoidance or through social-support seeking. The relative efficiency of these coping styles is alleged to vary according to the nature of the stressors facing the person (Compas, 1995; Coyne & Downey, 1991). For instance, while distraction coping would be most efficient for dealing with uncontrollable stressors, task-focused coping would be more relevant to controllable ones. Moreover, perceived ability to deal with stressors may also influence the use of task-focused versus avoidant coping (Compas, 1995).

A specific form of emotion-focused coping, rumination, have been proposed to be particularly relevant to depression development (Nolen-Hoeksema, 1987, 2002). Literally, rumination is a process of preservative attention directed toward specific, often internal, content and generally results in increased influence and salience of the content and in "sticky attention," where one becomes unable to focus on anything else (Abramson *et al.*, 2002). Relative to depression, rumination refers to self-focused, recurrent, and unproductive attention centered on one's negative mood states, resulting from negative life event exposure, and is alleged to amplify these mood states and to result in depression when the attention cannot be disengaged from such self-centered attention (Mor & Winquist, 2002).

Previous reviews on coping and depression suggest that rumination, emotion-focused, avoidant, and generic "maladaptive" coping (i.e., drug use, isolating oneself, confrontation, etc.) may indeed contribute to depression development, whereas adaptive coping and problem-solving skills may diminish depression risk (Compas, 1995; Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001, Compas, Langrock, Keller, Merchant, & Copeland, 2002). Regarding the precise role of rumination, Mor and Winquist's (2002) extensive meta-analysis established a distinction between two types of ruminative coping: private self-focus and public self-focus. A clear parallel could be made between these results and the previously reviewed self-discrepancy theory, private self-focus implying one's focus on one's

own deficiencies (actual-ideal self discrepancy) and public self-focus centering on one's perceived social inadequacy (actual-ought self discrepancy). Mor and Winquist's (2002) results revealed a clear effect of rumination on depression. They also showed that while private self-focus was more specifically related to depression, public self-focus was a better predictor of social anxiety.

Empirical support

We located seven studies in which the role of coping style on depression development was specifically evaluated, all of which are based on adolescent samples (Adams & Adams, 1993; Hops *et al.*, 1990; Lewinsohn *et al.*, 1994, 1995; Swartz & Koenig, 1996; Seiffge-Krenke & Klessinger, 2000; Spence *et al.*, 2002). Given the outstanding number of studies published on individual risk factors for depression and the potential key role of coping in environmental adaptation, it is surprising that so few studies focused on the effects of coping on depression development. Two of these studies evaluated the impact of problem-solving skills on depression development (Adams & Adams, 1993; Spence *et al.*, 2002), and both concluded that such skills did moderate the stressor-depression relationship. Indeed, Adams & Adams (1993) revealed that when facing a drop in academic grades, adolescents who perceived more problem-solving alternatives were protected against depression. In a related way, Spence *et al.* (2002) showed that adolescents facing negative life events had higher levels of depression at follow-up if they had a more negative problem-solving orientation.

Problem-solving orientation also had a significant main effect on depression development. Two other studies, based on the same sample, reported a negative relation between generic coping skills and depression development and a positive relation between emotion-focused coping and depression (Lewinsohn *et al.*, 1994, 1995)., Hops *et al.* (1990), however, failed to find a relation between generic coping skills and depression development. Finally, Seiffge-Krenke and Klessinger (2000) noted that the tendency to rely on avoidant coping predicted increases in depressive symptoms, and Swartz and Koenig (1996) obtained a positive relation between

rumination and later depression in females, but not males. They found no evidence, however, of a relation between distraction coping and depression or of an interaction between coping styles and stressors. It should be noted that Swartz and Koenig's (1996) study is the only one reporting any verification of gender-based variability in the results.

Summary

Although there are strong theoretical bases to support the role of behavioral competencies and coping style in depression development, very few of the studies meeting our inclusion criteria have attempted to verify these hypotheses. Moreover, these studies generally yielded inconsistent results. To date, there exists little evidence that social skills or academic competencies play a role in depression development, although some preliminary results suggest that such competencies may act as protective factors in the presence of stressors or that the relations may be long-term ones involving a potentially long chain of mediators. Furthermore, some results suggest that the tendency to rely on problem-solving coping may decrease the risk of depression development, particularly in the presence of stressors, and that ruminative or avoidant coping styles may increase this risk, regardless of exposure to stressors. We found no studies evaluating the role of the hypothesized contextual variation in coping efficiency and no studies considering the impact of the three proposed coping styles together or evaluating the recent conceptualization of voluntary and involuntary coping mechanisms proposed by Compas *et al.* (2001, 2002). Moreover, because very few of these studies were based on child samples, we cannot evaluate the developmental variability of the effects.

Previous Disorders

Theoretical Issues

The high level of comorbidity reported between child and adolescent depression and other psychosocial disorders (Angold & Costello, 2001; Angold *et al.*, 1999; Compas *et al.*, 1998; Kessler, 2002; Lewinsohn & Essau, 2002), such as externalized

behavioral problems (conduct disorders, oppositional-defiant disorders, etc.), anxiety-related problems, substance and alcohol abuse, personality disorders, and attention-deficit/hyperactivity (ADD/H), led many researchers to analyze the nature of the relations between these disorders. For instance, a recent integrated analysis of comorbidity led Angold *et al.* (1999) to conclude that whereas comorbidity between depression and anxiety or externalizing behavior problems was "real," the ADD/H-depression relation was an artifact of the comorbidity of these two disorders with externalizing problems (epiphenomenal comorbidity). Many hypotheses have also been suggested for bidirectional relationships between these multiple disorders. For instance, substance abuse may represent an attempt to self-medicate depressive symptoms and thus a consequence of depression (Lynskey, 1998). However, the varied adverse psychosocial consequences of substance abuse may themselves lead to depression (Lynskey, 1998). Insofar as depression development is considered, it can also be hypothesized that the adverse social consequences of externalizing behaviors, ADD/H, anxiety, and personality disorders may lead to depression (Kovacs & Devlin, 1998) or that the prolonged state of arousal associated with anxiety may deplete the biopsychosocial resources of the person and trigger depression (Zahn-Waxler *et al.*, 2000). This last suggestion has been called the *resource depletion hypothesis.*

Empirical Support

We found 28 studies meeting our inclusion criteria that evaluated the predictive role of other disorders in depression development, 4 of which were based on child samples (Khatri, Kupersmidt, & Patterson, 2000; Lynch & Cicchetti, 1998; McGrath & Repetti, 2002; Panak & Garber, 1992), 18 on adolescent samples (Bandura *et al.*, 1999; Choi *et al.*, 1997; Daley, Hammen, Davila, & Burge, 1998; Daley, Hammen, & Rao, 2000; Davies & Windle, 2001; DuBois *et al.*, 1995; Ge, Best, Conger, & Simons, 1996; Hops *et al.*, 1990; Kiesner, 2002; Lewinsohn *et al.*, 1994, 1995; Pine, Cohen, & Brook, 2001; Pine, Cohen, Gurley, Brook, & Ma, 1998; Rao, Hammen, & Daley, 1999; Robinson *et al.*, 1995; Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001; Sears & Armstrong, 1998; Windle & Windle, 2001), and 6 on long-term

follow-up samples (Capaldi & Stoolmiller, 1999; Fergusson & Woodward, 2000; Jaffe *et al.*, 2002; Kasen *et al.*, 1996; Krueger, 1999; Reinherz *et al.*, 1993).

We located only two studies on personality disorders, one of which demonstrated no relation between hypomanic personality and later depression development (Lewinsohn *et al.*, 1994) and one which showed that the relation between DSM-IV clusters A and B personality disorders and depression development was completely mediated by the impact of these problems on chronic and episodic stress (Daley *et al.*, 1998). Next, only three of these studies evaluated the potential role of ADD/H in depression development. One of them observed a relation between hyperactivity (temperamental hyperactivity) and juvenile onset depression (Jaffe *et al.*, 2002), while the other two found no evidence of such a relation in adolescents (Fergusson, & Woodward, 2000; Pine *et al.*, 1998). Many studies (19), however, did verify the role of externalizing behaviors problems in depression development and most of them converge in suggesting that such problems do indeed predict later depression (Bandura *et al.*, 1999; Choi *et al.*, 1997; Davies & Windle, 2001; Fergusson & Woodward, 2000; Hops *et al.*, 1990; Jaffe *et al.*, 2002; Kasen *et al.*, 1996; Kiesner, 2002; Krueger, 1999; Lewinsohn *et al.*, 1994, 1995; Lynch & Cicchetti, 1998; Panak & Garber, 1992; Robinson *et al.*, 1995). Only six of these studies obtained non-significant relations (Capaldi & Stoolmiller, 1999; DuBois *et al.*, 1995; Ge *et al.*, 1996; Khatri *et al.*, 2000; McGrath & Repetti, 2002; Windle & Windle, 2001). These diverging results could be easily explained by differences in measurement methods (i.e., peer-reported aggression in Khatri *et al.*'s study) and controlled factors (i.e., inclusion of many drug-related behaviors, in Windle & Windle's study), or by sampling variability (i.e., a more deprived sample in Ge *et al.*'s study). It therefore appears reasonable to conclude that current evidence indicates that externalizing behaviors do indeed predispose to later depression development.

Few studies evaluated the role of drug/alcohol use and abuse in depression development. Of those that did, one found no evidence that psychoactive substance *use* was related to subsequent depression development (Fergusson & Woodward,

2000), one indicated that substance *abuse* did predict later depression (Rohde *et al.*, 2001), and one concluded evidence that *marijuana use* was related to lower levels of depression and that the *use of other substances* had no effect on depression development (Windle & Windle, 2001). Such results suggest that while drug or alcohol *abuse* may engender depression, *using* them reasonably presents no discernible impact on depression development. If these results could be replicated, they would provide strong support to the current harm reduction approaches (versus abstinence-based ones) in substance abuse prevention (Paglia & Room, 1999).

Among the studies evaluating the predictive role of anxiety-related disorders in depression development, eight studies established such a relation (DuBois *et al.*, 1995; Hops *et al.*, 1990; Jaffe *et al.*, 2002; Lewinsohn *et al.*, 1994, 1995; Pine *et al.*, 1998, 2001; Reinherz *et al.*, 1993), and only one study reported non-significant results (Sears & Armstrong, 1998). However, many studies indicate that the precise relationship between anxiety and depression may vary according to the subtype of anxiety disorder. For instance, Hops *et al.* (1990) found that whereas "tension" predicted subsequent depression development, "anxiety" did not. In a related way, Pine *et al.* (1998) noted that "overanxiety" (i.e., generalized anxiety) constitutes a risk factor for depression while simple and social phobia, separation anxiety, and fearful spells did not. It remains to be seen whether these last results represent an artifact of the comorbidity of the different anxiety disorders. Finally, Reinherz *et al.* (1993) reported significant age variations in their results. Indeed, these authors indicated that while childhood anxiety problems do not predict adolescent depression, adolescent anxiety does.

Whereas Hops *et al.*'s (1990) study indicated that previous suicidal *ideation* does not predict subsequent depression development, Lewinsohn *et al.* (1994, 1995) revealed that previous suicide *attempts* were related to later depression. These results may indicate that the severity of suicidal behaviors is implicated in their effects on depression. However, given that depression represents a very significant predictor of suicidal behaviors and is highly stable, this relationship should be evaluated by

distinguishing the effects of suicidal behaviors on onsets versus recurrences of depression. Finally, three studies exposed a relation between generic "non-mood disorders" (Daley *et al.*, 2000; Rao *et al.*, 1999), "other disorders" (Lewinsohn *et al.*, 1994), and "risk behaviors" (Sears & Armstrong, 1998) and depression development.

Summary

Relations between previous disorders and later depression development seem fairly established. The clearest evidence concerns the role of externalized behavioral problems and of anxiety-related problems in depression development, although the mechanisms underlying these effects are still unknown. But the evidence concerning ADD/H and substance use and abuse is still inconsistent and would require further studies. Recent results suggesting that reasonable substance use may have no effect on depression development most certainly deserve further attention. Regarding personality disorders, current evidence remains preliminary and suggests that the effects of such problems may be completely mediated by their impact on stress exposure. Overall, prospective longitudinal studies evaluating the precise role of other psychosocial disorders on later depression development are clearly needed. Moreover, these studies should attempt to distinguish the effects of such problems on first onset versus recurrence of depression and should devote more attention to the evaluation of the specific risk mechanisms underlying the observed effects as well as their age and gender-related variations.

Other Possible Individual Antecedents

Intelligence and Motor Development

Given the preeminent role that intelligence has been found to play in the development of many behavioral problems and in general adaptation (e.g., Gottfredson, 1997 a, 1997 b; Smith, 1995), it is surprising that so few studies have attempted to evaluate the relation between intelligence and depression development. Overall, we located

only two relevant studies meeting our inclusion criteria (Fergusson & Woodward, 2000; Jaffe *et al.*, 2002). Both of these studies failed to identify a significant relation between general intelligence and depression development. However, Jaffe *et al.* (2002) observed that poor motor skill development at ages 3 to 9, a precursor of intelligence, was a significant predictor of later depression. Clearly, more studies are needed. Some theories may however provide preliminary hypotheses for such studies. For instance, Sternberg (1985) defines intelligence as a capacity to adapt to, to shape, and to select one's environment. Therefore, as environment is clearly implicated in depression development (see the following section), it is probable that intelligence plays a role in one's adaptation to the environment. Intelligence may thus moderate the relationships between environmental risk factors and depression development.

Price's social competition hypothesis of depression provides an additional perspective on this topic (Gardner & Price, 1999; Price, 1998; Price, Sloman, Gardner, Gilbert, & Rohde, 1994). According to Price's hypothesis, depression occurs when an organism refuses to consciously yield in the face of failure and is caused by evolutive automatic mechanisms designed to ensure one's survival through energy conservation. A combination of Sternberg's and Price's perspectives may thus lead to the hypothesis that intelligent individuals may be better equipped to deal with environmental challenges and thus, less likely to fail in the face of challenging situations or more likely to adapt to failure situations. However, when such individuals also present dysfunctional self-schemas, like a high level of interpersonal dependency, and the particular challenge involves failure relevant to this specific vulnerability, like a romantic breakup, they may decide not to accept the hopeless character of the situation. Consequently, intelligent persons may end up devoting a very high level of resources to change hopeless situations. The relationship between depression and intelligence may therefore be curvilinear: lower intelligence levels could be related to higher levels of depression (difficulty to shape or adapt to the environement) and higher levels may also be related to depression through moderating relations involving self-schemas and stressors. This hypothesis also provides an alternate interpretation to the non-significant linear relations observed in previous studies.

Health and Health-Related Problems

Given the major disruptions in life habits that may come from serious health problems, such problems have been invoked as a potential risk factor for depression development (Bennet, 1994). For instance, diseases such as leukemia may involve drastic changes in family relations by reducing the amount of shared family activities, in which the child is no longer able to participate. In contrast, diseases such as cystic fibrosis may not present such negative effects, being intimately associated with an increase in family support and shared activities, which are part of the treatment. A meta-analysis on this topic certainly suggests that the effects of illness on depression may vary according to the specific nature of the health problem (Bennet, 1994). More precisely, Bennet's (1994) results show that while an increased risk of depression may come from diseases such as asthma, recurrent abdominal pain, sickle cell anemia, and inflammatory bowel disease, other diseases such as cancer, cystic fibrosis, and diabetes mellitus do not seem to carry any risk of depression. According to Bennet's (1994), disease severity could moderate these effects. Unfortunately, Bennet's (1994) meta-analysis is based mostly on cross-sectional studies or on studies in which no control was provided for previous levels of depression.

In the present review, four studies meeting our inclusion criteria that evaluated the predictive role of health-related problems on depression development were located (Lewinsohn, Seeley, Hibbard, Rohde, & Sack, 1996; Lewinsohn *et al.*, 1994, 1995; Reinherz *et al.*, 1993), three of which are based on the Oregon Adolescent Depression Project (OADP) data set. Generic analyses of the OADP revealed that whereas objective physical health and lifetime number of physical problems represented significant risk factors for adolescent depression even in multivariate analyses including many known risk factors, self-rated physical health, functional difficulties, medication, and hospital stays did not (Lewinsohn *et al.*, 1994; 1995). Additional analyses focusing solely on health-related problems revealed that diseases and functional impairments significantly predicted later depression, whereas injuries and reduced activity did not (Lewinsohn *et al.*, 1996). This last result appears to support

Bennet's (1994) suggestion that the severity of health problems and associated impairments were significant moderators of the disease-depression relationship. Conversely, Reinherz *et al.* (1993) found that health problems in young ages (birth to age 6) were significant predictors of adolescent boys' depression, whereas later problems (ages 10 to 15) were only related to adolescent girls' depression. Although the underlying mechanisms remain unclear, these results pinpoint different developmental trajectories for boys and girls and deserve replication.

In a related way, we were able to locate seven studies evaluating the role of health-related behaviors, such as smoking and exercising, in adolescent depression development (Brown, Lewinsohn, Seeley, & Wagner, 1996; Choi et al, 1997; Fergusson & Woodward, 2000; Lewinsohn *et al.*, 1994, 1995; Stein *et al.*, 1996; Windle & Windle, 2001). Whereas some studies found no relation between cigarette use per se and depression (Fergusson & Woodward, 2000; Lewinsohn *et al.*, 1994), other studies agree that cigarette smoking increases the risk of later depression and that this risk is a function of the quantity of tobacco consumption, tobacco use being worse when heavy rather than occasional (Brown *et al.*, 1996; Choi et al, 1997; Stein *et al.*, 1996; Windle & Windle, 2001). The relation between cigarette consumption and depression also appears stronger for girls than for boys (Lewinsohn *et al.*, 1995). Regarding the role of exercising in depression development, Lewinsohn *et al.* (1994) failed to identify significant relations between lack of exercise or obesity and depression development. Choi *et al.* (1997), however, observed that participating in organized sports did reduce males' risk of depression. It remains unclear if this effect comes from the "athletic" or "organized" part of the activity.

Summary

In addition to the previously reviewed antecedents of depression development, which were all highly psychological and behavioral (cognitions, personality, and disorders), other potential risk factors were also proposed. The main difference between these factors (intelligence, health behaviors) and the previous ones is that, in theory, their

relation with depression development is deeply interrelated with environmental factors. For instance, theoretical propositions suggest that the effects of intelligence on depression development stem from the impact of intelligence on youth's adaptation to their environment. Similarly, the impact of health problems on depression development is generally seen as completely mediated by the impact of illness on social relationships with family members. This difference, given the long-lasting individual focus in depression research, may explain the relative lack of attention devoted to these factors. Interestingly, other factors of this kind, such as religiosity and religious behaviors (Smith, McCullough, & Poll, 2003), sexual orientation (Cochran, Sullivan, & Mays, 2003), musical preferences (Miranda & Claes, 2003 a) and behaviors (Miranda & Claes, 2003 b), and amount of Internet use (Kraut *et al.*, 1998) have also been proposed as playing a role in depression development, although knowledge of their effects awaits prospective evaluation in samples of youths. Overall, current results suggest that physically disabled or unhealthy youths and heavy tobacco smokers may present an increased risk of developing depression, whereas those involved in sports may be at reduced risk. However, it has been suggested that these relations may depend on subjects' age and gender, although no clear pattern has yet emerged on these topics. Clearly, these factors deserve further scientific attention. The same comment applies to the effect of intelligence on depression. While some theories justify the need to devote more attention to the role of intelligence in depression development, more studies will be needed before conclusions can be reached.

SOCIAL ANTECEDENTS

Generic Life Events

Theoretical Issues

Generic stressful life events, or stressors, are one of the most studied social risk factors for depression. Garmezy (1986, p. 298) defines stressors as: "(1) the presence

of a stimulus event, which induces (2) an element of change that modifies the organism's systemic and/or psychological equilibration, and (3) is capable of inducing a state of emotional arousal marked by concomitant neurophysiological, cognitive, and expressive components, which (4) has the potential of disrupting the organism's normative pattern of responding." In summary, stressors are events that may disturb an individual's state of biopsychosocial equilibrium by taxing his/her adaptive resources (Coyne & Downey, 1991). Early studies of stressor effects on depression supported this view and showed that such effects depended on their congruence with an individual's personal vulnerabilities and appraisals (e.g., Brown & Harris, 1978).

In stressor-oriented research, two types of stressors are usually distinguished (Kessler, 1997; Monroe & Simons, 1991): stressful life events, characterized by great severity and episodic nature; and chronic stressors/daily hassles, characterized by their lesser severity and longer duration. Some scholars, referring mostly to stressful life events, pointed out the need to distinguish among personal disappointments, losses, danger to self, and danger to others (Goodyer, 2001), or to differentiate stressful life events caused by the exposed person (dependent) from those caused by extraneous factors (independent) (Coyne & Downey, 1991).

Previous reviews generally concluded that exposure to stressful life events, particularly those involving losses and disappointments, and chronic stressors, particularly interpersonal ones, increased the risk of becoming depressed (Baron, 1993; Coyne & Downey, 1991; Garber & Horowitz, 2002; Goodyer, 2001; Kessler, 1997; Kessler *et al.*, 2001). Likewise, it is generally recognized that females may be more strongly affected by interpersonal events and events affecting others in their social networks, whereas men may be more affected by achievement-related events (Bebbington, 1996; Nolen-Hoeksema, 2002). The latter assumption, first proposed by Kessler and McLeod (1984) as the *cost of caring hypothesis*, states that females' greater reliance on social interactions and greater levels of empathy make them more sensitive than men to network stressors (stressors affecting or involving members of

their social networks, such as the illness of a friend, the divorce of a sister, or interpersonal conflicts).

Another generally accepted conclusion of these reviews is that stressful life events may be a more potent predictor of first episodes, rather than recurrences, of depression (Coyne & Downey, 1991; Garber & Horowitz, 2002; Goodyer, 2001; Kessler, 1997). To explain this relation, the bio-psychological *kindling hypothesis* was proposed (Gold *et al.*, 1988 a, 1988 b; Goodyer, 2001; Kessler, 1997; Meyer *et al.*, 2001; Post *et al.*, 1986). According to this hypothesis, repeated exposure to early stressors, or single exposure to severe stressors, may increase the sensitivity of the bio-psychological stress reactivity system to subsequent stress, such that progressively lower severity thresholds are needed to elicit depressive reactions. This hypothesis further suggests that, with the accumulation of depressive episodes, recurrence may become automated, or generated by minimal levels of objective or subjective stressors.

Finally, as indicated in the previous section, it is generally assumed that the effects of stressful life events on depression are moderated or mediated by psychological factors, such as self-esteem, cognitive style or coping (Brown & Harris, 1978; Garber & Horowitz, 2002). These mediating and moderating relations would also explain the fact that individuals exposed to devastating stressors do not always become depressed (Kessler, 1997) and the lack of specificity of depression as an outcome of life events (Angold & Costello, 2001; Kessler *et al.*, 2001).

Empirical Support

We located 25 studies meeting our inclusion criteria that evaluated the effects of generic stressful life events or chronic stressors on childhood and adolescence depression development. Among those, 2 studies were based on child samples (Lynch & Cicchetti, 1998; Nolen-Hoeksema *et al.*, 1992), 22 on adolescent samples (Allgood-Merten *et al.*, 1990; Daley *et al.*, 1998, 2000; Garber *et al.*, 2002; Garrison,

Jackson, Marsteller, McKeown, & Addy, 1990; Ge, Lorenz, Conger, Elder, & Simons, 1994; Hammen, Henry, & Daley, 2000; Hankin *et al.*, 2001; Hops *et al.*, 1990; Lewinsohn *et al.*, 1994, 1995, 1999, 2001; McFarlane *et al.*, 1995; Robinson *et al.*, 1995; Schwartz & Koenig, 1996 ; Segrin & Flora, 2000; Spence *et al.*, 2002; Sund & Wichstrøm, 2002; Tram & Cole, 2000; Windle, 1992; Zimmerman, Ramirez-Valles, Zapert, & Maton, 2000), and one on a mixed sample of children and adolescents (Abela, 2001). The majority of these studies relied on generic measures of episodic stressful life events or of chronic stressors and concluded that one or both of these variables significantly predicted depression development. Of the seven studies in which the effects of episodic and chronic stressors were evaluated together, five showed that both variables significantly predicted depression (Daley *et al.*, 1998, 2000, Lewinsohn *et al.*, 1994, 1995; Robinson *et al.*, 1995), one found that only episodic stress predicted depression development (Hammen *et al.*, 2000), and one demonstrated that only chronic stress was related to later levels of depression (Hops *et al.*, 1990). Unfortunately, the impact of more specific types of stressors (dependent, independent, disappointments, losses, etc.) could not be compared because of the generic character of the measures used in these studies.

Overall, only two studies failed to find significant relations between exposure to stressful life events and depression development (Lynch & Cicchetti, 1998; Zimmerman *et al.*, 2000). In both cases, the divergent results could possibly be attributed to sampling characteristics of the studies. For instance, Lynch and Cicchetti's (1998) study relied on a sample in which half of the subjects represented known cases of mistreated children. Similarly, Zimmerman *et al.*'s (2000) sample is composed of African American adolescent males of which 69% were high school dropouts. These subjects may all have been exposed to very high levels of stress, which may have created a range restriction problem in the evaluation of the effects of stressful life events on depression.

Ten studies attempted to verify if the stressor-depression relationship was indeed mediated and/or moderated by psychological factors. One of these studies noted that

the effects of stressful life events on depression development were partially mediated through their impact on perceived generic competencies (Tram & Cole, 2000). Seven studies concluded that the effects of stressful life events on depression were stronger for students presenting more negative cognitive styles (Abela, 2001; Garber *et al.*, 2002; Hankin *et al.*, 2001; Lewinsohn *et al.*, 2001; Robinson *et al.*, 1995), lower levels of social skills (Segrin & Flora, 2000), or poorer problem-solving abilities (Spence *et al.*, 2002). Finally, two studies indicated that youth' levels of self-esteem, body image, or coping style did not moderate the stressor-depression relationship (Allgood-Merten *et al.*, 1990; Schwartz & Koenig, 1996).

Studies evaluating gender-based variability in the effects of generic stressors yielded inconsistent results, one study confirming a greater female reactivity (Ge *et al.*, 1994), two studies reporting no gender difference (Lewinsohn *et al.*, 1999; Spence *et al.*, 2002), and one suggesting that life events may be a more potent predictor of depression among boys (Abela, 2001). Additionally, Hankin *et al.*'s (2001) study suggests that a negative cognitive style may moderate the stressor-depression relationships among boys only, whereas both stressors and cognitive style appeared to independently contribute to girls' depression development.

As previously noted, most of the proposed diathesis-stress interactions implicate cognitive risk factors. For this reason and to account for the reduced level of cognitive development observed in children, many scholars were led to suggest that for children to develop depression, they would have to be exposed to far more severe levels of negative life events than adolescents (e.g., Garber & Horowitz, 2002; Harrington & Dubicka, 2001). This hypothesis, however, has received little empirical support. In fact, one of the studies noted no effect of traumatic life events in children (Lynch & Cicchetti, 1998), one demonstrated that negative life events were more strongly related to depression among younger children than among older ones (Nolen-Hoeksema *et al.*, 1992), and one revealed that the effects of life events on depression development were moderated by adolescents', but not children's, cognitive style (Abela, 2001). Finally, the two studies that attempted to validate the

kindling hypothesis in adolescents confirmed that stressful life events were indeed more strongly related to first onsets rather than recurrences of depression (Daley *et al.*, 2000; Lewinsohn *et al.*, 1999).

Summary

In summary, the studies presented in the present section suggest at least three important conclusions. First, there is a clear and important effect of episodic and chronic stressors on depression development. Second, the effects of stressors are likely mediated and/or moderated by psychological factors, particularly cognitive ones. This result provides clear support to the *diathesis-stress* theories of depression. Third, current evidence suggests that the effect of negative life events on depression may be restricted to first episodes. This last result provides partial support to the kindling hypothesis of depression development.

However, many questions remain. For instance, whereas many scholars previously stressed the importance of distinguishing the impact of different types of stressors (dependent/independent, losses, disappointments, dangers, etc.) on depression development, the reviewed studies did not present such comparisons. Moreover, stressful life event scales, even more specific ones, generally consist of a generic mishmash of many social factors. In fact, given the preceding definition, most purported social risk factors for depression, such as sexual abuse, family conflict, victimization, conflict with teachers, etc., can be interpreted as stressful for the exposed individual. Given this lack of precision and contextuality, studies on the effects of generic stressful life events and chronic stressors on depression development are quite limited. Indeed, it is unlikely that each of the factors included in these instruments have similar effects on depression development (e.g., Monroe, & Simons, 1991). This source of confusion may explain, in part, the lack of consistent gender differences in reactivity to stressors reported in the selected studies. Indeed, the cost of caring hypothesis precisely states that girls should react more strongly than boys to the deleterious impact of network stressors but not of other types of stressors.

However, since the selected studies did not make this distinction, this hypothesis could not be verified. Additionally, the possibility that vulnerable individuals may be directly involved in the generation of higher levels of stress exposure for themselves has yet to receive systematic scientific attention (Monroe & Simons, 1991).

Family Factors

Family characteristics are clearly the most studied environmental risk factors for depression development. This interest initially came out as a result of classical psychoanalytical, attachment, and cognitive theory hypotheses which saw early parent-child relationships as distal determinants of presumed individual vulnerabilities to depression (i.e., internal working models, dependency, self-criticism, etc.). More recently, the family systems perspective also emphasized the potential direct role of family characteristics in children's and adolescents' psychosocial development (Becvar & Becvar, 1993; Erel & Burman, 1995). Essentially, the purported family factors implicated in depression development can be broadly subdivided into two dimensions: family structure and family relationships.

Family Structure

Theoretical issues

Family structure refers to family configuration and includes factors such as socio-economical status (SES), parental loss, and separation and parental divorce/remarriage. Generally, the impact of these factors on depression development is purported to be mediated though their effects on relational aspects of the family. For instance, poverty may expose families to an increased number of stressors and may thus limit parental availability to children (Monroe & Hadjiyannakis, 2002). Similarly, parental divorce and remarriage may carry the risk of seriously disrupting youths' lifestyles through two main mechanisms (e.g., Garmezy, 1986). First, such events represent a very stressful transition in youths' lives, often associated with reduced contact with one or both parents or with the need to adapt to a new family

member. Second, parental divorce often occurs in the context of increased family conflicts and tensions, which in themselves have been proposed to increase children's risk of developing depression.

Empirical support

1. Socio-economical status (SES). We found 17 studies meeting our inclusion criteria that evaluated the effect of SES on subsequent depression development, 2 of which are based on child samples (Lynch & Cicchetti, 1998; McLeod & Shanahan, 1996), 8 on adolescent samples (Choi *et al.*, 1997; DuBois *et al.*, 1995; Ge *et al.*, 1996; Joyner & Udry, 2000; Roeser & Eccles, 1998; Siegel, 2002; Sund & Wichstrøm, 2002; Zimmerman *et al.*, 2000), and 7 on long-term follow-up samples (Chase-Lansdale *et al.*, 1995; Fergusson & Linskey, 1997; Fergusson & Woodward, 2000; Jaffe *et al.*, 2002; Miech, Caspi, Moffitt, Entner Wright, & Silva, 1999; Reinherz, Giaconia, Hauf, Wasserman, & Paradis, 2000; Reinherz *et al.*, 1993). In these studies, we found little evidence of an effect of SES on depression development. In fact, only 6 of the 17 studies reported a significant effect of SES on depression development, and in some cases this effect is limited to some indicators of SES or to specific subgroups (Choi *et al.*, 1997; DuBois *et al.*, 1995; Fergusson & Woodward, 2000; Joyner & Udry, 2000; McLeod & Shanahan, 1996; Siegel, 2002). For instance, DuBois *et al.*'s (1995) results limit the effects of SES to moderate, rather than severe, depression, and Siegel (2002) indicates that these effects could be limited to females. Yet, the other studies do not support these conclusions (Choi *et al.*, 1997; Fergusson & Woodward, 2000). However, among the 11 studies reporting non-significant results, 5 relied on multivariate analyses in which other family characteristics (i.e., parental divorce and remarriage, parental support, parent-child conflict) were simultaneously considered as predictors (Chase-Lansdale *et al.*, 1995; Fergusson & Linskey, 1997; Ge *et al.*, 1996; Sund & Wichstrøm, 2002; Zimmerman *et al.*, 2000). Thus, if the effects of SES on depression development are indeed mediated through their impact on other family characteristics, the non-significant results found in these studies could be explained by the simultaneous inclusion of potential mediators in the analyses. However, while

this hypothesis could be more directly tested in mediation analyses, current evidence linking SES to depression development remains inconclusive.

2. Parental loss and separations. We located four studies meeting our inclusion criteria that evaluated the effects of parental loss and separation on depression development. Among these studies, two were based on adolescent samples (Daley *et al.*, 2000; Lewinsohn *et al.*, 1994) and two on long-term follow-up studies (Jaffe *et al.*, 2002; Reinherz *et al.*, 1993). Both studies on adolescents observed no relationship between parental death and depression development (Daley *et al.*, 2000; Lewinsohn *et al.*, 1994). However, both long-term follow-up studies demonstrated a predictive relationship between early separations from parents and depression development in early adulthood (Jaffe *et al.*, 2002; Reinherz *et al.*, 1993). This last result suggests that whereas losing a parent in adolescence does not appear to increase youths' risk of developing depression, being separated early in life from a parent may increase adolescents' and young adults' risk of depression. However, these studies are still few and their results hard to compare given their major methodological differences.

3. Parental divorce and remarriage. Among the 15 studies meeting our inclusion criteria that evaluated the role of parental divorce/remarriage in depression development, 6 were based on adolescent samples (Daley *et al.*, 2000; Ge *et al.*, 1996; Joyner & Udry, 2000; Lewinsohn *et al.*, 1994, 1995; Palosaari *et al.*, 1996), one on a child sample (McLeod & Shanahan, 1996), and 8 on mixed child and adolescent (Jekielek, 1998) or long-term follow-up samples (Chase-Lansdale *et al.*, 1995; Fergusson & Linskey, 1997; Fergusson & Woodward, 2000; Jaffe *et al.*, 2002; Kasen *et al.*, 1996; Reinherz *et al.*, 1993, 2000). Overall, the results appear inconsistent. For instance, seven studies found no effect of parental divorce, parental disruptions or marital status on depression development (Daley *et al.*, 2000; Ge *et al.*, 1996; Lewinsohn *et al.*, 1994, 1995; McLeod & Shanahan, 1996; Reinherz *et al.*, 1993, 2000). However, most of these studies relied on multivariate analyses in which other aspects of the family environment were simultaneously considered as predictors, such as parental support, parent-child conflict, witnessing family violence, poverty, etc.

(Daley *et al.*, 2000; Ge *et al.*, 1996; McLeod & Shanahan, 1996; Reinherz *et al.*, 2000). Eight studies, however, showed that youths exposed to parental divorces or to other changes in parents' marital status presented an increased risk of developing depression (Chase-Lansdale *et al.*, 1995; Fergusson & Linskey, 1997; Fergusson & Woodward, 2000; Jaffe *et al.*, 2002; Jekielek, 1998; Joyner & Udry, 2000; Kasen *et al.*, 1996; Palosaari *et al.*, 1996). Chase-Lansdale *et al.* (1995) even revealed that parental divorce was more clearly associated with the emergence, rather than amplification, of depressive symptoms. However, these studies also highlight the fact that these relationships are likely far more complex than simple main effects. For instance, Fergusson & Woodward (2000) and Jaffe *et al.*'s (2002) results suggest that the active ingredient in this relationship was the number of parental changes experienced by the youth rather than family status or exposure to parental divorce per se. Furthermore, whereas Chase-Lansdale *et al.* (1995) noted that parental divorce occurring in adolescence, but not childhood, was predictive of later depression, three other studies exposed a clear effect of childhood parental divorce on adolescent depression (Fergusson & Woodward, Jaffe *et al.*, 2002; Palosaari *et al.*, 1996).

The effects of parental divorce also seem to depend on other family characteristics. Indeed, Chase-Lansdale *et al.* (1995) observed that the deleterious effects of parental divorce on depression were eliminated when a stepparent was present in the family, suggesting that relational aspects of the family environment are potentially more important than parental divorce per se. Likewise, Palosaari *et al.* (1996) indicated that the effects of parental divorce in childhood were mediated by the resulting reduced closeness with the father. However, Reinherz *et al.* 1993) found that parental remarriage also represented a significant predictor of depression development for the exposed children, although this effect may be limited to girls. Conversely, living with a single custodian mom appears more deleterious for boys than for girls (Kasen *et al.*, 1996). These last results suggest that the presence of a father figure in the home may be particularly important for boys. Palosaari *et al.*'s (1996) results support this interpretation. However, the deleterious effects of parental remarriage for girls may stem from the often reported association between the presence of a stepfather at home

and the risk of incest (e.g., Finkelhor & Baron, 1986), although this hypothesis awaits verification.

Finally, parental divorce often occurs in the context of other family problems that, in themselves, have been related to depression. Interestingly, Jekielek (1998) discovered that parental divorce occurring in the context of preceding marital conflict was unrelated to depression development in youths. Furthermore, this study revealed that parental divorce represented a protective factor against depression development for youths exposed to prolonged marital conflicts. In the context of marital conflicts, divorce may be a better alternative for children.

4. Other structural elements. We found nine studies meeting our inclusion criteria that evaluated the impact of other structural elements of the family environment on depression development. Three of these studies were based on adolescent samples (McFarlane *et al.*, 1995; Sund & Wichstrøm, 2002; Zimmerman *et al.*, 2000), two on child samples (Lynch & Cicchetti, 1998; McLeod & Shanahan, 1996), and four on mixed or long-term follow-up samples (Fergusson & Linskey, 1997; Fergusson & Woodward, 2000; Reinherz *et al.*, 1993, 2000). With only two exceptions, these studies noted no effect of family size, birth order, maternal age and moving on depression development. Among the exceptions, McLeod and Shanahan (1996), using growth curve modeling, concluded that maternal age at birth was unrelated to initial symptoms of depression but significantly predicted later increases in symptoms. For their part, Reinherz *et al.* (1993) observed that, for girls, being the third or later child in the family, having older parents and having more than three siblings were all related to depression development. None of these relationships was significant for boys, suggesting that some aspects of the family environment could be more important for females. None of the other studies evaluated gender-based interactions.

Family Relations

Theoretical issues

Family relations refer directly to the quality of the interactions among family members and purportedly play a more direct role than family structure in depression development (e.g., Berg-Nielsen, Vikan, & Dahl, 2002; Goodman, 2002; Rapee, 1997; Repetti, Taylor, & Seeman, 2002). Usually, three different facets of family relations are distinguished in developmental research: parental support, parent-child conflict, and parental control (Baumrind, 1967; Darling & Steinberg, 1993; Goodman, 2002; Lewis, 1981).

The *parental support* dimension groups the "adaptive" interpersonal elements of the parent-child relation and includes concepts such as parental warmth, sensitivity, support and availability. Parental support has been proposed to bear positive effects for children (Cummings & Davies, 1994; Downey & Coyne, 1990; Goodman, 2002; Repetti *et al.*, 2002). In this relationship, the crucial element seems to be the child's sense of being accepted, loved and worthy of attention, and the integration of this element at the core of his self-definition. Without this feeling, the child will present an increased risk of developing depression. Previous reviews supported this proposition (Berg-Nielsen *et al.*, 2002; McCauley *et al.*, 2001; Rapee, 1997).

The *parent-child conflict* dimension refers to the more "problematic" elements of the parent-child relation and includes parental hostility, rejection, and unresponsiveness, as well as parent-child disagreements and family stressors. Parent-child conflict has been proposed to carry risk for exposed youths (Goodman, 2002; Repetti *et al.*, 2002). Indeed, being exposed to parental hostility, rejection or anger may convey to the child the message that he/she and his/her feelings are unworthy of consideration. Moreover, parent-child conflict may also affect children's representations of self-other relationships, of socially acceptable ways of expressing emotions, and of efficient coping mechanisms through modeling, reinforcement of maladaptive responses or through other learning processes (Goodman, 2002; Repetti *et al.*, 2002).

Moreover, parent-child conflict has been conceptualized as possibly the most serious chronic stressor to which children could be exposed (Goodman, 2002; Repetti *et al.*, 2002). Again, previous reviews supported these proposals (Berg-Nielsen *et al.*, 2002; McCauley *et al.*, 2001; Rapee, 1997).

The *parental control* dimension refers to parental efforts to exert some degree of control on the child's behaviors and includes proactive monitoring and democratic practices as well as intrusiveness, black-mailing, physical punishment, and inconsistency. Effective parental control has been proposed to play a very significant role in children's learning of efficient emotional regulation mechanisms and internalization of self-control standards (Goodman, 2002; Repetti *et al.*, 2002). Nevertheless, rigid/harsh parental control, through over-reliance on punishment, is purported to interfere with youths' autonomy, and to diminish their feelings of self-determination; it may be related to the early development of helplessness (Goodman, 2002). Conversely, some children may also internalize their parents' high standards and thus develop vulnerable self-schemas (Blatt & Zuroff, 1992; McCauley *et al.*, 2001). Lax or disengaged parental control may also, through different processes such as lack of reinforcement, impair children's learning of adaptive ways of emotional regulation and self-control (Goodman, 2002). Finally, some parents, especially those who themselves suffer from emotional problems, may display a combination of harsh and lax parenting. This inconsistent style of control is purported to carry the most deleterious impact for children, who then learn that there is no way for them to predict or influence their environment (Cummings & Davies, 1994; Downey & Coyne, 1990; Goodman, 2002). Taken together, these considerations suggest that parental attempts to "control" children's behaviors, to be efficient, should occur in a supportive context and designed to facilitate children's exploration of the environment.

A full understanding of family relationships should also take into account the quality of *marital relations*. Indeed, problematic marital relations have been conceptualized as a serious source of chronic stress for exposed children and as an important

determinant of children's emotional insecurity (Cummings & Davies, 1994; Cummings *et al.*, 2001; Davies & Cummings, 1994; Downey & Coyne, 1990). Indeed, children exposed to parental conflicts, especially unresolved ones, may become sensitized to conflicts, interpret them as threatening (especially if violence is involved), learn inefficient ways of dealing with them, and build up problematic representations of interpersonal relations (Cummings & Davies, 1994; Cummings *et al.*, 2001; Davies & Cummings, 1994). All of these potential consequences of marital conflicts may then seriously disrupt the child's handling of developmental tasks and thus threaten his/her lifelong development. Moreover, marital dissatisfaction and conflicts are usually energy- and time-consuming for parents, who may come to rely on more inefficient control strategies and become less available or more hostile toward children (Davies & Cummings, 1994). In extreme cases, youths may even be drawn into the conflict either through parents' attempts to manipulate each other using the child or through direct attempts to intervene (Cummings & Davies, 1994; Davies & Cummings, 1994).

Likewise, the quality of sibling relationships should also be considered if one wishes to clearly understand the effects of family relations on children development (Phares, Duhig, & Watkins, 2002). Unfortunately, we are aware of very few studies that considered the role of sibling relationships or of relationships with other family members on depression development. None of these studies met our inclusion criteria.

Finally, *child abuse and neglect*, which represent more extreme characteristics of dysfunctional families, also represent potential risk factors for children's and adolescents' depression development (Cicchetti & Toth, 1995; Kendall-Tackett, Williams, & Finkelhor, 1993). Neglect refers to "a failure to provide for children's basic physical, educational or emotional needs" (Wolfe, 1999, p. 8). Physical abuse refers to "the infliction or endangerment of physical injury as the result of punching, beating, kicking, biting, burning, shaking, or otherwise harming a child (Wolfe, 1999, p. 8)." Sexual abuse refers to "two overlapping but distinguishable types of

interactions: (a) forced or coerced sexual behavior imposed on a child, and (b) sexual activity between a child and a much older person, whether or not obvious coercion is involved (a common definition of "much older" is 5 or more years) (Browne & Finkelhor, 1986, p. 66)." Whereas child neglect and physical abuse mostly occurs within the family system, sexual abuse may occur within (incest) as well as outside this system (Wolfe, 1999). Since previous reviews did not report qualitatively different relations between these two forms of abuse and depression development, other than the fact that incest's relations to depression are generally stronger, we omitted this distinction in the present review (Kendall-Tackett *et al.*, 1993; Malinosky-Rummel & Hansen, 1993).

In previous reviews, preliminary attempts were made to account for the deleterious impact of child abuse and neglect on youths' psychosocial development. For instance, Kessler (2000) hypothesized that childhood exposure to parental violence may increase children's emotional reactivity through their disturbing effects on attachment relationships and through their contribution to the development of a negative cognitive style (resulting from the uncontrollability of the situation). Conversely, subsequent exposure to stressors may reactivate childhood memories of violence which will then result in impaired coping ability. In a related stress-based conceptualization of sexual abuse, Spaccarelli (1994) indicated that sexual abuse per se was associated with at least four kinds of stressors: (a) abuse stressors, such as sexual contact, coercion, demeaning attitudes, secrecy, and trust violation; (b) abuse-related stressors, such as family dysfunction, marital separation, isolation, and lack of support; (c) disclosure-related stressors, such as insensitive or repeated interviews, testimony, procedural delays, and disbelief. Spaccarelli (1994) further adds that the effects of these stressors would likely be moderated by children's cognitive appraisal of the abusive situation and usual coping mechanisms, as well as by the level of social support received from other family members.

Previous reviews generally concluded that youths' exposure to neglect, physical abuse, and sexual abuse represented significant risk factors for depression

development although these associations lacked specificity, abuse and neglect being also predictive of a plethora of other problems (Browne & Finkelhor, 1986; Coyne & Downey, 1991; Kendall-Tackettt *et al.*, 1993; Kessler, 2000; Weiss, Longhurst, & Mazure, 1999; Wolfe, 1999). Moreover, these reviews indicated that the deleterious impacts of childhood abuse and neglect were more severe for girls (Kendall-Tackett *et al.*, 1993), that sexual abuse carried more risk than physical abuse or neglect (Toth & Cicchetti, 1996), and that the consequences were influenced by the severity of the experience, genital contact, intercourse, and incest having more deleterious effects than other forms of sexual abuse (Browne & Finkelhor, 1986; Kendall-Tackett *et al.*, 1993).

Empirical support

1. Parental support. Sixteen studies that evaluated the effects of parental support on depression development met our inclusion criteria. Most (14) of these studies were based on adolescent samples (Choi *et al.*, 1997; Davies & Windle, 2001; Garrison *et al.*, 1990; Ge *et al.*, 1994, 1996; Hops *et al.*, 1990; Lewinsohn *et al.*, 1994, 1995; McFarlane *et al.*, 1995; Palosaari *et al.*, 1996; Slavin & Rainer, 1990; Windle 1992; Windle & Windle, 2001; Zimmerman *et al.*, 2000), whereas only one was based on a child sample (Sim, 2002) and one on a long-term follow-up sample (Duggal *et al.*, 2001). Although 4 of these studies revealed non-significant effects of parental support on adolescent depression development (Garrison *et al.*, 1990; Hops *et al.*, 1990; Slavin & Rainer, 1990; Windle & Windle, 2001), 8 demonstrated a clear main effect, higher levels of parental support being related to lower levels of depression (Davies & Windle, 2001; Duggal *et al.*, 2001; Ge *et al.*, 1996; Lewinsohn *et al.*, 1994, 1995; McFarlane *et al.*, 1995; Palosaari *et al.*, 1996; Sim, 2002; Zimmerman *et al.*, 2000). Moreover, 3 studies showed that the positive effects of parental support were limited to, or stronger for, girls than for boys (Choi *et al.*, 1997; Ge *et al.*, 1994; Windle, 1992), and one long-term follow-up study suggested that the effects of parental support may be more important in childhood than in adolescence, even when the evaluated outcome is adolescent depression (Duggal *et al.*, 2001). Palosaari *et al.*

(1996), however, suggest that while a youth's closeness to his/her father was related to lower levels of depression in boys and girls, the positive effect of mother-youth closeness was limited to boys.

Finally, 4 studies attempted to evaluate whether parental support may represent a protective factor against depression development for youths exposed to stressful life events. Three of these studies failed to support this hypothesis (Garrison *et al.*, 1990; Windle, 1992; Zimmerman *et al.*, 2000). However, Ge *et al.* (1994) found that family support did exert a protective effect against the deleterious impact of stressors, but that this effect was limited to older girls. More studies will be needed to verify if these results can be replicated.

2. *Parent-child conflict.* We located seven studies meeting our inclusion criteria that focused specifically on the effects of parent-child conflict on depression development. Three of these studies were based on adolescent samples (Ge *et al.*, 1996; Lewinsohn *et al.*, 1994, 1995), one on a child sample (Hilsman & Garber, 1995) and three on long-term follow-up samples (Fergusson & Linskey, 1997; Jaffe *et al.*, 2002; Kaitanen, Räikkönen, Keskivaara, & Keltikangas-Järvinen, 1999). Among these studies, the majority showed that parent-child conflict (Lewinsohn *et al.*, 1994, 1995), maternal hostility (Ge *et al.*, 1996; Katainen, Räikkönen, Keskivaara *et al.*, 1999), negative family life events (Fergusson & Linskey, 1997), and parent-child disagreements about discipline (Jaffe *et al.*, 2002) were predictive of increased levels of depression among participants. However, Jaffe *et al.* (2002) obtained non-significant effects of father hostility and maternal rejection on depression development. Furthermore, three of the seven studies, based on two samples, evaluated the impact of a specific kind of parent-child conflict: parent dissatisfaction with children grades. Two of these studies concluded that this factor had no impact on adolescent depression development (Lewinsohn *et al.*, 1994, 1995). Conversely, Hilsman and Garber (1995) noted that parent dissatisfaction with children's grades was associated with a short-term increase in children's depression levels, in the week

following report cards. Unfortunately, we are aware of no studies evaluating gender or developmental differences in youths' reactivity to parent-child conflict.

3. Parental control. We found only three studies which met our inclusion criteria and focused on the effects of parental control on depression development, one of which was based on an adolescent sample (Ge *et al.*, 1996), one on a child sample (Pomerantz, 2001), and one on a long-term follow-up sample (Fergusson & Woodward, 2000). Fergusson and Woodward's (2000) results revealed that parental punitiveness in childhood was not predictive of later depression. Similarly, Ge *et al.* (1996) oberved no effect of parental discipline on adolescent depression development. However, Pomerantz's (2001) reported that, although intrusive parenting was not directly related to children's depression development, it did increase the risk among children who already exhibited negative attributional styles and problematic self-concepts. This result suggests that ineffective parental control may represent an additional risk for children already at risk.

4. Marital problems. Only five studies that evaluated the effects of marital problems on depression development met our inclusion criteria. Of these, three were based on adolescent samples (Daley *et al.*, 2000; Davies & Windle, 2001; Unger, Brown, Tressell, & McLeod, 2000) and two on long-term follow-up samples (Fergusson & Woodward, 2000; Reinherz *et al.*, 1993). Only one of these studies failed to find significant relations between marital conflict and later depression development (Fergusson & Woodward, 2000). Three studies exposed a clear effect of marital conflict on adolescent depression development (Daley *et al.*, 2000), although this effect was reported to be stronger for girls (Reinherz *et al.*, 1993) as well as for children with more difficult temperaments (Davies & Windle, 2001). Additionally, Davies and Windle (2001) noted that, while marital discord was related to higher levels of depression for children with low levels of externalizing behavior problems, the concurrent presence of these two factors was predictive of persistently high levels of depression. Finally, two studies showed that the effects of marital conflict and

discord were completely mediated by parent-child conflict (Davies & Windle, 2001) and general family functioning (Unger *et al.*, 2000).

5. Child abuse and neglect. Studies on child abuse and neglect represent a specific case in depression research in that they were often designed to study the consequence of child abuse and neglect rather than predictors of depression development. In this context, few of the many available studies met our inclusion criteria because they did not attempt to control previous levels of depression. Among studies meeting our inclusion criteria, seven studies evaluated the relations between youths' exposure to abuse, neglect and violence, and depression development. One of these studies was based on a sample of children (Lynch & Cicchetti, 1998), one on a mixed sample (Boney-McCoy & Finkelhor, 1996) and five on long-term follow-up samples (Brown, Cohen, Johnson, & Smailes, 1999; Duggal *et al.*, 2001; Fergusson, Horwood, & Linskey, 1996; Fergusson & Linskey, 1997; Fergusson & Woodward, 2000). Most of these studies revealed a clear relation between sexual abuse, physical abuse or neglect and depression development. Only two studies failed found non-significant effects of childhood physical abuse on depression development (Fergusson & Woodward, 2000; Lynch & Cicchetti, 1998). Similarly, three other studies reported that physical abuse effects were generally lower than those of sexual abuse or tended to disappear when sexually abusive experiences were simultaneously taken into account in multivariate analyses (Boney-McCoy & Finkelhor, 1996; Brown *et al.*, 1999; Fergusson & Linskey, 1997). The one study in which the severity of sexual abuse experiences was taken into account exposed that sexual abuse involving intercourse was more importantly related to depression development than other forms of sexual abuse (Fergusson *et al.*, 1996). Finally, in an attempt to distinguish the effects of many different kinds of childhood violence experiences, Boney-McCoy and Finkelhor (1996) revealed that whereas sexual assaults, simple assaults, kidnapping, and parental violence were predictive of later depression development, exposure to non-parental family violence and to aggravated assaults by non-family members were not.

6. Other relational elements. We located four studies meeting our inclusion criteria that evaluated the impact of other aspects of family relationships on depression development. All of these studies were based on long-term follow-up samples (Duggal *et al.*, 2001; Katainen, Räikkönen, Keskivara *et al.*, 1999; Reinherz *et al.*, 1993, 2000). Unfortunately, none of them evaluated the role of sibling relationships on depression development, although Reinherz *et al.* (2000) observed that alcohol and drug-related problems in siblings were predictive of adolescent depression development. Among the other studies, Duggal *et al.* (2001) concluded that youths whose parents received low levels of social support and children of highly stressed parents presented an elevated risk of developing depression. Similarly, Katainen, Räikkönen, Keskivara *et al.* (1999) indicated that maternal role satisfaction significantly predicted depression development for girls. Finally, Reinherz *et al.* (1993) found that 9-year-old children with poor perceptions of their overall role in the family significantly had a higher risk of developing depression in adolescence.

Summary

The results presented in this section show that many dimensions of the family environment represent risk factors for child and adolescent depression development. Indeed, children exposed to abusive experiences and to marital conflicts within their families, and children having conflictual relationships with their parents appear to present an increased risk of developing depression. Alternatively, parental support appears to be related to a lower risk of developing depression in children and adolescents. Some preliminary evidence even suggests that having a positive relationship with one's parents may represent a protective factor for children exposed to a variety of stressors, although this result should be replicated before unambiguous conclusions can be reached.

Conversely, little evidence indicated that structural family variables could exert an impact on depression development. Current hypotheses suggesting that the effects of family structure on depression development are likely mediated by the resulting

family instability and turmoil may explain this result. Unfortunately, many studies in which the effects of family structure on depression development were evaluated simultaneously considered other family characteristics in their analyses and did not specifically test mediational hypotheses. Nevertheless, preliminary results suggest that the deleterious effects of parental divorce could be reduced when the child's closeness to his/her father is maintained or when a surrogate father enters the family and indicate that parental divorce may even be beneficial to children if it occurs in the context of prolonged marital conflicts. Whereas these last results should be replicated, they reinforce the idea that the effects of family structure and relationships on depression development are not independent from each other.

In the previous section, we presented Kessler and McLeod's (1984) cost of caring hypothesis of depression and concluded that current evidence regarding the effects of stressful life events on depression development was insufficient to validate this hypothesis. We also noted that such studies were limited by their reliance on instruments in which a plethora of social factors, seen as stressful, were indiscriminately considered. In this section, current evidence suggests that the effects of family characteristics on depression development may be stronger for girls than for boys. As many family characteristics can be considered as stressful for the exposed child (parental loss and separations, parental divorce, parent-child conflict, abuse and neglect, marital conflict), this result strongly supports the cost of caring hypothesis.

Given the attention devoted to family factors in depression development, it is surprising that so few studies attempted to verify the impact of parental control dimensions on children's and adolescents' development or to precisely evaluate the moderators and mediators underlying the effects of family characteristics. These questions certainly deserve to be more accurately evaluated in the context of methodologically strong prospective studies. In a related way, such studies should also devote more attention to age-based variations in the effects. For instance, developmental psychology indicates that whereas children's developmental tasks mostly imply family integration, adolescents focus more on gaining autonomy from

parents and on learning intimacy and reciprocity in peer group context (Cicchetti & Rogosh, 2002; Cicchetti & Schneider-Rosen, 1986; Garnefski, 2000). The effects of families on depression development may therefore become secondary to peer-group effects in adolescence.

Peer Relationships

Theoretical Issues

Given the important role of peer relationships in child, and particularly adolescent, development, it is not surprising that peer-related factors have been proposed to play a role in the emergence of depressive symptoms. In fact, among the important developmental tasks faced by children and adolescents are the formation of intimate relationships with same- and opposite-sex peers, the development of romantic involvement, and the emergence of prosocial behaviors and empathy (Cicchetti & Rogosh, 2002; Cicchetti & Schneider-Rosen, 1986; Petersen & Lefert, 1995). Developmental differences in peer group characteristics allow us to postulate that the impact of peer-related factors should also differ according to age and gender (Cicchetti & Rogosh, 2002; Cicchetti & Schneider-Rosen, 1986; Petersen & Lefert, 1995). Indeed, whereas child peer groups are often composed of same-sex individuals and serve instrumental play-oriented functions, adolescent peer groups usually combine individuals from both sexes and fill intimacy and reciprocity functions. Moreover, girls often seek intimacy, connectedness, and reciprocity in peer relationships more than boys, who tend to rely on peers for more instrumental, companionship, and fun-oriented functions (Cyranowski *et al.*, 2000; Cross, & Madson, 1997; Helgeson, 1994; Rudolph, 2002; Taylor *et al.*, 2000). Overall, it remains clear that, as humans form a primarily social and gregarious species, children's and adolescents' development will be closely related to the quality of their friendship networks. Furthermore, as development follows a sequence of tasks whose successful resolutions serve as building blocks for the next ones, the quality of peer relationships will also be intimately affected by children's resolution of previous

tasks, such as gaining relative autonomy from parents, developing a coherent self-concept, acquiring adequate emotion regulation capacities, etc. (e.g., Cicchetti & Rogosh, 2002; Cicchetti & Schneider-Rosen, 1986; Petersen & Lefert, 1995).

The many peer-related variables studied in relation to depression development can be grouped into at least three generic dimensions: *supportive peer behaviors*, *hostile peer behaviors and victimization*, and *social integration/isolation* (e.g., Coyne & Downey, 1991; Dill, & Anderson, 1999; Finch, Okun, Pool, & Ruehlman, 1999). Supportive peer behaviors and social integration have often been hypothesized to play a protective role against depression development for children and adolescents exposed to different kinds of stressors and life transitions (Coyne & Downey, 1991). Conversely, the potential impact of hostile peer behaviors or social isolation on children's and adolescents' depression development has generally been hypothesized to come from their stressfulness (Coyne & Downey, 1991). Conclusions from previous reviews generally supported these hypotheses (Baron, 1993; Chakraborty & McKenzie, 2002; Dill & Anderson, 1999; Finch *et al.*, 1999; Garber & Horowitz, 2002; Hawker & Boulton, 2000). In a detailed meta-analysis, Finch *et al.* (1999) even found that the observed positive effects of social support may come from individuals' perceptions of the quality of their social support networks rather than from the actual actions of the members of this network.

Finally, other authors have pointed out the importance of distinguishing the effects of *romantic involvement* and breakups on adolescent depression development from the effects of the preceding dimensions (Cicchetti & Rogosh, 2002). Indeed, it is often in the context of their romantic relationships that adolescents learn the reciprocity- and intimacy-related skills needed to form mature romantic ties. Because they do not yet master these skills, adolescents' romantic relationships can often be tumultuous and stress generating.

Empirical Support

Supportive peer behaviors

Twelve studies that focused on the effects of peer support on depression development met our inclusion criteria. One of these studies was based on a child sample (Sim, 2002), ten on adolescent samples (Cheng, 1998; Lewinsohn *et al.*, 1994, 1995; McFarlane *et al.*, 1995; Slavin & Rainer, 1990; Stice & Bearman, 2001; Sund & Wichstrøm, 2002; Windle, 1992, 1994; Zimmerman *et al.*, 2000), and one on a long-term follow-up sample (Katainen, Räikkönen, & Keltikangas-Järvinen, 1999). Six of these studies revealed no effect of peers' and non-family adults' social support on depression (Lewinsohn *et al.*, 1994, 1995; Sim, 2002; Sund & Wichstrøm, 2002; Windle, 1994; Zimmerman *et al.*, 2000). However, three studies showed that social support negatively predicted girls', but not boy's, risk of developing depression (Katainen, Räikkönen, & Keltikangas-Jarvinen, 1999; Slavin & Rainer, 1990; Stice & Bearman, 2001).

In a study investigating the protective role of friends' support on depression development in stress-exposed adolescents, Windle (1992) demonstrated that peers' social support diminished the risk of developing depression, but only among youths exposed to low and moderate levels of stress. Yet, friends' support amplified the risk of depression for individuals exposed to high levels of stress. Although the reasons for this effect remain unclear, McFarlane *et al.*'s (1995) results may provide a preliminary explanation. Indeed, these authors found that receiving higher levels of social support from peers tended to be associated with small increases in stress exposure, possibly due to the increased risk of being exposed to network stressors. However, McFarlane *et al.*'s (1995) results also suggest that social support costs in terms of stress exposure generally tend to be balanced by its benefits on increased levels of social self-efficacy. In this context, Windle's (1992) result may be due to the fact that costs of social support may outweigh its benefits for individuals already experiencing high levels of stress.

Finally, Cheng's (1998) extensive study attempted to differentiate the effects of different dimensions of social support in adolescent boys and girls. Their results showed that adolescents' perceptions of social support were negatively related to depression development whereas the size of their social support network was not. Among boys, those who received more instrumental support (tangible behavioral or material help) presented a lower risk of developing depression. This dimension did not influence girls' risk of developing depression. Conversely, girls' risk of depression was negatively related to the levels of socioemotional support they received from their peers, a variable which had no effect for boys. This result, which should also be replicated, appears coherent with the purported different functions of boys' and girls' peer relationships.

Hostile peer behaviors and victimization

Among studies meeting our inclusion criteria, only four evaluated the relationships between peer hostility and depression development. Two of these studies used child samples (Boivin, Hymel, & Bukowski, 1995; Khatri *et al.*, 2000), one used an adolescent sample (Windle, 1994), and one used a long-term follow-up sample (Jaffe *et al.*, 2002). With the exception of Jaffe *et al.*'s (2002) study, which noted an effect of generic peer-related problems on later depression development, none of the other studies demonstrated a significant prospective effect of victimization or peer hostility on depression development. While these results contrast highly with the conclusions from previous reviews, it remains possible to explain this discrepancy. Indeed, preliminary evidence suggests that depression may, in itself, represent a significant predictor of victimization (Hodges & Perry, 1999). The significant results from the studies included in previous reviews could thus come from the fact that most of them were cross-sectional and did not control for previous levels of depression. Future studies on these topics should thus focus more clearly on the potential bidirectionality of the effects.

Social integration/isolation

Nine studies met our inclusion criteria and evaluated the relations between social integration/isolation and depression development. Two of these studies were based on child samples (Boivin *et al.*, 1995; Panak & Garber, 1992), four on adolescent samples (Choi *et al.*, 1997; Kiesner, 2002; Stein *et al.*, 1996; Sund & Wichstrøm, 2002), and three on long-term follow-up samples (Fergusson & Woodward, 2000; Reinherz *et al.*, 1993, 2000). Overall, the results obtained appeared highly inconsistent. Three of these studies indicated a clear negative relationship between depression development and the quality of social integration measured directly or through social preference scores (Boivin *et al.*, 1995; Kiesner, 2002; Stein *et al.*, 1996). Conversely, measures of attachment to peers were not showed to predict depression development (Fergusson & Woodward, 2000; Sund & Wichstrøm, 2002). Similarly, Reinherz and colleagues (Reinherz *et al.*, 1993, 2000) concluded that depression levels were significantly predicted from self-evaluated unpopularity, while Boivin *et al.*'s (1995) study found no effect of self-rated loneliness or peer-rated withdrawal. While these discrepancies could easily be explained by differences in measurement methods, two additional studies suggest that the effects of social integration/isolation on depression development may involve more complex mediating or moderating relationships than those considered in the previous studies. First, Choi *et al.*'s (1997) results indicate that the deleterious effects of social isolation on depression were limited to girls. Secondly, Panak and Garber (1992) showed that the negative impact of peer-rated rejection was mediated by individuals' perceptions of being rejected. The lack of consideration of potential mediators or moderators in the previous studies may thus be responsible for their divergent results.

Romantic involvement

Given the aforementioned importance of romantic involvement in adolescent development, it was surprising to find so few studies evaluating the role of such relationships in depression development. Indeed, only two studies could be located in which our inclusion criteria were respected and which focused on the relations

between romantic involvement and depression development. In the first of these studies, Monroe, Rohde, Seeley, and Lewinsohn (1999) reported that romantic breakups represented significant predictors of later depression development, and that this effect was limited to first episodes and did not vary on the basis of gender. In the other study, Joyner and Udry (2000) did not evaluate the effects of romantic breakups, but preferred to focus directly on romantic involvement and on adolescents' desires for romantic involvement. Their results showed that both variables were predictive of depression development, suggesting that being involved in a romantic relationship in adolescence, or even wanting to be involved, may represent risk factors for depression. They also found that these effects were stronger for girls, especially the youngest ones. But in an additional analysis partly based on cross-sectional data, Joyner and Udry (2000) concluded that these effects were not due to romantic involvement per se. Rather, they appeared to be mediated by the impact of romantic involvement on the emergence of problems in family and school functioning and on the risk of going through romantic breakups.

Other characteristics of peer relationships

Among the studies meeting our inclusion criteria, seven attempted to evaluate the relations between depression development and other characteristics of peer relationships. Of these studies, six were based on adolescent samples (Brent, Moritz, Bridge, Perper, & Canobbio, 1996; Brent *et al.*, 1994; Hogue & Steinberg, 1995; Rosenfield *et al.*, 2000; Windle & Windle, 2001) and one was based on a long-term follow-up sample (Fergusson & Woodward, 2000). First, some of these studies attempted to evaluate the effects of deviant peer associations. Among these studies, three noted no relation between depression development and generic peer deviance/behavioral disorders (Fergusson & Woodward, 2000), peer cigarette use (Stein *et al.*, 1996) and peer substance abuse (Windle & Windle, 2001). However, one of these studies revealed that affiliating with alcohol-using peers was a significant predictor of adolescent depression development (Windle, & Windle, 2001). Knowing that alcohol abuse is a significant predictor, correlate, and consequence of depression (see the previous section), Hogue and Steinberg's (1995)

results may shed a new light on this association. In a detailed sociometric analysis of adolescent friendship networks, Hogue and Steinberg (1995) showed that, not only did depressed adolescents tend to select depressed peers (selection), but adolescents' depression levels also tend to be affected by (socialization) and predictive of (contagion) peers' depression levels. Therefore, if depressed adolescents tend to use alcohol and to give rise to depressive feeling in their peers, the above-mentioned link between affiliation with alcohol-using peers and depression development may be an artifact of the comorbidity between depression and alcohol use.

In another study, Rosenfield *et al.* (2000) reported a predictive association between adolescents' levels of empathy with friends' discomfort and depression development. This relation may represent one of the mechanisms underlying the contagion and socialization effects found in Hogue and Steinberg's (1995) study. Finally, Brent and colleagues (1994, 1996) found that exposure to a peer's suicide represented a significant risk factor for adolescent depression development, even 19 months later (Brent *et al.*, 1996). Furthermore, this relationship was even stronger when adolescents knew of the victims' suicidal intentions (Brent *et al.*, 1994).

Summary

The studies reviewed in this section suggest that low levels of social support may represent a risk factor for girls' depression development and that high levels of social support may even protect them in the presence of stressful life events. The most likely causal process behind the effects of social support is a cognitive one (perception). Indeed, Finch *et al.*'s (1999) meta-analysis and Cheng's (1998) study both suggested that the effects of adolescents' perceptions and satisfaction regarding the availability and quality of social support far outweighs the effects of "real-life," enacted social support. Yet, the protective role of peers' social support against the deleterious effects of stressful life events may fade out in the presence of extreme levels of stress. McFarlane *et al.*'s (1995) results are coherent with this proposition and suggest two mechanisms which may underlie the effects of peer support: small increases in

exposure to network stress (the cost) and large increases in social self-efficacy (the benefits). Consequently, for adolescents already exposed to extreme levels of stressful life events, the costs of social support may be sufficient to offset its benefits. Unfortunately, these studies have not, to our knowledge, been replicated. Results regarding the role of social integration in depression development bear great similarity to those on social support. However, the relationships between social integration and depression development may be stronger for girls. Alternately, the effect of these variables may be mediated by youths' perceptions of social integration. Once again, more studies will be needed to validate this proposal.

The specific role of negative peer behaviors and victimization in depression development remains uncertain due to a lack of prospective studies attempting to disentangle the impact of depression on peer hostility from the impact of peer hostility on depression. Similarly, even if current evidence suggests that romantic breakups may predict an increased risk of depression development, we located only two studies evaluating the impact of romantic involvement on depression development. Finally, preliminary evidence also suggests that peer psychosocial problems, particularly alcohol use and depression, may represent significant risk factors for depression development. The mechanisms behind these effects could possibly implicate adolescents' levels of empathy with friends' negative emotions. Rose's (2002) recent theoretical model also suggests a complementary mechanism through which affiliation with deviant peers may lead to depression: co-rumination. According to Rose (2002), co-rumination represents a shared form of rumination (see the previous section on coping styles) occurring within dyadic relationships and may represent a risk factor for depression development. Co-rumination within peer groups may also embody one of the mechanisms through which social support may take a toll on an adolescent's well-being in the context of stressful life events.

Overall, two main conclusions may be reached from this section. First, few longitudinal studies attempted to investigate the effects of peer relationships on adolescent depression development. Second, very few of these studies were based on

child samples. Even if friendships are purported to play a more important role in adolescent development, this lack of studies is a serious obstacle to our understanding of depression development.

School Life

Theoretical Issues

From kindergarten to early adulthood, youngsters will spend a significant part of their waking life at school. This fact alone clearly underscores the importance of school life in child and adolescent development. In addition, schools are key socialization areas for children and adolescents, as well as the medium by which they will learn, directly or indirectly, most of the skills they need to function properly in society. School life also encompasses many non-academic aspects of children's and adolescents' social existence, such as the beginning of friendships, romance, and autonomy from parents. School life has therefore been proposed to play an important role in children's and adolescents' psychosocial development (Bronfenbrenner, 1977; Mortimore, 1995; Roeser, Eccles, & Strobel, 1998; Rutter *et al.*, 1997), and the study of its effects on human development has often been cited as one of the key priorities facing developmental research (Boyce *et al.*, 1998; Rutter, 1999, 2000; Rutter *et al.*, 1997; Zaslow, & Takanishi, 1993).

School life dimensions may comprise stress-generating experiences (i.e., conflict with teachers, feelings of insecurity, failures) and thus increase youths' risk of developing problem behaviors, as well as very positive experiences (i.e., teacher warmth and support, school success, peaceful learning environment) and thus contribute to help at-risk youths to adopt more adapted developmental trajectories. Actual theories attempting to explain the relationships between school life and psychosocial development invoke the fact that youth's experiences at school may, or may not, help them to fulfill their basic developmental needs (Eccles, Lord, & Midgley, 1991; Eccles *et al.*, 1993; Moos, 1979). For example, Eccles and colleagues (Eccles *et al.*,

1991, 1993) argued that whereas adolescents' basic developmental needs imply autonomy, intimacy, identity formation, sexuality, and abstract thinking, middle schools are often characterized by increased discipline and control, academic and social competitiveness, social network disruptions, and lower cognitive demands. The resulting mismatch may then create an increased risk of psychosocial problems in youths, especially in those who are already vulnerable to such problems (Eccles *et al.*, 1991, 1993; Janosz, Georges, & Parent, 1998). More precisely, adolescents whose school life is characterized by a mismatch between developmental needs and socialization experiences may come to internalize the idea that their needs are unworthy of attention and develop chronic feelings of helplessness, which in turn may lead to depression (Haaga *et al.*, 1991).

According to Bronfenbrenner's (1977) ecosystemic model, most of the previously reviewed antecedents of depression development can be grouped under the labels of "ontosystems," or youths' personal characteristics, and "microsystems," or youths' immediate interactions with socialization agents (peers, parents, etc.). Conversely, schools are best conceptualized as mesosystems. According to Bronfenbrenner (1977, p. 515), mesosytems refer to the "interrelations among major settings containing the developing person [or microsystems] at a particular point in his or her life." Indeed, schools represent complex social systems in which different microsystems interact (teacher-student relationships, peer groups, parental educative practices) and which also possess their own specific characteristics. Accordingly, students' school life quality will be determined by a combination of various factors related to their psychological characteristics and socialization experiences and to the specific characteristics of their schools (Janosz *et al.*, 1998).

Because individuals may choose and modify environments, it is unlikely that students with different psychological characteristics will be exposed to similar experiences at school (Monroe & Simons, 1991; Mortimore, 1995; Rutter, 1999). In addition to students' psychological background characteristics (neuroticism, self-esteem, behavioral disorders), which may indirectly influence the quality of their school life,

more specific psychological characteristics (school adaptation, academic motivation, academic achievement, etc.) represent more direct determinants of students' school life quality. Therefore, a complete understanding of the effects of school life on depression development should take these specific in-school psychological characteristics into account. The relationship between these characteristics and depression development was already covered in previous sections of this review and will not be further highlighted here.

Similarly, we previously noted that children's and adolescents' socialization experiences within families and peer groups influenced their risk of developing depression. Additionally, these experiences may also indirectly impact the quality of their school life. For instance, parents may choose to send their children to schools which conform most to their own values and practices. Moreover, youths also tend to reproduce at school (and other settings) the various skills and interactional patterns that they previously learned in contact with peers and parents (Cicchetti & Rogosch, 2002; Cicchetti & Schneider-Rosen, 1986; Cicchetti & Toth, 1998). Furthermore, three kinds of socialization experiences may also be more directly involved in the quality of students' school life: parental school-related educative practices, school-based interactions with peers, and interactions with school adults (teachers and other members of the school personnel). Current knowledge about the impact of school-related socialization experiences involving peers and family members on depression development was already presented in previous sections and will not be repeated in the present section.

Finally, school life also implies more than the sum of the previously described microsystems. In themselves, schools are social systems with their own rules and characteristics which are relatively independent from the socialization experiences and individual characteristics of the specific students attending them (Janosz *et al.*, 1998). For example, whereas a specific student may have never been personally victimized at school, victimization may still be a frequent problem in his or her school. Consequently, studying the effects of school life on depression development

implies that specific school characteristics should also be considered. Three methods have generally been used to evaluate school environment characteristics. First, some scholars relied on students' perceptions of the characteristics of their school environment. This approach is generally referred to as the evaluation of school *psychological environment* (Kuperminc, Leadbeater, & Blatt, 2001; Roeser & Eccles, 1998; Roeser, Eccles, & Sameroff, 1998; Roeser, Eccles, & Strobel, 1998). Second, students' perceptions could also be aggregated at the school level to obtain a less subjective estimate of school characteristics. Third, structural school characteristics could be directly evaluated through observation, school records, and demographic information (architectural design, school size, curricular diversity, students' and teachers' demographic characteristics, deterioration of school buildings, etc.). Both the second and third approaches represent attempts to more objectively evaluate school environment characteristics as potential sources of influence on student development (Anderman, 2002; Moos, 1979; Olsson & VonKnorring, 1999). Unfortunately, we are aware of no studies meeting our inclusion criteria in which the relation between aggregated or structural characteristics of schools and depression development was directly evaluated.

Empirical Support

Interactions with school adults

Only two studies that met our inclusion criteria focused on the relations between students' interactions with school adults and depression development. One of those used a sample of children (Sim, 2002), while the other was based on a sample of adolescents (Roeser & Eccles, 1998). Both of these studies demonstrated that higher levels of teacher support and acceptance were related to a lower risk of developing depression, although this effect may be limited to females, at least in childhood (Sim, 2002).

School psychological environment

Only one of the studies in which the effects of perceived school environment on depression development were evaluated met our inclusion criteria. This study, which was based on a sample of adolescents, observed no relationships between school facilitation of students' autonomy - an aspect of schools' disciplinary practices - and students' levels of depression (Roeser & Eccles, 1998). The authors also noted that students who perceived their schools as emphasizing learning over achievement presented a lower risk of developing depression, whereas those who perceived their schools as places where getting good grades is more important than personal development and learning presented a higher risk. Until replication studies can be conducted, these results certainly do not allow us to draw strong conclusions about the role of school psychological environment on depression development.

Summary

The results presented in this section indicate that at least some aspects of school life may play a role in depression development. For instance, students' positive relationships with teachers may diminish their risk of developing depression. Similarly, one study suggests that students who perceive their schools as emphasizing learning and competency development may present lower risk of developing depression. However, the promising character of these results still does not allow us to reach strong conclusions regarding the effects of school life on students' depression development: overall, the impact of school life on students' depression development has received relatively little scientific attention, especially in children.

Given the potential importance of schools in youngsters' development, we agree with previous scholars that the study of school effects should be considered a priority in depression development research (e.g., Boyce *et al.*, 1998; Rutter, 1999, 2000; Rutter *et al.*, 1997; Zaslow, & Takanishi, 1993). To guide efforts in this direction, three main limitations of current knowledge should be addressed in future studies. First, the impact of many facets of school life on depression development remains to be

evaluated in an integrated and coherent fashion. For instance, it is highly probable that most of the observed relations could vary according to objective school characteristics (aggregated students' perceptions or structural characteristics) whose effects have yet to be systematically evaluated. Second, in the design of such studies, additional controls should be provided for individual background characteristics and for out of-school socialization experiences, to account for the fact that these variables may strongly influence the quality of students' school life (Monroe & Simons, 1991; Mortimore, 1995; Rutter, 1999, 2000). Consequently, to demonstrate that school life characteristics are implicated in depression development, it should be demonstrated that the observed relations do not represent an artifact of students' background characteristics and socialization experiences. Finally, although this limitation was not apparent in the present section because very few studies met our inclusion criteria, the study of school life's effects on children's and adolescents' depression development has been plagued by a lack of conceptual agreement on what, exactly, should be included in the definition of school life. Whereas school life has been defined in many different ways (for examples, see Gottfredson, 1984; Moos, 1979; Moos & Trickett, 1974; Mortimore, 1995; Purkey & Smith, 1983; Rutter, 1983), most of these conceptualizations can be viewed as incomplete, missing some known important facets of school life. Moreover, this lack of consensus also limits the comparability of the results obtained in previous studies as most of them relied on different measures of school life characteristics.

To date, the strongest evidence of school effects on students' depression development comes from two independent prevention programs. In the first of these, Felner *et al.* (1993) built a program designed to facilitate students' elementary-to-middle school transfer. The central elements of this program include the formation of small groups of transitioning students who remain together during core classes ("schools within school") and a redefinition of the role of the homeroom-teachers to provide greater support and resources for students. Repeated evaluations of this program revealed that it had a clear impact on reducing students' levels of depression following school transition (Felner *et al.*, 1993). In the Mastery Learning program, Kellam and

colleagues (Kellam, Rebok, Mayer, Ialongo, & Kalodner, 1994) implemented an enriched first language curriculum and trained teachers to provide more individualized teaching. Additionally, they developed a formative evaluation process which stated that 80% of classroom students had to master 80% to 85% of the module's objectives before the teacher could move on. Interestingly, in addition to allowing students to get better grades, this program also reduced their depression levels. Unfortunately, both programs involved many components whose effects were not evaluated separately, so the causal processes involved in their effects could not be identified.

Neighborhood Environment

Theoretical Issues

In Bronfenbrenner's (1997) ecological model, neighborhood influence on the developing individual appears to lie somewhere between what he calls the meso- and exo- systems. Exosystem is an

> "extension of the mesosystem embracing other specific social structures, both formal and informal, that do not themselves contain the developing person but impinge upon or encompass the immediate settings in which that person is found, and thereby influence, delimit, or even determine what goes on there." (Bronfenbrenner, 1977, p. 515)

Indeed, it is within neighborhoods that the relationships between children, families, and schools develop. As such, neighborhood characteristics may exert an impact on each of these more specific systems, as well as on their interrelations, indirectly influencing children's development (Reiss, 1995). Moreover, neighborhoods may also more directly create socialization opportunities for children by influencing their exposure to specific institutions (youth groups, community centers, hospitals) and to agreeable or unpleasant aspects of community life (violence, disorder, supportive neighbors) (Ingoldsby & Shaw, 2002; Leventhal & Brooks-Gunn, 2000; Perkins & Taylor, 1996). For example, neighborhood street gangs may exert an influence (drug, crime) within schools or may terrorize citizens, making it more likely that the most

wealthy or healthy families will move out of the neighborhood. Poverty rates will then rise in the neighborhood, making it more likely that street gangs will endure. Children living in this neighborhood will thus be more likely to attend more problematic schools and to be exposed to community violence. Overall, as everybody is exposed, lifelong, to neighborhoods, the society-level impact of this variable may be quite important.

Neighborhood effects on depression development in children and adolescents received relatively little scientific attention during the last decade. Given that research on neighborhood effects faces the same limitations as school life studies, this observation is not surprising. Moreover, because neighborhoods represent even more complex social systems than schools, these limitations may be even more salient. For instance, whereas it is quite simple to define the territorial boundaries of a school, neighborhood territorial boundaries appear to be more diffuse. Indeed, the term "neighborhood" refers to spatially-bounded communities, but also implies a social reality in addition to a structural one (e.g., Coulton, Korbin, & Su, 1996; Ingoldsby & Shaw, 2002; Leventhal & Brooks-Gunn, 2000). Different approaches have thus been used to draw neighborhood limits (Coulton *et al.*, 1996): (a) city block groupings (official boundaries); (b) groupings based on the mapping-out of social interaction patterns among residents (sociological boundaries); and (c) absence of groupings and reliance on residents' self-reports of "their" neighborhoods' reality following the postulate that each one of them has a idiosyncratic sense of his/her neighborhood boundaries (phenomenological boundaries).

This lack of consensus on the exact nature of neighborhood boundaries does not ease the task of researchers interested in identifying the impact of neighborhood characteristics on children's and adolescents' psychosocial development. Moreover, this lack of consensus also encompassed the description of neighborhood characteristics. Indeed, most authors still tend to use their own idiosyncratic vision and measures of neighborhood environment. However, a deeper look at studies and reviews on such issues revealed that, as it was the case for schooling, most proposed neighborhood characteristics can be grouped into three dimensions (e.g., Coulton *et*

al., 1996; Leventhal & Brooks-Gunn, 2000; Reiss, 1995): (a) youths' socialization experiences involving neighborhood members (victimization, violence exposure, and relationships with neighbors); (b) youths' personal (neighborhood psychological environment) or aggregated perceptions of their neighborhoods; and (c) structural neighborhood characteristics (architecture, demographic, and ethnic composition, residential instability, job availability, etc.).

Empirical Support

We located only one study meeting our inclusion criteria that evaluated the impact of neighborhood characteristics on depression development (Lynch & Cicchetti, 1998). In this study, the authors evaluated the impact of children's exposure to community violence and found no effect of such exposure on depression development. As previously noted, the specific nature of the sample used (i.e., more than half of the children were known cases of maltreatment) in this study seriously limits the extent to which these findings can be generalized.

Summary

As it was the case for the study on the impact of school life on depression development, a priority for researchers interested in the study of neighborhood factors on depression development should be to reach a conceptual agreement on how, exactly, to define neighborhood characteristics. Indeed, because neighborhoods represent highly complex social systems, real advances in the understanding of their effects on youths' depression development do not seem possible without the reliance on integrated conceptual models. The almost complete lack of scientific attention devoted to the role of neighborhood characteristics in depression development could certainly be explained by the combined effects of this lack of consensus and of the high monetary costs of research designed to evaluate the effects of neighborhoods on youth's development. Notwithstanding these difficulties, because of the potentially important societal impact of neighborhoods, such studies should still be seen as a priority for depression development research.

CONCLUSION

Scientific study of depression development in children and adolescents has grown significantly over the last two or three decades. Indeed, depression development is now such a vibrant and active field of scientific inquiry that it is hard to believe that not so long ago, depression was seen as an impossible state in children and adolescents (see Claes, 1983). The current knowledge base is impressive and offers very promising hypotheses regarding the role of individual and social factors in depression development.

Individual Antecedents

Evidence is rapidly growing of the impact of at least some individual factors on depression development. Some antecedents of depression development have been clearly identified. In fact, current results indicate that children and adolescents presenting high levels of Neuroticism (or emotional reactivity), Introversion, conduct disorders, psychoactive substance abuse, and anxiety-related disorders or low levels of self-esteem or self-perceived competencies, particularly in the social, academic, and body-image areas, seem to be at a higher risk of developing depression. Preliminary evidence also suggests that negative attributional style, low reliance on problem-solving coping strategies, high reliance on ruminative or avoidant coping strategies, and poor health and health behaviors may also represent risk factors for depression development. More studies are clearly needed to investigate the effects of the latter factors, as well as the effects of additional factors for which current evidence is either absent, contradictory, or limited to cross-sectional studies, such as Conscientiousness, Agreeableness, and Openness to experience, vulnerable self-schemas, sexual orientation, intelligence, musical preferences and behaviors, religiosity, and Internet use.

Nevertheless, despite these encouraging results, conclusions from many previous reviews that attributed a determining role to individual factors in depression development appear unwarranted in light of the present review. Indeed, whereas individual factors are by far the most studied and cited hypothetical antecedents for depression development in psychological research, possibly because of the traditional tendency of psychology to focus on individuals' internal characteristics, evidence of the role played by many such factors in depression development remains scarce and limited to a few prospective studies. As Coyne (1999) cynically, yet adequately, puts it:

> "Too often what is involved in such research is that depressed individuals' statements about themselves and their relationships automatically get interpreted as evidence of enduring cognitive structures, a sociotropic trait, or working models of relationships, and these reified entities are then given causal priority over any interpersonal process." (p. 368) "The sheer repetitiousness of such claims has seemingly given them an unwarranted credibility; in each successive study, prior papers are cited in which such claims were also made without empirical evidence." (p. 369)

To solve this problem, Coyne (1999) stresses the need to go beyond what he calls the "fundamental attributional error in depression theory and research" (p. 369), or the tendency to attribute risk to fixed individual factors, and to focus on interpersonal and environmental factors. This review clearly supports this proposition and indicates that individual antecedents of depression cannot be efficiently studied without simultaneously considering mediating and moderating relationships involving environmental and interpersonal factors. For instance, problematic cognitive style or vulnerable self-schemas most likely contribute to depression development by increasing youths' sensitivity to the effects of congruent environmental stressors. Self-esteem, self-perceived competencies, and coping styles, in addition to their direct impact on depression development, may also serve as protective factors for individuals exposed to stressful life events. Likewise, the effects of Neuroticism on depression may be mediated by its impact on stress-reactivity and exposure, whereas the effects of Introversion are most likely mediated by its impact on social support

and interpersonal conflicts. Unfortunately, most of these proposed person-environment interactions were not systematically evaluated in the studies reviewed and must be seen as theoretical propositions rather than as definite results. Moreover, whereas multiple hypotheses were formulated to explain the potential role of individual factors in depression development, few of the studies reviewed were designed to systematically test these hypotheses. One exception is the diathesis-stress hypotheses of the cognitive theories of depression, which received at least partial empirical support. This last result further reinforces Coyne's suggestion to move beyond single variable studies toward more integrated studies in which it will be possible to consider the combined and interactive effects of individual and environmental factors.

Social Antecedents

When it comes to the role of social factors in depression development, the results clearly indicate that children and adolescents exposed to episodic or chronic stressors, marital conflicts, parent-child conflicts, physical/sexual abuse and neglect, peer rejection, and peer hostility present an increased risk of developing depression, especially if they already present a high level of individual vulnerability. Similarly, children living in families characterized by high levels of parental support and interacting with receptive and supportive peers appear to present a lower risk of developing depression. Preliminary evidence also suggests that changes in family structure may indirectly influence depression development by exerting a destabilizing impact on family relationships. Besides, the effects of parental divorce may also be moderated by other family factors, such as marital conflict and the quality of the resulting relationships between children and paternal figures. For most of these factors, a generic trend apparent throughout this review suggests that girls may be more sensitive than boys to the deleterious or positive impact of factors involving close relationships, such as peers and family members.

Conversely, the relationships between many other social factors and depression development have not been sufficiently studied. For instance, previous studies almost completely neglected to evaluate the potential impact of parental control and monitoring, affiliation with deviant (alcohol-using or depressed) peers, social integration, romantic involvement, school life, and neighborhood environment on depression development. Clearly, one priority of depression research should be to concentrate on the impact of these factors, given the important role they are known to play in children's and adolescents' psychosocial development.

Once again, very few of the studies reviewed attempted to systematically validate the various hypotheses formulated to explain the mechanisms involved in the effects of social factors on depression development. Indeed, except for some studies which showed that the effects of stressors on depression development were moderated by cognitive factors, the proposed relationships between social factors and depression development were generally studied in isolation. For example, whereas neighborhoods are purported to have an indirect impact on youths' development through their effects on family and school functioning, very few studies evaluated these propositions. None of them met our inclusion criteria. Additionally, very few studies attempted to take into account the fact that individuals may choose and modify their environments (i.e. that personal risk factors may be involved in individuals' exposure to risky environments). Whatever the results, it is unlikely that this compartmentalized view of depression development would someday be found to represent human the full complexity of human beings.

An Integrated Synthesis

This last comment could possibly represent the most important conclusion of this review. Indeed, so many individual and social factors have been reported to be undoubtedly or possibly implicated in child and adolescent depression development that it is unlikely that a complete understanding of the mechanisms involved could be reached through studies relying on such a compartmentalized view of human

development. Whereas significant advances have been made on this topic over the last decade and the call for multidisciplinarity appears to have been answered in depression development research (e.g., Mrazek and Haggerty, 1994), we believe that the full potential of this multidisciplinarity remains underused. Indeed, while most depression-development-oriented research groups now comprise scholars from different backgrounds, they still tend to focus on a limited number of potential antecedents and to verify compartmentalized hypotheses with increasingly complex research designs. There are two possible explanations to this phenomenon. First, the number of articles published on the subject of depression development is so high that few scholars have the time to sort through them all. This difficulty is further reinforced by the current "publish or perish" context which forces scholars to publish as much as they can, thus multiplying small scope studies.

The need to move beyond this fragmented and compartmentalized view of depression development is reinforced by the present review. Indeed, very few (if any) of the factors involved in depression development were found to individually explain more than 5% of depression variance. Conversely, in studies where multiple antecedents were simultaneously considered, the percentage of explained variance in depression development increased to 30%-40% (Bandura *et al.*, 1999; Joyner & Udry, 2000; Roeser & Eccles, 1998; Spence *et al.*, 2002; Windle, 1992, 1994). Lewinsohn *et al.*'s (1994) study probably provides the strongest example of this phenomenon. Indeed, using a stepwise logistic regression analysis in which multiple risk factors were simultaneously considered to predict later depressive onsets, these authors noted that the best fitting set of predictors allowed for 67.3% of the participants to be classified into cases and non-cases of depression, with a sensitivity of 66.9% and a specificity of 71.4%. Unfortunately, current theoretical models of depression development, which seldom consider more than three to five variables, are still insufficient to support integrated research designs. Indeed, very few attempts have been made to theoretically depict the full complexity of depression development: (a) Cicchetti and Toth's (1998) application of the organizational theory of human development to children of depressed parents psychosocial development; (b) Goodman and Gotlib's

(1999, 2002) attempt to provide a broad overview of the biopsychosocial factors interfering with the psychosocial development of children of depressed parents; and (c) Cyranowski *et al.*'s (2000) psychobiological model of the emergence of gender differences in depression rates. Conversely, even these models are limited in that they only focus on specific subject subgroups.

Despite the limitations outlined above, the knowledge base on depression development has experienced a major evolution over the last decade. Indeed, the present review clearly indicates that we now know enough to support the development of such an integrated conceptual vision of depression development. Moreover, the emergence of a new developmental psychopathology paradigm in developmental research (Compas *et al.*, 2002; Rutter, 1986; Rutter & Sroufe, 2000), together with the associated organizational (Cicchetti & Rogosh, 2002; Cicchetti & Schneider-Rosen, 1986), transactional (Sameroff, 2000; Sameroff & Chandler, 1975), person-environment mismatch (Eccles *et al.*, 1991, 1993), and ecosystemic (Bronfenbrenner, 1977) theories, provides the interested scholars with powerful tools from which to build such an integration.

Briefly, the organizational theory of human development postulates that psychosocial adaptation depends on the quality of the organization within and between an individual's internal and behavioral subsystems and that human development progressively unfolds as the result of successive reorganizations within and between these subsystems (Cicchetti & Rogosh, 2002; Cicchetti & Schneider-Rosen, 1986). These successive reorganizations are generally referred to as "developmental tasks" and are proposed to be associated with a global restructuring of the internal organization of individual systems and to be triggered by the reorganization or modification of the environmental systems to which the individual is exposed (Erickson, 1968; Gladstone & Beardslee, 2002; Goodman, 2002; Petersen & Lefert, 1995; Piaget, 1972). In a complementary way, the transactional theory postulates that human development unfolds as a consequence of successive bidirectional transactions among individuals and their environments (Sameroff, 2000; Sameroff & Chandler,

1975), and the person-environment mismatch theory indicates that psychosocial problems result from a mismatch between individuals' competencies and needs, and environmental resources and restrictions (Eccles *et al.*, 1991, 1993). Finally, the ecosystemic model of human development postulates that human development unfolds under the influence of multiple environmental systems which are, themselves, interdependent (Bronfenbrenner, 1977).

Overall, the integration of these models with the results from the present review suggest that depression most likely represents the final common pathway of a multiplicity of environmental and individual factors interacting with each other in a reciprocal manner. What remains to be evaluated is which specific forms of person-environment discrepancies will lead to depression as compared to other psychopathologies. However, the developmental perspective of these models also suggests that different factors, or combinations of factors, may be implicated in depression development at different life stages and that life transitions are associated with biopsychosocial reorganizations of sufficient magnitude to radically modify youths' life trajectories. This integration clearly underlines three very important and stimulating challenges now facing depression development research.

The Methodological Challenge

The first of these challenges is a methodological one and refers to three elements. First, even though new hypotheses could be tested with less than optimal exploratory designs, the knowledge base regarding most of the risk and protective factors implicated in depression development is so extensive that scholars should now attempt to rely on more solid methodologies. Indeed, although many studies published during the last decade attempted to identify the mechanisms involved in depression development, very few of them met our inclusion criteria. Although some factors, such as Internet use and musical behaviors, may still deserve cross-sectional designs or validation through already available databases, our knowledge of the precise role of the other factors implicated in depression development would now

greatly benefit from stronger methodological designs. For instance, prospective longitudinal studies and accelerated longitudinal designs of large samples (to provide sufficient statistical power for complex analytical schemes) allowing for the transactional evaluation of the interactive effects of multiple individual and environmental risk and protective factors on multiple outcomes (depression, anxiety, etc.) are sorely needed. More refined methodological and statistical suggestions on this topic can be consulted in many recent publications (Curran & Willoughby, 2003; Granic & Hollenstein, 2003; Rutter, 2000; Rutter *et al.*, 1997; Sameroff & Mackenzie, 2003; Sullivan, 1998; Willett, Singer, & Martin, 1998).

Second, the operationalization of many potential antecedents of depression development appears to urgently need scientific attention. For example, cognitive vulnerabilities or vulnerable self-schemas have been operationalized in so many different ways that the results of studies based on these concepts are increasingly hard to compare. Moreover, an increasing amount of evidence indicates that many of the psychological risk factors purported to be implicated in depression development, such as vulnerable self-schemas, neuroticism, self-esteem, and self-efficacy, may in fact represent the same underlying vulnerability. For example, Roberts and Kendler (1999) concluded that self-esteem did not contribute to predicting depression once Neuroticism was taken into account. Moreover, in a paper combining a meta-analysis with three studies based on seven samples, Judge, Erez, Bono, and Thoresen (2002) revealed that self-esteem, locus of control, Neuroticism, and generalized self-efficacy were so interrelated as to be best represented as a single higher-order construct. New approaches are beginning to bridge these different concepts. For example, efforts to distinguish the effects of perceived competencies in different domains (instead of generic self-esteem) from those of "objective" competencies are a first step in this direction (e.g. Cole *et al.*, 1998; 1999). Clearly, additional attempts to integrate these different areas of inquiry and to build bridges across domains will be needed if we hope to someday reach a consensus on the role of individual's characteristics in depression development.

Conversely, whereas the study of individual factors implicated in depression development suffers from this form of "over-operationalization," the study of social risk factors suffers more from an "under-operationalization." Indeed, most scholars interested in the study of social factors on depression development tended to rely on generic life event scales without considering that the various circumstances measured by these scales were likely to exert differential impacts. Other than family-level influences, whose operationalization received considerable attention, peer groups, school life, and neighborhoods received a very limited amount of scientific consideration. Indeed, we rarely found two studies in which identical aspects of environmental influences were measured with similar instruments.

Third, although the studies reviewed often provided control for previous levels of depression, these controls were often insufficient to disentangle the effects of antecedents on the emergence versus aggravation of depressive states. Briefly, statistical controls of previous levels of depression are generally used to account for the bidirectionality of the relationships observed between depression and purported risk factors. For example, depression represents a known predictor of school adaptation problems (Kessler, Foster, Saunders, & Stang, 1995). Consequently, to clearly conclude that school adaptation problems predict depression development, one must demonstrate that the effects observed are not due to students' baseline levels of depression. Although doing so allows depression antecedents to be identified more clearly, it remains insufficient for research in which the ultimate goal is to guide preventive or clinical efforts. In fact, depression prevention programs usually target non-depressed individuals and strive to help them to remain well (Morin & Chalfoun, 2003; Mrazeck & Haggerty, 1994). Prevention programs should therefore be based on risk factors related to the emergence of depression rather than on factors related to its aggravation. For instance, if a risk factor predicts elevated levels of depression in already depressed individuals only and shows no relationship with depression development in previously well individuals, this factor would be useless for preventionists, but very useful for clinicians. Kessler (1997) therefore urges scientists

to systematically verify if the relationships between risk factors and depression development are moderated by subjects' baseline levels of depression.

The Theoretical Challenge

The second challenge is of a theoretical nature and covers three elements. These three elements can be subsumed under the fact that they refer to attempts to provide an integrated picture of the many mechanisms involved in depression development. First, this review indicated that whereas classical determinants of child and adolescent development, such as psychological functioning, stressors, and family environment, received considerable scientific attention, many environmental factors were generally neglected. The obvious lack of scientific attention devoted to the effects of peer groups, school life, and neighborhoods on children's and adolescents' depression development is surprising, especially because most broad theories of human development agree that these factors may play an important role in the overall quality of psychosocial development. Although this may be due to the aforementioned absence of consensus regarding the operationalization of these factors, this lack of attention represents a serious limitation to our understanding of depression development. We therefore believe that the study of these factors should become a priority for depression research in the next decade. In a related way, although we excluded these factors from the present review, recent results strongly indicate that a complete understanding of the psychosocial mechanisms implicated in depression development will not be possible without the simultaneous consideration of biological risk and protective factors (Caspi *et al.*, 2003; Goodyer, Herbert, Tamplin, & Altham, 2000; Susman, Dorn, Inoff-Germain, Nottelman, & Chrousos, 1997). Indeed, these results suggest that neuroendocrine (Goodyer *et al.*, 2000; Susman *et al.*, 1997) and genetic (Caspi *et al.*, 2003) factors could be implicated in youths' stress reactivity to environmental stressors and thus represent important moderators of the stress-depression relationship (for a related discussion, see Curtis & Cicchetti, 2003).

Second, integrated theoretical models should be directly evaluated. Indeed, there is a dire need for depression research to focus on the simultaneous evaluation of the impact and reciprocal interactions among the many aforementioned psychosocial factors. Clearly, given the complexity of depression development, such integration would require the use of strong theoretical bases to allow the direct evaluation of integrated research questions and hypotheses. So many factors appear to be implicated in depression development that each specific case of depression is unlikely to result from the combined action of all of these mechanisms. Alternate research designs should be used to account for this phenomenon. Foremost, protective factors potentially implicated in depression development should be studied more thoroughly. Preliminary evidence presented in this review suggests that self-esteem, perceived competencies, social skills, problem-solving coping orientation, parental support, and supportive peer behaviors may represent protective factors for youths exposed to stressful life events. However, the protective role these factors play in depression development was evaluated in no more than one or two studies, and the potential protective role of additional variables, such as teacher support, has yet to be empirically verified. Similarly, other forms of moderating or mediating relationships between these various factors are yet to be systematically evaluated (for a more detailed discussion of these topics, see Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001; Sher, Gotham, & Watson, 2004). Clearly, the proposed conceptual framework strongly suggests that the various factors implicated in depression development interacted with each other in a transactional manner. The multiple forms of transactional relationships have yet to be systematically evaluated.

Furthermore, in addition to classical variable-centered analytical strategies such as those used in the studies reviewed here, future studies need to rely more often on person-centered analyses (Von Eye & Bergman, 2003). Person-centered analyses are rapidly gaining popularity in developmental research, although this popularity does not seem to have spread into depression development research yet. The main reason for this popularity is that classical variable-centered approaches usually portray relations as they apply to the average individual. As this average individual seldom

exists, person-centered approaches were designed to consider individual variability regarding cross-sectional and longitudinal patterns of risk factor aggregation and symptomatic expressions (Bergman, 2000; Nagin, 1999; Von Eye & Bergman, 2003). Person-centered approaches thus provide an interesting way to complement traditional analyses while considering inter-individual variability in the observed associations. For this reason, we strongly advocate that scholars design longitudinal studies in which the various time-dependant variables (versus stable) will be measured at each time point. Such repeated-measures longitudinal designs will allow person-centered specialists to complement classical variable-centered analyses while taking into account the full richness of inter-individual variability.

Third, integrated reviews should constantly be produced to guide research efforts. First and foremost, other scholars should attempt to repeat reviews such as this one. Indeed, this review, as many previously published ones, may have missed some key studies that would allow for more refined interpretations. For instance, for the sake of brevity, prevention trials were omitted from this review even though such studies do represent an ethically admissible way of experimentally testing etiological theories (e.g., Howe, Reiss, & Yuh, 2002; Morin, & Chalfoun, 2003). Moreover, our own theoretical bases (systemic and developmental) may have influenced our reading and understanding of the results. As an example, the decision to limit ourselves to studies in which "pure" measures of depression were used, although justified, means that studies relying on mixed measures of internalizing disorders were not reviewed. However, these studies represent a significant proportion of the studies based on children's populations. Consequently, other scholars may come to very different conclusions based on the same empirical evidence, and this difference would allow for an even more refined understanding of the mechanisms involved in depression development. As an example of such a discrepancy, interested readers may compare Mrazek and Haggerty's (1994) and Durlak and Wells' (1997) reviews.

The Generalization Challenge

The third challenge involves the generalization of results. Indeed, even though current knowledge about depression development appears impressive, it remains seriously limited by the relative neglect of the universality or specificity of the mechanisms identified. Indeed, the current research base is limited to European or North American Caucasian youths, and very few attempts have been made to evaluate the possibility of generalizing the identified mechanisms to other cultures or ethnic groups. In fact, preliminary evidence indicates that these mechanisms may vary according to ethnicity (Choi, 2002; Hill, Bush, & Roosa, 2003; Lee & Larson, 2000; Rumbaut, 1994; Tsai & Chentsova-Dutton, 2002). Moreover, and perhaps more importantly, the potential gender-based differences and developmental variability in the effect of the various antecedents were rarely evaluated systematically in the reviewed studies. In this review, preliminary evidence indicated that some results differed according to subjects' gender and developmental stage. For instance, preliminary results suggest that a negative cognitive style may increase adolescents', but not children's, reactivity to congruent stressors. Conversely, the protective role of self-esteem against the deleterious effects of stressors may be limited to children rather than adolescents. The positive effects of parental support also appear be stronger in childhood than in adolescence. Additionally, the effects of many social factors on depression development appeared to be more important for girls than for boys, and body image dissatisfaction seemed to increase only adolescent girls' risk of developing depression. As children's and adolescents' developmental tasks greatly differ, most of the results presented cannot be expected to be similar in child and adolescent populations. Similarly, given the known gender differences in adolescent depression prevalence, at least some of the mechanisms involved in depression development could be expected to differ in males and females. For these various reasons, theories of depression development should be able to explain the fact that before adolescence, prevalence rates of depression are the same among boys and girls whereas adolescent girls and adult women present rates of depression twice as great as those of their male counterparts.

Preventing Depression

Although answering the previously identified challenges will doubtless greatly assist the design of preventive interventions, current knowledge already suggests important guidelines. First, many factors were found to be implicated in depression development and the relation between most of these factors and depression development were generally small. Early screening procedures designed to identify at-risk subjects should therefore be refined to include severe risk factors that are sure to create a very high level of non-specific risk in exposed youths (such as sexual abuse), as well as combinations of less severe risk factors. As screening tools for identifying subjects at risk of depression are almost non-existent, intensified efforts will be needed in this area (e.g., LeBlanc & Morizot, 2000).

Similarly, as depressive episodes most likely occur following the combined or interactive action of multiple mechanisms, preventive and curative interventions for depression should be refined to allow for the simultaneous consideration of at least some of the many individual and social mechanisms likely involved. Interestingly, a previous review of depression prevention reached a very similar conclusion by demonstrating that, to date, the most effective prevention programs attempted either to modify individuals' adaptation to their environment or to improve the ability of educative environments to efficiently meet individuals' developmental needs (Morin & Chalfoun, 2003).

Finally, the few studies in which systematic attempts were made to differentiate the impact of psychosocial risk factors on the emergence versus the aggravation of depressive symptoms suggest that some factors may be more valid targets for preventive programs than others. For instance, preliminary evidence suggests that the effects of stressful life events, romantic breakups, and parental divorce may be limited to the emergence of depressive symptoms rather than to their aggravation. Conversely, the deleterious effects of a negative attributional style may be limited to the aggravation of depressive symptoms. Consequently, preventive interventions

would likely be more effective if they targeted exposure to stressors, parental divorce, and romantic breakups (or individuals' ability to face such events) than if they targeted youths' attributional style. Again, this conclusion supports those of a previous review in which it was suggested that preventive interventions directly anchored in the results from risk and protective factor research were generally more effective than those inspired by successful treatment programs (Morin & Chalfoun, 2003).

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APPENDICES

Appendix A

Methodological characteristics of the 91 selected studies

Authors	Country	Subjects	% males/ % females	Age	Duration (number of measurement points)	Instrument (s)
Abela (2001).	USA	S1: 230 3rd graders. S2: 152 7th graders.	S1: 53.3 / 46.7 S2: 52.6 / 47.4	S1: 8.9 S2: 12.9	6 weeks (6).	CDI.
Adams and Adams (1993).	USA	150	51.3 / 48.7	14-18	1 semester (2).	RADS.
Allgood-Merten, Lewinsohn and Hops (1990).	USA	664	45.7 / 54.3	13.26-18.80 (9th to 12th grade)	1 month (2).	CES-D.
Bandura, Pastorelli, Barbaranelli and Caprara (1999).	Italy	282	52.5 / 47.5	11.5	2 years (3)	Aggregated: CDI and peers/teacher evaluations.
Boivin, Hymel and Bukowski (1995).	Canada	567	49.2 / 50.8	10.8 (9-12; 4th-5th grade)	1 year (2).	CDI
Boney-McCoy and Finkhelor (1996).	USA	1433 Representative sample	53.9 / 46.1	NR	15 months on average, 8 to 24 (2).	DIS (T1 = lifetime; T2 = past month).
Brent, Moritz, Bridge, Perper and Canobbio (1996).	USA.	S1: 132 friends of suicide victims S2: 141 controls	S1: 51.5 / 48.5 S2: 54.9 / 45.1	S1: 21.2 (T2) S2: 20.4 (T2)	2.8-2.9 years (T1: 7 months after peer suicide; T2, 2.8-2.9 years after).	K-SADS.
Brent, Perper, Moritz, Liotus, Schweers and Canobbio (1994).	USA.	S1: 121 friends of suicide victims S2: 138 controls	S1: 54.2 / 45.8 S2: 43.6 / 56.4	S1: 19.8 (T2) S2: 18.8 (T2)	12-18 months (T1: 7 months after peer suicide; T2, 12-18 months after).	K-SADS.
J. Brown, Cohen, Johnson and Smailes (1999).	USA	639	52.3 / 47.7	5 (1-10)	17 years (4).	DIS-C. No control of T1 D but control of numerous early familial risk factors.
R.A. Brown, Lewinsohn, Seeley and Wagner (1996).	USA	1507	48 / 52	16.6	Average of 13.8 months (2).	T1: K-SADS. T2: LIFE. Incidence.
Capaldi and Stoolmiller (1999).	USA	201 boys (high crime area)	100 / 0	NR (6th grade at T1, 12th at T2).	6 years (2).	6th grade: CDRS 12th grade: CES-D.
Caspi, Moffitt, Newman and Silva (1996).	NZ	962 Representative sample.	50 / 50 (close to)	Birth cohort	21 years (ages 0, 3, 5, 7, 9, 11, 13, 15, 18, 21).	DIS (at age 21).

Authors	Country	Subjects	% males/ % females	Age	Duration (number of measurement points)	Instrument (s)
Chase-Lansdale, Cherlin and Kiernan (1995).	UK	10353 Representative sample.	50 / 50 (close to)	Birth cohort	23 years (ages 0, 7, 11, 16, 23).	Malaise Inventory (age 23) and teacher / parent reports of internalizing (ages 7 & 16).
Cheng (1998).	China	167	40 / 60	14.87 (12-17)	6 months (2).	BDI.
W.S. Choi, Patten, Gillin, Kaplan and Pierce (1997).	USA	6863	NR	12-18	4 years (2).	Kandel and Davies (1982) 6 items Depression Scale (dichotomized). Incidence
Cole, Jacquez and Maschman (2001).	USA	Average of 468 per data collection point.	50 / 50 (close to)	8.9 (3rd grade)	4 years (8).	CDI.
Cole, Martin, Peeke, Seroczynski and Fier (1999).	USA	S1: 349 3rd graders S2: 458 6th graders	S1: 52.1 / 47.9 S2: 46.1 / 53.9	S1: 8.9 S2: 11.9	2 years (4).	CDI.
Cole, Martin, Peeke, Seroczynski and Hoffman (1998).	USA.	S1: 201-261 3rd graders S2: 210-318 6th graders	S1: 47.1 / 52.9 S2: 53.2 / 46.8	S1: 8.8 S2: 11.9	2 years (4).	CDI
Cole, Martin, Powers and Truglio (1996).	USA	S1: 490 3rd graders S2: 455 6th graders	S1 & S2: 50.8 / 49.2	S1: 8.37 S2: 11.36	6 months (2).	Aggregated: self and parent CDI, peer nomination index, and teacher rating index.
Daley, Hammen, Davila and Burge (1998).	USA	134 girls	0 / 100	18.29 (16-19)	2 years (4; college transition).	SCID
Daley, Hammen and Rao (2000).	USA	128 girls	0 / 100	18.29 (16-19)	5 years (5; college transition).	SCID
Davies and Windle (2001).	USA	360	51 / 49	15.5 (10th and 11th grade)	2 years (4).	CES-D
DuBois, Felner, Bartels and Silverman (1995).	USA	435	40.9 / 59.1	12.5 (9-16)	2 years (2).	CDI (categorical), 4 groups based on T1 and T2 D levels.
Duggal, Carlson, Sroufe and Egeland (2001).	USA	168	54.2 \ 45.8	Birth cohort	17.5 years (ages: 12, 18, 24, 30, 42, 48, 54 and 64 months and 6, 7, 8, 11, 16, and 17 years).	Aggregated (continuous and categorical): CBCL, YSR, TRF, CDRS, K-SADS (1st, 2nd and 3rd grades, and ages 16 and 17.5 combined).

Authors	Country	Subjects	% males/ % females	Age	Duration (number of measurement points)	Instrument (s)
Fergusson, Horwood and Lindsey (1996).	NZ	1019	50 / 50 (close to)	Birth cohort	18 years (ages 0, 4 months, annual from 1 to 16, 18).	CIDI at age 18 (previous 2 years) No control of D but control of numerous early background risk factors.
Fergusson and Linskey (1997).	NZ	1025	50 / 50 (close to)	Birth cohort	18 years (ages 0, 4 months, annual from 1 to 16, 18).	CIDI at age 18 (previous 2 years). See previous study.
Fergusson and Woodward (2000).	NZ	488 girls	0 / 100	Birth cohort	18 years (ages 0, 4 months, annual from 1 to 16, 18).	CIDI at age 18 (previous 2 years). See previous study.
Garber, Keiley and Martin (2002).	USA	240 of whom 185 with maternal history of D.	45.8 / 54.2	11.86	5 years (6).	Adolescent: CDI, YSR. Mother: CDI, CBCL.
Garrison, Jackson, Marsteller, McKeown and Addy (1990).	USA	550	55 / 45	11-15 (7^{th} grade)	3 years (3).	CES-D.
Ge, Best, Conger and Simons (1996).	USA	388 families	47 / 53	NR (7^{th} grade)	3 years (4).	T1 to T3: SCL-90-R. T4: CES-D, TRF, and NEO-PI (parents).
Ge, Lorenz, Conger, Elder and Simons (1994).	USA	376 (sibling sample)	49.2 / 50.8	12.9 (9-17)	3 years (4).	SCL-90-R.
Hammen, Henry and Daley (2000).	USA	121 girls	0 / 100	18.29 (16-19)	2 years (4: college transition).	SCID.
Hankin, Abramson and Siler (2001).	USA	270	43.3 / 56.7	16.18 (14-18; 9^{th} to 12^{th} grade)	5 weeks (2).	HDSQ-R and BDI (hopelessness, others, total).
Hilsman and Garber (1995).	USA	439	41.7 / 58.3	11.39 (5^{th} and 6^{th} grade).	10 days (2: T1; T2 = 1 day after report card; T3 = 5 days later).	CES-D for children.
K.B. Hoffman, Cole, Martin, Tram and Seroczynski (2000).	USA	360	49 / 51	11.9 (10.8-13.5; 6^{th} grade)	1.5 years (4).	CDI.
Hogue and Steinberg (1995).	USA	3677	43.6 / 56.4	NR (9^{th} to 11^{th} grade)	1 year (2).	Peer and self report 13-items CES-D.
Holsen, Kraft and Røysamb (2001).	Norway	645	50.5 / 49.5	13 (7^{th} grade)	5 years (3: age 13, 15, 18).	Depressed mood/ depressive tendencies scale (Alsaker, 1992).
Hops, Lewinsohn, Andrews and Roberts (1990).	USA	2160	30.3-48.2 / 51.8-69.7	16 (9^{th}, 10^{th}, 11^{th}, 12^{th} grade)	1 month (2).	CES-D: groups based on T1 and T2 categorical scores.

Authors	Country	Subjects	% males/ % females	Age	Duration (number of measurement points)	Instrument (s)
Jaffe, Moffitt, Caspi, Fombonne, Poulton and Martin (2002).	NZ	998	50 / 50 (close to)	Birth cohort	26 years (ages 0, 3, 5, 7, 9, 11, 13, 15, 18, 21, 26).	DIS-C (ages 11, 13, 15) and DIS (ages 18, 21, 26).
Jekielek (1998).	USA	1097	50.8 / 49.2	8-14	4 years (5).	BPI.
Joyner and Udry (2000).	USA	8181 Representative sample	47.2 / 52.8	12-17	1 year (2).	CES-D.
Kasen, Cohen, Brook and Hartmark (1996).	USA	648	49.2 / 50.8	1-10 (T1) and 9-18 (T2)	8 years (2).	Affective problems (T1) and DIS-C (T2).
Katainen, Räikkönen and Keltikangas-Järvinen (1999).	Finland	302 Representative sample	42.1 / 57.9	15	5 years (2).	BDI.
Katainen, Räikkönen, Keskivaara and Keltikangas-Järvinen (1999).	Finland	389	48.8 /51.2	6 at T1	9 years (3: ages 6, 9, 15).	Age 15 BDI and affective problems at T1.
Khatri, Kupersmidt and Patterson (2000).	USA	471	46 / 54	4th (10.3), 5th (11.4), and 6th (12.4) grade	1 year (2).	YSR.
Kiesner (2002).	Italy	215	51.2 / 48.8	13.2 (6th and 7th grade)	1 year (2).	CDI.
Krueger (1999).	NZ	961	50 / 50	Birth cohort	3 years (2).	DIS (major depression and dysthymia).
Lewinsohn, Allen, Seeley and Gotlib (1999).	USA	1507	48 / 52	16.6	Average of 13.8 months (2).	K-SADS and BDI at T1, LIFE at T2. Incidence.
Lewinsohn, Gotlib and Seeley (1995).	USA	1507	46.3 / 53.7	16.6	Average of 13.8 months (2).	K-SADS at T1, LIFE at T2. Incidence.
Lewinsohn, Joiner and Rohde (2001).	USA	1507	46 / 54.	16.6	Average of 13.8 months (2).	K-SADS at T1, LIFE at T2. Incidence.
Lewinsohn, Roberts, Seeley, Rohde, Gotlib and Hops (1994).	USA	1508	46 / 54	16.5	Average of 13.8 months (2).	K-SADS at T1, LIFE at T2. Incidence.
Lewinsohn, Seeley, Hibbard, Rohde and Sack (1996).	USA	1410	46.9 / 53.1	16.5	Average of 13.8 months (2).	K-SADS at T1, LIFE at T2. Incidence.
Lynch and Cicchetti (1998).	USA	245: 188 maltreated and 134 controls.	62.7 / 37.3	7-12	1 year (2).	CDI.
McFarlane, Bellissimo and Norman (1995).	Canada	682	52.3 / 47.7	NR (10th grade, under 19)	6 months (2).	Inventory to Diagnose Depression.

Authors	Country	Subjects	% males/ % females	Age	Duration (number of measurement points)	Instrument (s)
McGrath and Repetti (2002)	USA	227	53.2 / 46.8	9.5 (grade 4th)	2 years (3).	CDI.
McLeod and Shanahan (1996).	USA	613	NR	4-5	4 years (3).	CBCL.
Miech, Caspi. Moffitt, Entner Wright and Silva (1999).	NZ	939	50 / 50 (close to)	Birth cohort (ages 15, 21)	6 years (2).	DIS-C (age 15) and DIS (age 21).
Monroe, Rohde, Seeley and Lewinsohn (1999).	USA	1507	46.5 / 53.5	16.52	Average of 13.8 months (2).	K-SADS at T1, LIFE at T2. Incidence.
Nolen-Hoeksema, Girgus and Seligman (1992).	USA	255 to 508 (depending on data collection point)	NR	NR (3rd and 4th grade at T1)	4 years (9).	CDI
Palosaari, Aro and Laippala (1996).	Finland	1656	43.5 / 56.5	15.9 (9th grade)	6 years (2).	Self-esteem control (T1) and BDI-13 (T2).
Panak and Garber (1992).	USA	521	46 / 54	NR (3rd, 4th and 5th grade)	1 year (3).	CDI.
Pine, Cohen and Brook (2001).	USA	776	50 / 50 (close to)	13.7	9 years (3: mean ages 13.7, 16.4 and 22.1)	DIS-C.
Pine, Cohen, Gurley, Brook and Ma (1998).	USA	712	50 / 50 (close to)	13.7	9 years (3: mean ages 13.7, 16.4 and 22.1)	DIS-C.
Pomerantz (2001).	USA	806	48.5 / 51.5	11.68 (5th and 7th grade)	6 months (2).	CES-D
Rao, Hammen and Daley (1999).	USA	149 girls	0 / 100	18.29 (16-19)	5 years (6; college transition).	SCID
Reinherz, Giaconia, Hauf, Wasserman and Paradis (2000).	USA	360	50 / 50	Long term follow-up (age 5 to 21)	17 years (4: ages 5, 6, 9, 21).	Control of internalized behavior problems at ages 5, 6 and 9 and DIS at age 21.
Reinherz, Giaconia, Pakiz *et al.* (1993).	USA	385	50.6 / 49.4	Long term follow-up (age 5 to 18)	14 years (4: ages 5, 9, 15, 18).	DIS (age 18) with retrospective control of previous D.
Robinson, Garber and Hilsman (1995).	USA	239	42 / 58	12 (6th grade)	1 year (3; school transition).	CDI.
Roeser and Eccles (1998).	USA	1046 67% African American, 33% Caucasians	50 /50	NR (7th grade)	2 years (2).	SCL-90-R (T1) and CDI (T2).
Rohde, Lewinsohn, Kahler, Seeley and Brown (2001).	USA	940	43 / 57	14-18	8 years (3: mean ages 14-18 at T1; 17.8 at T2; 24 at T3).	K-SADS at T1, LIFE at T2, parent SCID at T3.

Authors	Country	Subjects	% males/ % females	Age	Duration (number of measurement points)	Instrument (s)
Rosenfield, Vertefeuille and McAlpine (2000).	USA	803	47 / 53	12-16 (8th and 10th grade)	1 year (2).	CES-D.
Rudolph, Kurlakowsky and Conley (2001)	USA.	S1: 471 5th graders S2: 587 5th and 6th graders	S1: 50.1 / 49.9 S2: 49.6 / 50.4	S1: 11.2 S2: 11.7	6-7 months (2).	CDI
Schwartz and Koenig (1996)	USA	285	35.1 / 64.9	15.99 (14-18; 9th to 12th grade)	6 weeks (2).	BDI
Sears and Armstrong (1998).	Canada	131	38.9 / 61.1	14.09 (12-17)	2 years (2).	BDI
Segrin and Flora (2000).	USA	102	34.7 / 65.3	17.9 (17-19)	6 months (2: before and after college transition).	BDI.
Seiffge-Krenke and Klessinger (2000).	Germany	194	46.9 / 53.1	13.9	3 years (4).	YSR
Siegel (2002).	USA	675	53.5 / 46.5	12-17	2.5 years (2).	CDI
Sim (2002).	Korea	187	51.3 / 48.7	9.8 (9-10; 4th grade)	2 years (3).	CDI
Slavin and Rainer (1990).	USA	333	47.1 / 52.9	15.5 (9th, 10th and 11th grade)	8 months (T1).	CDI
Spence, Sheffield and Donovan (2002).	Australia	733	54.1 / 45.9	12.91 (12-14; 8th grade)	1 year (2).	BDI.
Stein, Newcomb and Bentler (1996).	USA	461	28.9 / 71.1	7th to 9th grade at T1 and mean age of 25.47 at T4)	12 years (4: 4 years intervals – early adolescent, late adolescent, early adult, adult).	Controlling for negative affect at T1-T2, CES-D at T3-T4.
Stice and Bearman (2001).	USA	227 girls	0 / 100	14.9 (13-17; 9th and 10th grade)	20 months (3).	Burns Depression Checklist (Burns, 1997).
Stice, Hayward, Cameron, Killen and Taylor (2000).	USA	1124 girls	0 / 100	14.7 (13-16.9)	3 years (4).	CES-D (T1) and SCID (T2).
Stice, Presnell and Bearman (2001).	USA	496 girls	0 / 100	11-15	1 year (2).	K-SADS.
Sund and Wichstrøm (2002).	Norway	2360	49.2 / 50.8	13.7 (12.5-15.7)	1 year (2).	MFQ (dichotomized).
Tram and Cole (2000).	USA	468	55 / 46	14.5 (13-17; 9th grade)	6 months (2).	Aggregated CDI and peers/teachers evaluations.
Unger, Brown, Tressell and McLeod (2000).	USA	107	37.4 / 62.6	15.5 (12-18)	2 years (3).	CES-D.
Windle (1992 b).	USA	277	39 / 61	15.7	6 months (2).	CES-D.
Windle (1994).	USA	1098	48 / 52	16.21	6 months-1 year (2)	CES-D.

Authors	Country	Subjects	% males/ % females	Age	Duration (number of measurement points)	Instrument (s)
Windle and Windle (2001).	USA	1218	48 / 52	15.54 (10^{th} and 11^{th} grade)	1.5 years (4)	CES-D
Zimmerman, Ramirez-Valles, Zapert and Maton (2000).	USA	173 African-American boys. 69% school dropouts.	100 / 0	16.8	6 months (2)	BSI.

Abbreviations. *BDI*: Beck Depression Inventory; *BPI*: Behavior Problem Index; *BSI*: Brief Symptom Inventory; *CBCL*: Child Behavior Checklist; *CDI*: Children Depression Inventory; *CDRS*: Children's Depression Rating Scale; *CES-D*: Center for Epidemiological Studies Depression Scale; *CIDI*: Composite International Diagnostic Interview; *D*: Depression; *DIS(-C)*: Diagnostic Interview Schedule (-for children); *HDSQ-R*: Hopelessness Depression Symptom Questionnaire; *LIFE*: Longitudinal Interval Follow-up Evaluation; *MFQ*: Moods and Feelings Questionnaire; *NEO-PI*: NEO Personality Inventory; *NR*: not reported in paper; *NZ*: New Zealand; *RADS*: Reynolds Adolescent Depression Scale; *(K-)SADS*: (Kiddie-) Schedule for Affective Disorders and Schizophrenia; *SCID*: Structured Clinical Interview for DSM-IV Axis I Disorders; *SCL-90-R*: Symptoms Checklist 90-Revised; *TRF*: Teacher Report Form; *UK*: United Kingdoms; *USA*: United States of America; *YSR*: Youth Self Report.

Appendix B

Results of the 91 selected studies

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Abela (2001).	HMR (pr^2)	3rd: none. 7th: AS X LE (0.07 – LE effectslimited to negative AS). Both: catastrophization X LE (0.02 - see previous). m: none. f: self-blame X LE (0.03 – see previous).	None	3rd: LE (0.02). 7th: LE (0.05). Both: LE (0.03). m: LE (0.04). f: none.	3rd: AS, AS X LE. 7th: AS. Both: catastrophization. m: self-blame; self-blame X LE. f: LE; self-blame.
Adams and Adams (1993).	ANCOVA (NR)	None.	None	GPA change X perceived PS alternatives (More D if GPA drops and few PS alternatives).	GPA change; perceived PS alternatives.
Allgood-Merten *et al.* (1990).	HMR (ΔR^2)	m: SE (0.01). f: SE (0.03).	None.	m: LE (0.05). f: LE (0.01).	Self consciousness; feminity/ masculinity; body image; LE X individual variables.
Bandura *et al.*, (1999).	SEM (b)	T2: AC efficacy (-0.15); EB (0.18/0.19); SO efficacy (-0.14/-0.15). T3: EB (0.21/0.22); SO efficacy (-0.11).	None.	None.	T2: prosocial behaviour; AC achievement. T3: AC efficacy; prosocial behaviour; AC achievement.
Boivin *et al.* (1995).	HMR (ß)	None.	None.	Initial models: peer assessed social preference (0.015).	Initial models: peer assessed withdrawal and victimisation, loneliness. Final model[7]: peer assessed withdrawal victimisation, and social preference, loneliness.

[7] In their study, Boivin *et al.* (1995) verified how initial levels and changes in peer-assessed withdrawal, social preference and victimization, and in self-evaluated loneliness could predict changes in self-reported depression. Since the prediction of change in depression from changes in other variables reflects covariation instead of risk, only models using initial levels of dependant variables are reported.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Boney-McCoy and Finkhelor (1996).	HLR (OR)	None.	Parental violence (2.82).	Overall victimisation (2.86); sexual assault (4.21); simple assault (2.00); kidnapping (3.16).	Interaction between victimisation and gender; aggravated assault by non-family member; non-parental family violence; violence to the genitals.
Brent *et al.* (1996).	Cox proportional hazard modelling (OR)	Knowledge of the victim's suicidal intentions (4.4).	None.	Exposure to peer suicide: (a) 6 months following exposure (5.8); 7-18 months (2.5); 19+ months (1.4).	None.
Brent *et al.* (1994).	Log-linear analysis (RR)	None.	None.	Exposition to peer suicide (1.7).	Exposition to peer suicide X D.
J. Brown *et al.* (1999).	HLR (OR)	None.	None.	Adolescent model: any A (2.63); neglect (1.41); S-A (2.80). Adult model: Any A (3.95); neglect (3.45); P-A (3.83); S-A (3.22).	Adolescent model: P-A Adult model: None.
R.A. Brown *et al.* (1996).	HLR (OR)	Cigarette smoking (3 + times a week: 1.89).	None.	None.	None.
Capaldi and Stoolmiller (1999).	HMR (b)	None.	None	None	6th grade EB.
Caspi *et al.* (1996).	ANOVA and HLR (OR)	Inhibited temperament at age 3 (2.2).	None.	None.	Undercontrolled temperament at age 3; gender X temperament styles at age 3.
Chase-Lansdale *et al.* (1995).	HMR (b^2) and HLR (OR)	Continuous: IB at age 7 (6.13); AC achievement at age7 (0.99); IB X gender (2.09: greater for f). Categorical: IB at age 7 (113.8).	Continuous: PD at age 11-16 / no stepparent (1.27); IB X PD (0.43: PD more effects for low IB). Categorical: PD at age 11-16 (1.58); IB X PD (0.06; see previous)	None.	Continuous: PD at age 7-11; PD at age 11-16 / stepparent; SES. Categorical: PD at age 7-11; SES; IB X gender; AC achievement at age 7.
Cheng (1998).	HMR (ß)	None.	None.	m: perceived SS (-0.25); instrumental enacted SS (-0.25). f: perceived SS (-0.27); socioemotional enacted SS (-0.25).	m: socioemotional enacted and network SS. f: instrumental enacted and network SS.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
W.S. Choi *et al.* (1997).	MLR (OR)	f: smoking (regular, 2.05; experimenter, 1.36;); rebellion (1.32). m: smoking (regular, 1.86).	f: high SES (0.69-0.70); family SS (0.76). m: high SES (0.59-0.73).	f: isolation (1.65). m: participation in organized sports (0.74).	f: perceived AC performance; participation in organized sports. m: perceived AC performance; smoking (experimenter); SS and isolation; rebellion.
Cole, Jacquez *et al.* (2001).	HMR (R^2)	Self-appraisal (AC-C, SO-C, BI, behavioral conduct, sports C) (0.29 to 0.37).	None	None	None
Cole, Martin *et al.* (1999).	HMR (ß)	AC-C overestimation relative to teachers (-0.09 to –0.16 in 3rd, 6th, 7th, 8th).	None.	None.	AC-C overestimation X gender; AC-C overestimation (4th, 5th).
Cole *et al.* (1998).	HMR (ß)	Underestimation (relative to teachers and peers) of behavioral conduct (7th; 0.14) and BI (7th, 0.15; 8th, 0.13).	None.	None.	Underestimation of AC-C, sports C, SO-C, behavioural conduct (3rd to 6th, 8th); BI (3rd to 6th); gender X C (5 domains) underestimation.
Cole *et al.* (1996).	SEM (ß)	3rd: None. 6th: SO-C (-0.25).	None.	None.	3rd: AC-C and SO-C. 6th: AC-C.
Daley *et al.* (1998).	SEM (NR)	(1) Episodic stress: predicted from cluster A and B personality disorders. (2) Interpersonal chronic stress: predicted from D.; cluster A and B personality disorders, and initial stress. (3) D: predicted from interpersonal chronic stress and episodic stress			Personality disorders X stress.
Daley *et al.* (2000).	HLR (OR)	Non-mood disorder (3.49).	Witnessing family violence before age 16 (3.83).	Episodic (1.64) and chronic (2.57) stress; D X chronic stress (ß = -1.14–chronic stress affect those without D).	Parental conflict / disruption / death before age 16; non mood disorders (final model); D X non mood disorders; D X parental conflict / disruption / death / violence; D X episodic stress.
Davies and Windle (2001).	HMR (NR for main effects; R^2 for X)	EB; delinquency; poor task orientation; dysrythmicity; low adaptability; high activity.	Marital discord; PCS; marital discord X task orientation (0.011 - effects of marital discord if low task orientation); marital discord X childhood EB X time (0.010 - at T2, marital discord is significant if low EB / both factors = persistent high D.; only EB = declining D; else = low D).	None.	Controlling for PCS, marital discord becomes non significant.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
DuBois *et al.* (1995).	$\chi 2$ and ANOVAS (NR)	D based on a 19 cut-off point: anxiety. D based on a 14 cut-off point: SE; anxiety.	D based on a 19 cut-off point: none. D based on a 14 cut-off point: low SES.	None.	D based on a 19 cut-off point: SES, SE; EB. D based on a 14 cut-off point: EB, moodiness / shyness, and learning difficulties; GPA
Duggal *et al.* (2001).	HMR (final model R^2)	None.	Childhood initial: PCS, parenting support; A; maternal stress. Adolescent initial: model: later maternal stress, early PCS. Childhood final (0.19): A; early maternal stress. Adolescence final (0.19): early PCS.	None.	Childhood: none. Adolescence: later PCS; early parenting support; A, early maternal stress.
Fergusson *et al.* (1996).	HLR (OR)	None	None.	Non-contact sexual S-A (3.6); contact S-A without intercourse (3.0); S-A with intercourse (5.4).	None.
Fergusson and Linskey (1997).	HLR (OR)	None.	Initial: P-A (parents). Final: childhood S-A, parental changes, family LE.	None.	Initial: none. Final: P-A (parents), family history of offending, maternal age, parental illicit drug use, SES, family type.
Fergusson and Woodward (2000).	HLR (OR)	EB (age 13); AC ability (age 12).	Maternal education (birth); parental changes (age 0-13). EB effect completely mediated by attachment to parents (age 15).	None	Maternal age, SES, and family type; P-A and punitiveness; FC; parental offending and drug use; attention problems, IQ and early menarche; deviant peers, cigarette drug or alcohol use; early onset sexual intercourse; peer attachment / deviance school truancy / suspension.
Garber *et al.* (2002).	GCM (NR).	Initial: AS (associated curves). Final: AS.	None.	Initial: LE (associated curves). Final: LE in presence of negative AS.	None
Garrison *et al.* (1990).	MHR (ΔR^2; T1; T2)	None.	Familial adaptability (0.01; ns).	Undesirable LE (0.01).	Desirable LE; familial cohesion; family factors X LE.
Ge, Best *et al.* (1996).	HMR (ΔR^2) and ANOVA (NR)	None.	Continuous: mother (0.03) and father (0.04) warmth; mother hostility (0.02). Categorical: same.	None.	EB; SES; parental divorce; father hostility; mother and father discipline.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Ge *et al.* (1994).	GCM (indicators)	None.	f: maternal warmth / support (0.76); maternal warmth / support X age, D, and LE (0.39 to 0.59; girls with low maternal warmth / support are more vulnerable to maturation, D, and LE). m: none.	f: LE (associated intercepts, 0.81; slopes, 0.36; LE intercept with D slope, 0.37). m: LE (associated intercepts, 0.79).	f: None. m: LE (association of slopes and of LE intercept with D slope); maternal warmth and support; maternal warmth and support X age, D, or LE.
Hammen *et al.* (2000).	HLR (OR)	None.	Childhood adversity (1.18).	Objective LE (1.18); childhood adversity X objective LE (0.86: LE always associated with higher D; childhood adversity predicts D when LE are low).	Chronic stress.
Hankin *et al.* (2001).	Setwise HMR (analysis of partial variance; pr)	Total BDI: AS (0.22); AS X LE (0.16 – see m bellow); AS X LE X gender (NR - for m both AS and LE are needed to increase of D; for f main effects of LE and AS). HDSQ-R: AS (0.34). Hopelessness BDI: AS (0.20); AS X LE (0.18 – see previous); AS X LE X gender (see previous). Other BDI: AS (0.20); AS X LE X gender (see previous).	None.	Total BDI: LE (0.14). HDSQ-R: LE (0.20). Hopelessness BDI: LE (0.13). Other BDI: LE (0.23).	Total BDI: none. HDSQ-R: AS X LE X gender. Hopelessness BDI: None Other BDI: AS X LE.
Hilsman and Garber (1995).	HMR (ΔR^2)	T2 D: perceived AC-C (0.011). T3 D: Grade stressors X perceived AC-C and AS (grade stressor impact D in presence of negative cognitive style – 0.007).	T2 Dep.: parent stressor (0.015). T3 D: none.	T2 D: grade stressor (0.012). T3 D: none.	T2 D: AS, AS X LE. T3 D: parent & grade LE, perceived AC-C, AS.
K.B. Hoffman *et al.* (2000).	HMR (ß)	Reflected self-appraisal (-0.16 to -0.17); Discrepant self-appraisal (-0.14 to -0.20).	None.	None.	None.
Hogue and Steinberg (1995).	HMR (ß)	(1) Selection: T1 adolescents' D predict T2 peers' D (0.21). (2) Socialization: T1 peers' D predict T2 adolescents' D controlling for T1 D among boys (0.09) (friendship groups may heighten – effect size = 0.41 – or diminish – effect size = 0.22 – boys D). (3) Contagion: T1 adolescents' D predict T2 peers' D controlling for T1 D (0.09).			None.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Holsen *et al.* (2001).	SEM (ß)	*Age 15 D.* f: age 13 BI (-0.34) / m: none. *Age 18 D.* f: none / boys = age 15 BI (-0.19).	None.	None.	*Age 15 D.* f: none / m: age 13 BI. *Age 18 D.* f: age 15 BI / boys = none.
Hops *et al.* (1990).	Repeated measures ANOVAs (NR)	EB; subjective probability (sense of control); SE (more for f); personal beliefs (irrational beliefs); tension; self image; positive AS; reflective and public self-consciousness.	None.	Micro-stressors.	Anxiety; positive affect (extraversion); coping; masculinity; self-reinforcement; suicidal ideation; negative AS; LE; BI; SS; family environment (many dimensions; interpersonal sensitivity.
Jaffe *et al.* (2002).	Regression framework (NR). Comparison of juvenile VS adult onset D.	Juvenile onset only: poor motor skills (3-9); inhibited temperament (3); worried-fearful (5-9); hyperactivity (5-9); antisociality (5-9). Adult and juvenile onset: none.	Juvenile onset: parental criminality; parent figure changes (birth to 9); parental losses (birth to 11). Adult and juvenile onset: disagreement about discipline (5-9).	Juvenile onset: peer problems (7-9). Adult and juvenile onset: moving (birth to 9); S-A (birth to 11).	Overall: Intelligence (7-9): mother rejection (3); SES (birth to 9); undercontrolled temperament (3).
Jekielek (1998).	HMR (ß)	None.	PC T1 (0.03); marital disruption T1-T4 (0.11); PC X disruption (-0.08 – if high PC at T1, divorce is related to lower D).	None.	None.
Joyner and Udry (2000).	HMR (b)	Desire for RI (0.279).	Parents with low education (0.296); one parent graduated from college (-0.244); living with both parents (-0.349).	1st RI between T1-T2 (0.518); continuous RI (0.438); 1st (0.512) or continuous (0.425) RI between T1-T2 X gender (more effect for f); continuous RI X age X gender (-0.328; more effect for younger f)[8].	Parent with post high school education; RI only before T1; RI X age or gender; 3-way X with before T1 RI.

[8] After the results presented in the table, Joyner & Udry (2000) evaluated potential mediators of the RI-D relationship. These additional analyses were based on cross-sectional measures of outcomes and mediators and are not reported. However, the results suggest that additional variables may also predict T2 depression: quality of mothers-adolescent relationships at T1 (-0.67), difficulties with homework (-0.097), involvement in two RI (0.271) or more (0.308), and experiencing a romantic breakup in the previous month (0.546). Moreover, the deleterious effect of RI may to be completely mediated by resulting changes in family and academic functioning, as well as by the number and stability of RI.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Kasen *et al.* (1996).	HLR (OR)	EB (temperament, 2.12).	Single custodian mother for m (5.19); stepfamily for f (marginal, 4.26).	None.	Immaturity; IB; temperament X gender; temperament X family structure; 3-way X.
Katainen, Räikkönen and Keltikangas-Järvinen (1999).	SEM (ß)	f: none. m: sociability (-0.26).	None.	f: significant other's SS (other than friend or family; -0.17). m: None.	None[9].
Katainen, Räikkönen, Keskivaara *et al.* (1999).	SEM (ß)	None.	Maternal role satisfaction for f (0.26); mother's hostility (0.21-0.23; predicted from child temperament– 0.57-0.64 – and from maternal role satisfaction – 0.28-0.32).	None.	None.
Khatri *et al.* (2000).	HMR (ß)	None.	None.	None.	Peer-reported aggression and victimization alone and in interaction with gender.
Kiesner (2002).	HMR (ß)	EB (0.13).	None.	Peer status / Social preference score (-0.11).	None.
Krueger (1999).	HLR (OR) HMR (ß)	HLR: (1) Traits: well-being (0.80); stress reaction (1.52); aggression (0.68); (2) Factors: N (1.27). HMR: (1) Traits: well-being (-0.08); stress reaction (0.11); alienation (0.09); harm avoidance (-0.09);(2) Factors: N (0.12)	None.	None.	HLR: (1) Traits: social potency; control; achievement; social closeness; alienation: harm avoidance; traditionalism; (2) Factors: agency; communion; constraint. HMR: (1) Traits: social potency; aggression; achievement; social closeness; control; traditionalism; (2) Factors: agency; communion; constraint.

[9] Katainen, Räikkönen and Keltikangas-Järvinen (1999) did evaluate a more complete model including temperament, social support and prior depression, as well mediating relationships between theses variables. However, since the predictors and mediators were measured at the same moment, these results are not presented in the table.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Lewinsohn, Allen *et al.* (1999).	HLR (LR)	None.	None.	LE (5.98); previous D X LE (9.37; LE related to 1st onset with a threshold of 3+ LE); previous D X previous BDI X AS (10.40; high scores on AS and BDI related only to recurrences).	AS; gender X other factors; AS X previous D, previous BDI or LE; LE X previous BDI or AS; previous D X previous BDI.
Lewinsohn *et al.* (1995).	ANOVA (NR)	Emotional reliance; physical health; lifetime number of physical symptoms; suicide attempt; anxiety; negative cognitions; rate of tobacco use for f; EB; coping skills.	PCS; PCC.	Daily hassles; LE; grades' dissatisfaction.	See Lewinsohn *et al.* (1994) for a more complete description of the non significant results.
Lewinsohn *et al.* (2001).	HLR (OR)	None.	None.	Dysfunctional attitudes X LE (marginal – higher % of D when both are high); AS X LE (0.91 – high LE: AS no effect; low LE: effect of AS).	Other disorders; dysfunctional attitudes; AS; LE; dysfunctional attitudes X AS; X with D and 3-way X.
Lewinsohn *et al.* (1994).	ULR and MLR (OR)	ULR: IB (2.8); EB (1.6); pessimism (1.6); AS (1.4); self-consciousness (1.1); emotional reliance (1.1); SE (1.5); coping (1.1); physical health (1.5); lifetime number of physical symptoms (1.1); anxiety (2.4); suicide attempt (6.1). MLR: "other" disorders (4.6); suicide attempt (3.8); IB (1.8); lifetime number of physical symptoms (1.03).	ULR: PCS (1.6); PCC (1.4). MLR: none.	ULR: daily hassles (1.0); LE (1.1); non-completion homework (1.3); dissatisfaction with GPA (1.7). Multivariate: None	ULR: family structure; SO-C; goals; maturation level; attractiveness; friends' SS; death of parents; desirability; low vocabulary; hippomanic personality; SA; "other" disorder; school failures, absences, lateness; parents GPA dissatisfaction; poor self rated physical health; obesity; functional difficulties; exercise; medication; hospital; smoking (rate & ever). Multivariate: not reported for the sake of brevity.
Lewinsohn *et al.* (1996).	Multiway frequency table (%)	Disease (9.8% / 5.9%); functional impairment (19.4% / 7.3%).	None.	None.	Injury; reduced activity.
Lynch and Cicchetti (1998).	HMR (ß)	EB (0.08); SE (-0.05).	None.	None.	# children at home; IB; maternal education; maltreatment; traumatic LE; community violence witness; victimization;.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
McFarlane *et al.* (1995).	HMR (ß)	SO efficacy (-0.129).	Family SS (-0.260).	Stress (0.130). Path analysis model revealed peer SS effect was totally mediated by it effects on stress (0.13) and SO efficacy (0.29).	Family composition; parental occupation; (peer SS).
McGrath and Repetti (2002)	HMR (summary)	4^{th} to 5^{th}: AC/SE. 5^{th} to 6^{th}: none. 4^{th} to 6^{th}: SO/SE; SO distortion.	None.	None.	4^{th} to 5^{th}: EB; global SE; SO/SE; AC and SO distortion. 5^{th} to 6^{th}: EB; global SE; AC/SE; SO/SE; AC and SO distortion. 4^{th} to 6^{th}: EB; global SE; AC/SE; AC distortion .
McLeod and Shanahan (1996).	Latent GCM. Only results pertaining to prospective associations are reported.	None.	Initial D: none. D increases: mother's age at birth (0.04).	Initial D: poverty before T1 (0.28). Increases D: none.	Initial D: mother age at birth; marital status; maternal education. D increases: maternal education; marital status; poverty T1-T3.
Miech *et al.* (1999).	HLR and HMR (NR).	None.	None.	None.	Age 15 SES.
Monroe *et al.* (1999).	HLR (OR)	None.	None.	RI break-up (1.92; effect holds when controlling for LE, hassles, gender, and prior D); prior D X RI break-up (predicts 1^{st} onset, OR = 2.72).	Gender X RI break-up; gender X prior D.
Nolen-Hoeksema *et al.* (1992).	HMR (summary)	(1) AS related to future D at older ages only. (2) LE related to later D. at younger ages only. (3) AS X LE is proximally related to D. at older age only and significant only in 4 out of 18 regressions (LE more negative effects on pessimistic children). (4) Experiencing D is often related to deterioration of AS.			Achievement helplessness; social helplessness.
Palosaari *et al.* (1996).	Hierarchical log-linear model (NR)	f: SE (predicted from childhood PD). m: SE.	f: PD (indirect); closeness with father (mediate the effect of PD). m: PD (direct); closeness with mother; closeness with father	None.	f: closeness with mother. m: none.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
			(also predicted from PD)		
Panak and Garber (1992).	HMR (ΔR^2)	Model 1: (a) T2 D[10]: T1 aggression (0.01); (b) T3 D: none. Model 2: (a) T2 D: negative AS (0.10); (b) T3 D: negative AS (0.02).	None.	Model 1: (a) T2 D: perceived rejection (0.04; predicted from PR rejection); (b) T3 D: T1 (0.06) and T1-T2 (0.07) changes in perceived rejection. Model 2: (a) T2 D: none; (b) T3 D: AS X T1-T2 changes in PR rejection (0.01; higher effects of rejection for negative AS subjects).	Model 1: (a) T2 D: PR rejection; (b) T3 D: T1 and T1-T2 changes in aggression and PR rejection. Model 2: (a) T2 D: PR rejection; AS X PR or perceived rejection; (b) T3 D: T1 and T1-T2 changes in PR rejection; AS X perceived rejection.
Pine *et al.* (2001).	MLR (OR)	T1 to T2: fear (3.1). T1 to T3: fear of dark (2.1); overanxiety (2.5). T2 to T3: none.	None.	None.	T1 to T2: fear of dark; overanxiety (2.5) / T1 to T3: fear; parent-rated fear of dark / T2 to T3: fear; fear of dark; overanxiety (2.5).
Pine *et al.* (1998).	MLR (OR)	Overanxiety (2.29); EB (2.20).	None.	None.	SES; simple phobia; social phobia; separation anxiety; fearful spells; ADHD.
Pomerantz (2001)[11].	HMR (ß)	AS (-0.12); C estimation (-0.10).	AS (-0.10) and C estimation (-0.09) X intrusive support (negative AS or C underestimation = D increases if intrusive support).	None.	Parental intrusive support; parental intrusive support X D, grade, gender, or SES.
Rao *et al.* (1999).	Survival analysis (ßе)	Non-affective disorders in adolescence (2.2).	None.	None.	None.
Reinherz *et al.* (2000).	HLR (OR) & stepwise MLR (NR).	HLR: teacher reported IB at 6 (2.08); parent reported IB at 9 (2.87) and IB (2.53) at 9. Stepwise: perceived unpopularity at 9; parent reported IB at 9.	HLR: sibling drug-alcohol problems (3.14). Stepwise: none.	HLR: perceived unpopularity at 9 (3.71). Stepwise: perceived unpopularity at 9.	HLR: gender X; parental age; family size; SES; PD; parental alcohol-drug problems; parent, teacher and self-reported EB; parent-reported IB at 5, teacher reported IB at 9. Stepwise: not reported for brevity.

[10] To predict T2 and T3 D, Panak and Garber (1992) also verified the effects of changes (T1-T2) in the variables. Because theses effect on T2 depression reflect covariation instead of prediction, they are not reported.

[11] Pomerantz (2001) also evaluated the effects of parental intrusive support on the basis on maternal reports (n = 74). However, due to small sample size (low statistical power), theses results are not presented in the table.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Reinherz, Giaconia, Pakiz *et al.*, (1993).	$\chi 2$ and t-tests (OR)	m: health problems: neonatal (10.3) and at 0-6 (7.3); self and mother rated anxiety at 15 (4.2; 5.1); dependence (5.1). f: low SE (3.1); self (ages 9, 15) and mother (age 15) rated anxiety (1.2; 7.5; 4.5); health problems at age 10-15 (5.5); pregnancy (13.6).	m: poor perception of family role at 9 (5.3); PC at 10-15 (9.3); parental remarriage (3.7). f: 3rd+ birth order (3.7); older parents at birth (2.8-3.3); 3+ siblings (2.7); poor perception of family role at 9 (2.4); death of parent (7.0).	m: unpopularity at 9 (3.0). f: unpopularity at 9 (2.8).	m: SES; parental age; birth order and family size; PD; health problems at 6-15; GPA; death of parent; shyness at 5; anxiety at 5-9; SE. f: SES; PD; parental remarriage; PC; health problems before 9; GPA; dependence and shyness at 5; mother rated anxiety at 5-9.
Robinson *et al.* (1995).	HMR (ΔR2)	Transition (T2) D: D & EB (0.455); AS and SE (0.032); AS X LE (0.019); AS X SE X LE (0.008)[12]. T3 D: D and EB (0.486).	None.	Transition (T2) D: LE and hassles (0.028). T3 D: none.	Transition (T2) D: SE X LE; SE X AS. T3 D: LE, AS, SE and interactions.
Roeser and Eccles (1998).	HMR (ß)	GPA (-0.18).	None.	School ability (0.15) and task (-0.07) goal structure; positive teacher regard (-0.26).	SES; school facilitation of student autonomy.
Rohde *et al.* (2001).	MLR (OR)	Problematic drinking (1.5); diagnosis of alcohol abuse / dependence (2.3).	None.	None.	None.
Rosenfield *et al.* (2000).	HMR (ß)	Empathy with friends discomfort (-0.47; explain m-f differences).	None.	None.	School grade; parental education; empathy with friends' happiness and sadness.
Rudolph *et al.* (2001)	Multiple regressions (ß)	Perception of control (cohort 1: -0.19; cohort 2: -0.11).	None	None	Helplessness (cohort 2 only).
Schwartz and Koenig (1996)	HMR (ß)	Ruminative coping (0.16, significant only for f: 0.21).	None.	Negative LE (0.13).	AS; AS X LE; distraction; rumination X distraction; self-consciousness; coping X LE.
Sears and Armstrong (1998).	HMR (ß)	Risk behaviors (0.19).	None.	None.	Anxiety; anxiety X gender or D; risk behaviour X gender, anxiety, or D.
Segrin and Flora (2000).	MHR (ΔR2)	SO skills (0.01); SO skills X LE (0.03; higher levels of SO skills protect against LE).	None.	LE (0.15).	None.

[12] The AS X LE X SE interaction decomposition indicates that: (a) at high levels of self-esteem, attributional style has no effects and stress is related to an increase in depression; (b) at low levels of self-esteem, positive attributional style protects against the effects of stress whereas negative attributional style allows stress to exert a negative effect; (c) in the absence of stress, low self-esteem is related to an increase in depressive symptoms.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Seiffge-Krenke and Klessinger (2000).	HMR (ß)	T3 D: T1 avoidant coping (0.19). T4 D: T1 avoidant coping (0.16).	None.	None.	T3 D: T2 avoidant coping. T4 D: T2-T3 avoidant coping
Siegel (2002).	HMR (ß)	BI (-0.17 m; -0.13 f); SE (-0.20 m; -0.18 f)[13].	Household income (-0.14 f).	None.	Household income for m; body mass index.
Sim (2002).	SEM (ß)	None.	m: T1 and T2 family SS (-0.24). f: T1 family SS (-0.26).	m: none. f: T1 teacher SS (-0.26).	m: T1-T2 friend and teacher SS. f: T1-T2 friend SS, T2 family and teacher SS.
Slavin and Rainer (1990).	HMR (ß)	None.	None.	f: adult SS (0.15); peer SS (-0.11). m: none.	f: PCS m: PCS, adult SS, peer SS.
Spence *et al.* (2002).	HMR (ß)	AS (-0.12); negative problem solving orientation (0.14).	None.	LE (0.27); problem solving X LE (0.07; D high when both high).	LE X AS; gender X; D X.
Stein *et al.* (1996).	Path analysis (ß).	T2 D from T1: cheerfulness (-0.14); extroversion (-0.20). T3 D from T2: cigarette use (0.14).	None.	T2 D from T1: none. T3 D from T2: SO relations (-0.18).	T2 D from T1: cigarette use; SO relations; friends' cigarette use. T3 D from T2: friends' cigarette use; extroversion.
Stice and Bearman (2001).	GCM (% explained σ)	BI (initial, 6.7%; slope, 4.5%); dieting (initial, 5%; slope, 9.9); bulimic symptoms (initial, 6.9%; slope, 7.5%).	None.	SS (initial, controlled variables).	Body mass index.
Stice *et al.* (2000).	Cox proportional hazard (Hazard ratios)	Bivariate: BI (1.31); dieting (1.51); bulimic symptoms (1.79). Multivariate: dieting (1.40); bulimic symptoms (1.40).	None.	None.	Bivariate: body mass index. Multivariate: body mass index; BI.
Stice *et al.* (2001).	HMR (ß)	Body mass index (0.29); BI (0.41); dieting (0.28).	None.	None.	None.
Sund and Wichstrøm (2002).	HLR (OR)	None.	Insecure attachment to parents (1.36; especially alienation, 1.51).	LE (1.12).	SES; family type; # of friends, # of moves; attachment to peers; attachment X LE, attachment X D.

[13] Siegel (2002) also evaluated the impact of changes in BI and of ethnic group X changes in BI on T1-T2 changes in depression. As these results represent covariation instead of prediction, they are not reported in the table.

Authors	Analysis (indicator)	Individual factors	Family factors	Other social factors	Non-significant results
Tram and Cole (2000).	HMR (ß)	Moderator model: self-rated C (-0.20). Mediator model: self-rated C (-0.22; significant partial mediator of LE).	None.	Moderator model: negative LE (0.14). Mediator model: negative LE (0.12).	Global: positive LE. Moderator model: self-rated C X negative LE (no moderation). Mediator model: none.
Unger *et al.* (2000).	SEM (ß; summary)	Specific to model 1: T1 FC regarding parent issues is related to T2 D (0.18) and to T1 difficulties in family functioning (0.28), which predict difficulties in family functioning at T2 (0.64). Specific to model 2: T1 FC regarding adolescent issues is related to T1 difficulties in family functioning (0.45), which predict of T2 difficulties in family functioning (0.64). Generic: T2 difficulties in family functioning are related to T2 D (0.39 to 0.42) and predict T3 D (-0.29).			
Windle (1992 b).	HMR (ß)	None.	f: family SS (-0.17). m: none.	f: LE (0.17). m: friend SS X LE (0.22: friend SS buffer against low-moderate LE, and augment the effect of high LE).	f: friend SS, friend or family SS X LE. m: friend and family SS, LE, family SS X LE.
Windle (1994).	HMR (ß)	None.	None.	None.	Overt / covert hostility with friends, reciprocity and self-disclosure with friends.
Windle and Windle (2001).	HMR (ß)	Marijuana use (-0.10); flexibility (-0.16); heavy cigarette use (1/2 pack or more on at least 3 of the 4 occasions; 0.08).	None.	Alcohol using peers (0.07).	Parental smoking; PCS; peers' SA; delinquency; alcohol use; other illicit drug use; general activity ; positive mood; task orientation; moderate cigarette use.
Zimmerman *et al.* (2000).	Multiple regression (ß)	None.	PCS (-0.19).	None.	Friend SS; LE; PCS X LE; friend SS X LE; parental education; household structure; desirability.

Abbreviations. *A*: abuse (*S-A*: sexual abuse; *P-A*: physical abuse); *AC*: academic; *AS*: attributional style; *BI*: body image/physical appearance; *C*: competence; *D*: depression; *EB*: externalizing behavioral problems / conduct-oppositional disorders; *f*: females; *FC*: family conflict. *GCM*: growth curve modeling; *GPA*: grade point average or academic performance; *HLR*: hierarchical logistic regression; *HMR*: hierarchical multiple regression; *IB*: internalizing behavior problems; *LE*: life events; *m*: males; *MLR*: multivariate logistic regression; *N*: neuroticism; *NR*: not reported in paper; *OR*: odds ratio; *PC*: parental conflict (between parents); *PCC*: parent-child conflict; *PCS*: parent-child support; *PCR*: parent-child quality of relation; *PD*: parental divorce; *PR (…)*: peer rated; *PS*: problem solving; *RI*: Romantic involvement; *RR*: risk ratio; *SA*: substance use or abuse; *SE*: self esteem; *SEM*: structural equation modeling; *SES*: socio-economical status; *SO*: social; *SS*: social support; *ULR*: univariate logistic repression; *(…) X (…)*: interaction.

Chapitre III

School life and depression development following high school transition : risk analyses from the Montreal Adolescent Depression Development Project (MADDP)[14]

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[14] Soumission probable de l'article: *Development and Psychopathology, Developmental Psychology* ou *Genetic, Social, and General Psychology Monographs*

ABSTRACT

The main objective of the present study was to evaluate the relationship between school life and depression development in adolescents. More precisely, this study sought to determine which specific aspects of school life (in-school psychological characteristics, school-related socialization experiences, perceived school environment) could be considered as risk factors for depression development once students' background characteristics are taken into account. The possibility that these relationships could be moderated by gender and by students' previous levels of depression was also evaluated. These exploratory questions were evaluated with data from the transitional component of the Montreal Adolescent Depression Development Project (MADDP), a one-year (three-measurement-point) follow-up study of 1167 seventh grade students (52.7% males; 47.3% females, mean age 12.75 years) having just experienced high school transition. The results clearly suggested that various aspects of students' school life represent significant predictors of depression development, particularly among girls. Moreover, these results also reinforced Kessler's (1997) suggestions regarding the importance of differentiating risk factors according to their impact on the emergence or aggravation of depressive symptoms. One of the main conclusions from this study is that school-based prevention programs, especially those targeting school violence, would be likely to diminish students' risk of developing depression following high school transition.

Key words: depression, adolescence, risk factors, school life, transition.

Depression, through its lifelong chronic, recurrent, comorbid, and disabling nature, clearly represents a preeminent challenge for mental health researchers and preventionists (Angold, Costello, & Erkanli, 1999; Harrington & Dubicka, 2001; Kessler, 2002; Kessler, Avenevoli, & Merikangas, 2001; Lewinsohn & Essau, 2002; Murray & Lopez, 1996 a, 1996b). Developing efficient depression prevention programs was therefore identified as a key priority for developmental research by national and international health organizations (Dawson & Tylee, 2001; Mrazek & Haggerty, 1994). Developing such programs requires a precise and integrated understanding of the many risk and protective factors implicated in depression development (Coie *et al.*, 1993; Kazdin, 1993; Mrazek & Haggerty, 1994). Moreover, as depression usually develops during adolescence and shows great continuity across the lifespan, the impact of these factors would have to be studied in child and adolescent populations (Kessler *et al.*, 2001; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Newman, Moffitt, Caspi, Magdol, Silva, & Stanton, 1996).

From ages 6 to 16, children and adolescents spend a significant part of their waking life at school. It is thus not surprising that studying the relationships between school life and mental health has oftentimes been identified as a key priority for developmental research (Boyce *et al.*, 1998; Rutter, 1999; Rutter *et al.*, 1997; Zaslow & Takanishi, 1993). Schools represent some of youths' central life settings as well as a key socialization area. School life also encompasses many non-academic aspects of children's and adolescents' social existence, such as the beginning of friendships, romance, and autonomy from parents. Consequently, school life may play a vital part in the fulfillment of youths' basic developmental needs for affiliation, security, autonomy, bonding, and achievement (Bronfenbrenner, 1977; Eccles, Lord, & Midgley, 1991; Moos, 1979; Mortimore, 1995; Roeser, Eccles, & Strobel, 1998).

Depression prevalence rates were found to increase in recent adolescent birth cohorts (Fombonne, 1998; Lewinsohn, Rohde, Seeley, & Fisher, 1993), and current hypotheses indirectly suggest that school life may be implicated in this phenomenon. For instance, Eccles and colleagues (Eccles *et al.*, 1991, 1993) argued that whereas adolescents' basic developmental needs imply autonomy, intimacy, identity formation, and abstract thinking, modern middle schools (versus elementary ones) are often characterized by increased discipline and control, academic and social competitiveness, social network disruptions, and lower cognitive demands. The resulting mismatch may then create an increased risk for the development of psychosocial problems, especially in students who already present a vulnerability to such problems (Eccles *et al.*, 1991, 1993; Janosz, Georges, & Parent, 1998). More precisely, adolescents whose school life is characterized by a mismatch between developmental needs and socialization experiences may come to internalize the idea that their needs are unworthy of attention and develop chronic feelings of helplessness, which in turn may lead to depression (Haaga, Dyck, &Ernst, 1991). The fact that depression often develops in early to mid-adolescence, following high school transition (Cyranowski, Frank, Young, & Shear, 2000; Nolen-Hoeksema, 2002), provides further support to Eccles *et al.*'s (1991, 1993) hypothesis.

Additionally, Diekstra (1995) and Robins (1995) indicated that the lives of modern adolescents are now characterized by an earlier onset of biological maturation (puberty), by lengthier academic training and by the breakdown of traditional sources of social support (e.g., intact and extended families, community cohesion, churches, etc.). In this context, modern adolescents have to deal earlier with adult bodies and physiological functions without being able to assume adult roles. Diekstra (1995) and Robins (1995) hypothesized that these new challenges represent one potential explanation for the increased rates of depression observed among modern adolescents. In addition to these challenges, schools may themselves contribute to adolescents' exposure to various forms of stressful experiences (e.g., being bullied at school, having a conflictual relationship with one's teachers, etc.): the relationship between stress exposure and depression is a well-documented phenomenon (Ge,

Lorenz, Conger, Elder, & Simons, 1994; Lewinsohn, Allen, Seeley, & Gotlib, 1999). Moreover, due to the breakdown of traditional institutions, modern adolescents often must face alone the identity crisis that may result from these new challenges and stressful experiences (Diekstra; 1995; Robins, 1995). Schools occupy a privileged position to provide modern youths with alternate sources of social support to help them build up an integrated sense of identity. Once again, social support represents a known protective factor against depression development (Cheng, 1998; Kiesner, 2002; Stein, Newcomb, & Bentler, 1996). Unfortunately, as Eccles *et al.* (1991, 1993) indicated, modern schools may not be equipped to deal with these new challenges.

Surprisingly, although many risk and protective factors were studied in relation to depression development in children and adolescents (for a review, see Morin, Janosz, & Larivée, in preparation), very few studies attempted to understand the precise role of various dimensions of school life in depression development. This is the objective of the present exploratory study.

SCHOOL LIFE AND DEPRESSION DEVELOPMENT

Schools are highly complex social systems and possess a reality of their own. In schools, multiple sources of influence converge to influence students' development. Accordingly, students' school life quality will be determined by a combination of various factors related to their psychological characteristics and socialization experiences and to the specific characteristics of their schools (Janosz, Georges *et al.*, 1998). Reaching a complete understanding of the effects of school life on depression requires the simultaneous consideration of these multiple sources of influence.

Psychological Characteristics

Because individuals may choose and modify environments, it is unlikely that students with different psychological characteristics will be exposed to similar experiences at

school (Mortimore, 1995; Rutter, 1999). Scholars should therefore attempt to evaluate whether the effects of school life on depression are real or an artifact of students' psychological background characteristics (neuroticism, anxiety, self-esteem, behavioral disorders, body image), themselves related to depression development (Jaffe *et al.*, 2002; Krueger, 1999; Lewinsohn *et al.*, 1994; Siegel, 2002; Stice & Bearman, 2001). However, specific psychological characteristics (school adaptation, school motivation, academic achievement, etc.) represent more direct determinants of students' school life quality. The effect of these elements on depression development should thus be more directly evaluated. Preliminary evidence indicates that at least some of these in-school psychological characteristics may be involved in depression.

Many studies showed significant negative relations between depression development and various dimensions of students' motivation at school, such as academic self-efficacy, perceived academic competencies, and involvement in school extracurricular activities (Bandura, Pastorelli, Barbaranelli, & Caprara, 1999; Gore, Farell, & Gordon, 2001; Hilsman & Garber, 1995; Lewinsohn *et al.*, 1994; Mahoney, Schweder, & Stattin, 2002). Nevertheless, studies usually failed to demonstrate a relationship between academic achievement and depression development (e.g., Bandura *et al.*, 1999; Cole, Martin, Powers, & Truglio, 1996; Lewinsohn *et al.*, 1994; Reinherz *et al.*, 1993). This last result should, however, be considered cautiously, as Chase-Lansdale, Cherlin, and Kiernan (1995) reported an association between academic achievement at age 7 and depression development in young adulthood. Fergusson and Woodward (2000) also noted a long-term relation between academic achievement at age 12 and depression at age 18.

For their part, Lewinsohn *et al.* (1994) concluded that students who were dissatisfied with their academic achievement and/or who did not regularly complete their homework presented an increased risk of developing depression. Yet, they obtained no significant relationship between depression and school failures, truancy, and lateness, suggesting that only some facets of school-based motivation and misbehaviors are related to depression. Further studies also reported a relation

between depression and bullying, another specific form of school misbehavior (Austin & Joseph, 1996; Kaltiala-Heino, Rimpelä, Marttunen, Rimpelä, & Rantanen, 1999; Kaltiala-Heino, Rimpelä, Rantanen, & Rimpelä, 2000; Nansel *et al.*, 2001).

Socialization Experiences

Previous studies clearly showed that children's and adolescents' socialization experiences within families and peer groups influenced their risk of developing depression (Cheng, 1998; Ge, Best, Conger, & Simons, 1996; Ge *et al.*, 1994; Jaffe *et al.*, 2002; Jekielek, 1998; Kiesner, 2002; Lewinsohn *et al.*, 1994, 1999; Stein *et al.*, 1996; Tiet *et al.*, 2001). Additionally, these experiences may also indirectly affect the quality of school life. For instance, parents may choose to send their children to schools which conform most to their own values and practices. Moreover, youths also tend to reproduce at school (and other settings) the various skills and interactional patterns that they previously learned in contact with peers and parents (Cicchetti & Rogosch, 2002; Cicchetti & Schneider-Rosen, 1986; Cicchetti & Toth, 1998). It therefore appears important to evaluate whether the effects of school life on depression development represent an artifact of students' background socialization experiences or whether these effects are real and specific to school-based socialization experiences. Three kinds of socialization experiences may be more directly involved in the quality of students' school life: parental school-related educative practices, school-based interactions with peers, and interactions with school adults (teachers and other members of the school personnel). Preliminary evidence suggests that such experiences may influence youths' risks of developing depression.

Regarding the role of parental school-related educative practices, Hilsman and Garber (1995) indicated that parental dissatisfaction with children's grades was related to a small increase in children's depressive symptoms in the following days, but not a week later. Conversely, Lewinsohn *et al.* (1994) found no support for a relation between parental dissatisfaction with adolescents' school grades and depression development in a longer term follow-up study.

Second, preliminary evidence indicates that specific aspects of in-school peer relationships may also be associated with depression development. For instance, Gazelle and Ladd (2003) suggested that facing peer exclusion at kindergarten entry could be particularly predictive of depression development for anxious solitary children. Other studies noted similar relationships between depression development and peer rejection or conflict at school (Brendgen, Vitaro, Bukowski, Doyle, & Markiewicz, 2001; Jaffe *et al.*, 2002; Kiesner, 2002), victimization at school (Austin & Joseph, 1996; Hodges & Perry, 1999; Kaltiala-Heino *et al.*, 1999, 2000; Nansel *et al.*, 2001), and affiliation with peers presenting high levels of school adaptation problems (Cantin, Wanner, Brendgen, & Vitaro, 2002).

Finally, some studies revealed a relationship between various aspects of students' socialization experiences with school adults and depression. Generally, these studies revealled that higher levels of teacher support and positive teacher regard were related to lower risk of subsequently developing depression (Kaltiala-Heino *et al.*, 1999; Roeser & Eccles, 1998; Roeser, Eccles, & Sameroff, 1998), although this effect may be more important for girls (Sim, 2002). Similarly, some scholars were able to identify a significant relationship between school and teacher-related stress and depression development (Siddique & D'Arcy, 1984; Turner & Cole, 1994).

School Environment

As mesosystems, schools represent more than the sum of the previously described microsystems[15]. In themselves, schools are social systems with their own rules and characteristics which are relatively independent from the socialization experiences and individual characteristics of the specific students attending them (Janosz, Georges *et al.*, 1998). For example, whereas being repeatedly victimized may characterize the

[15] According to Bronfenbrenner (1977), microsystems represent youths' immediate interactions with socialization agents (peers, family members). Mesosystems refer to the "interrelations among major settings containing the developing person [or microsystems] at a particular point in his or her life" (Bronfenbrenner, 1977, p. 515).

school experience of a specific student, very few other students may be victimized in the school. Consequently, studying the effects of school life on depression development implies that specific school characteristics should also be considered. Three methods have generally been used to evaluate school environment characteristics. First, some scholars relied on students' perceptions of the characteristics of their school environment. This approach is generally referred to as the evaluation of school *psychological environment* (Kuperminc, Leadbeater, & Blatt, 2001; Roeser & Eccles, 1998; Roeser, Eccles, & Sameroff, 1998). Second, students' perceptions could also be aggregated at the school level to obtain a less subjective estimate of school characteristics. Third, structural school characteristics could be more directly evaluated through observation, school records, and demographic information (i.e., architectural design, size, curricular diversity, demographic characteristics, deterioration of buildings, etc.). Both the second and third approaches represent attempts to evaluate more objectively school characteristics as potential sources of influence on students' development (Anderman, 2002; Moos, 1979). Unfortunately, we are aware of no studies in which the relation between aggregated or structural school characteristics and depression was directly evaluated.

Some studies reported a significant relationship between the overall quality of school climate, or the generic atmosphere of the school environment, and lower levels of depression among students (Garnefski, 2000; Kuperminc *et al.*, 2001; Way & Chen, 2000). Other studies, however, failed to replicate these finding (Hadley-Ives, Stiffman, Elze, Johnson, & Dore, 2000) or found this effect to be limited to girls (Kuperminc, Leadbeater, Emmons, & Blatt, 1997). Measurement differences could explain this discrepancy, as the school climate scales used in these studies were generally idiosyncratic. Similarly, other studies used scales combining measures of students' academic motivation, in-school socialization experiences, and school climate perceptions and proposed that lower scores on these scales were predictive of lower levels of depression (Anderman, 2002; Aseltine & Gore, 1993; Eccles, Early, Frasier, Belansky, & McCarthy, 1997; Resnick *et al.*, 1997). In a more detailed cross-sectional analysis of the relationships between school climate perceptions and

depression, Morin and Janosz (2002) noted a negative relationship between students' levels of depression and several dimensions of school climate quality (e.g., students' impressions of evolving in a school where relations are positive, where safety is emphasized, where learning is valorized, and where justice is the norm).

Among more specific aspects of school psychological environment, some studies discovered that school-based discrimination and injustice were related to higher levels of depression among students (Resnick *et al.*, 1997; Roeser, Eccles, & Sameroff, 1998), whereas school practices designed to facilitate proactive social relationships (Kasen, Johnson, & Cohens 1990) and efficient disciplinary practices (Eccles *et al.*, 1997) were related to lower levels of depression. However, scholars generally failed to find significant associations between students' levels of depression and their perceptions of their schools' curricular meaningfulness and valorization of learning (Kasen *et al.*, 1990; Roeser, Eccles, & Sameroff, 1998).

In the only prospective longitudinal study in which students' previous levels of depression were controlled, Roeser and Eccles (1998) verified the relationships between specific aspects of school environment and adolescents' depression development. In this study, no relationship was noted between students' levels of depression and school facilitation of student autonomy – an aspect of school discipline. The authors also concluded that students who perceived their schools as emphasizing learning over achievement presented less risk of developing depression, whereas those who perceived their schools as places where getting good grades is more important than personal development and learning presented a higher risk.

Remaining Questions

From the previous results, at least three limits of current knowledge are apparent. First, the impact of many facets of school life on depression development remains to be evaluated in an integrated, coherent and methodologically sound fashion. Indeed, current studies generally focused on very limited and idiosyncratic aspects of school

life and seldom provided controls for students' personal and social background characteristics. Moreover, given that schools are highly complex environments, characterized by multiple intertwined elements (Janosz, Georges *et al.*, 1998), a complete and precise understanding of school life effects on depression development ideally requires the simultaneous consideration of these elements.

Second, some of these results pinpoint gender differences regarding school life effects on depression development. Consequently, as these studies are still few, the moderating role of gender should be more thoroughly examined. Given that gender differences in depression prevalence emerge during early adolescence, following high school transition (Cyranowski *et al.*, 2000; Nolen-Hoeksema, 2002), changes in school life from elementary to middle school may represent potentially important determinants of these differences. Indeed, some hypotheses posit that the fact that adolescent girls experience more than boys the simultaneous occurrence of pubertal and school-related social changes may explain their increased rates of depression (Bebbington, 1996). Moreover, Eccles and colleagues (Eccles *et al.*, 1991, 1993) indicate that middle schools are often characterized by academic and social competitiveness and by social network disruptions. Both of these characteristics are known predictors of depression, particularly amongst females (Bebbington, 1996; Kessler & McLeod, 1984; Hankin & Abramson, 1999; Nolen-Hoeksema, 2002).

Finally, although the previously cited studies often provided control for previous levels of depression, these controls were often insufficient to disentangle the effects of school life characteristics on the *emergence* versus *aggravation* of depressive states. Briefly, statistical controls of previous levels of depression are generally used to account for the bidirectionality of the observed relationships between depression and purported risk factors. For example, depression represents a known predictor of school adaptation problems (Kessler, Foster, Saunders, & Stang, 1995). Consequently, to clearly conclude that school adaptation problems predict depression development, one must demonstrate that the effects observed are not due to students' baseline levels of depression. Although doing so allows depression antecedents to be

identified more clearly, it remains insufficient for research in which the ultimate goal is to guide preventive efforts. Indeed, depression prevention programs usually target undepressed individuals and strive to help them to remain well (Morin & Chalfoun, 2003; Mrazeck & Haggerty, 1994). Prevention programs should therefore be based on risk factors related to the *emergence* of depression rather than on factors related to its *aggravation*. For instance, if a risk factor predicts elevated levels of depression in already depressed individuals and shows no relationship with depression development in previously well individuals, this factor would be useless for preventionists, although very useful for clinicians. Kessler (1997) therefore urges scientists to systematically verify if the relationships between risk factors and depression development are moderated by subjects' baseline levels of depression (Baron & Kenny, 1986).

Depending on how scholars define depression, two approaches can be used to this end. First, in a categorical conception of depression in which one defines depression as a diagnostic entity qualitatively distinct from other related states (such as subclinical symptom levels), a subject is seen as either depressed or non-depressed (APA, 1994). In such a view, scholars can either eliminate already depressed subjects from their analyses (e.g., Lewinsohn *et al.*, 1994) or verify interactions between predictors and previous levels of depression defined in a present/absent manner. Resulting interactions can then be decomposed to evaluate if the proposed risk factors differently predict onsets versus recurrences of depression (e.g., Lewinsohn *et al.*, 1999). Second, the dimensional view depicts depression as a "normative" phenomenon positioned on a continuum somewhere between a state of complete emotional well-being and of handicapping depression (Akiskal, 2001; Zahn-Waxler *et al.*, 2000; Zuckerman, 1999). In such a view, interactions are more complex to interpret. Indeed, a significant interaction may mean that a risk factor exerts more significant effects at the lowest levels of the depressive spectrum or the reverse. However, risk factors may also be more or less potent at the midpoint of the spectrum.

THE PRESENT STUDY

The present exploratory study will attempt to provide preliminary answers to these remaining questions. More precisely, this study was designed to evaluate the specific nature of the relation between school life and depression development in adolescents. School life was defined in a global manner and encompasses three major dimensions: (a) *in-school psychological characteristics*, such as school motivation (academic self-efficacy, academic involvement, and extracurricular involvement) and school adaptation (school misbehaviors, academic delay, and academic achievement); (b) *school-related socialization experiences* involving parents (parental academic support and pressure), peers (loneliness at school, transitional difficulties, friends' school adaptation, and minor, major and sexual victimization at school), and school adults (school-related daily hassles, warm and supportive teacher-student relationships, conflictual teacher-students relations, and dissatisfaction with school discipline, academic control, help practices and encouragement); and (c) *students' perceptions of their schools'* climates (inter-students and teacher-student relational, bonding, justice, educational, and security climates), problems (minor violence, major violence, school-related problems), and practices (discipline, consultation, classroom management, extracurricular activities, support, school-family collaboration). More specifically, the present study will address the following question:

Which specific aspects of school life predict adolescent depression development once students' background characteristics are taken into account?

(a) Are these relationships different for boys and girls (moderated by gender)?

(b) Are these aspects equally relevant to the prediction of depressive symptoms' emergence among previously well students, of their aggravation among previously symptomatic students, and of their aggravation among previously clinically depressed students (are these relationships moderated by students' previous levels of depression)?

METHODOLOGY OF THE MADDP

Sample and Procedure

The Montreal Adolescent Depression Development Project (MADDP) is an ongoing prospective longitudinal study of over 1000 adolescents. All seventh grade students from five Montreal-area high schools (two private and three public) were approached and asked to participate in the project in September 2000, right after high school transition. Parents of the 1553 eligible participants were informed through a letter of the objective of the project and had the option to call the research team if they wished to withdraw their child from the study. The letter was accompanied by a consent form that described the initial transitional project, which comprised three measurement points across the school year[16]: September/October 2000 (two classroom periods, Time 1), January/February 2001 (two classroom periods, Time 2), and May/June 2001 (one classroom period, Time 3). Only 10 parents decided to withdraw their children from the study.

During the first data collection point, the remaining 1543 students were asked to read and sign a consent form similar to the parental one. They were also told that refusal or acceptance to sign this form would not prevent them from participating or not in later testing sessions if they changed their minds. Valid answers were provided by 1289 participants to both Time 1 questionnaires, which included most of the control variables: 66 participants refused to participate, 104 participants were absent, and 84 participants failed to provide valid answers to the questionnaires[17].

From these 1289 participants, 1167 (90.54%) provided valid answers to the Inventory to Diagnose Depression-Lifetime Version (IDD-L) administered at Time 3 (May/June 2001): 13 participants opted out of the study, 62 were absent at that time, 41 failed to

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[16] From this initial part of the project (the *transitional component*), the MADDP was continued to include three yearly measurement sessions. In Quebec, high school years extend from 7th through 11th grade. At time of writing of this paper, 10th grade evaluations were underway.

[17] More details regarding this last exclusion criterion are available upon request from first author.

provide valid answers to the questionnaire, and 6 were present but failed to complete the IDD-L. These 1167 subjects represent the sample used in the present analyses.

This final sample was predominantly of a French-speaking Caucasian background (78.2%) and almost equally split across gender (52.7% males; 47.3% females). Of these students, 50.6% attended public schools, 29.5% attended private schools, and 19.9% attended a public school for gifted students. Regarding school curricula, 19.54% of the students followed a regular program, 32.13% an enriched program, 29.31% a program for gifted students, and 19.02% attended a special education program for disadvantaged students. At Time 1, the average age of the participants was 12.75 years ($SD = 0.65$).

As all eligible students had the option to participate in the five testing sessions, students from the final sample could be compared with non-participants on most of the variables used in this study. Results from these attrition analyses revealed that non-participants generally differed from participants in that they presented a more problematic profile of psychosocial adaptation on most variables. More precisely, non-participants generally presented lower levels of personal adaptation (lower self-esteem, more behavioral disorders, higher levels of anxiety, etc.), came from more dysfunctional families, had more problems with their peers, and described their in-school socialization experiences and school environments more negatively.

Instruments

Dependent Variable: Depressive Symptoms

Depressive symptoms were evaluated at Time 1 and Time 3 using the French translation (Pariente, Smith, & Guelfi, 1989) of Zimmerman and Coryell's (1987 b) Inventory to Diagnose Depression – Lifetime Version (IDD-L). This instrument was developed to specifically answer the main criticisms generally addressed to self-reported depression severity scales: (a) non-specificity, or the fact that severity scales often include items which are not related to diagnostic criteria; (b) incompleteness, or the fact that severity scales sometimes do not cover the entire range of diagnostic

criteria; (c) diagnostic usefulness, or the fact that one can score high on a severity scale without meeting diagnostic criteria[18]; and (d) temporal specificity, or the fact that severity scales draw a time-specific portrait ignoring the possibility of recent remission. To answer the non-specificity and incompleteness criticisms, the authors chose items directly related to depression diagnostic criteria and covering their entire range (APA, 1994). The resulting instrument comprises 22 items, which are scored on a five-point behaviorally anchored rating scale[19]. Second, to answer the usefulness criticism, the authors developed three alternate scoring procedures for the IDD-L. Thus, a severity score highly similar to that of other self-reported depression scales can be obtained by adding participants' results on the different items (severity scoring). The authors also suggested cut-off scores for each symptom that allow for a more precise form of severity score based on the number of symptoms presented by participants (symptom scoring). Finally, once symptoms are scored, one can simply apply DSM-IV criteria to obtain a categorical diagnostic of depression. Finally, to address the time-specificity question, the IDD-L asks participants to answer by referring to the week of their life in which they felt the most depressed.

Validation studies showed that both the present and lifetime versions of the IDD present strong cross-cultural psychometric properties and a very high level of diagnostic sensitivity and specificity (e.g., Ackerson, Dick, Manson, & Baron, 1990; Krause, Philipp, Maier, & Schlegel, 1989; Sakado, Sato, Uehara, Sato, & Kameda, 1996; Zimmerman, & Coryell, 1987 a, 1987 b, 1988, 1994). In this study, the alpha for the severity scoring was 0.87 at Time 1 and 0.91 at Time 3, and the KR-20 coefficient for the symptom scoring was 0.79 at Time 1 and 0.84 at Time 3.

In this study, a continuous measure of depression based on the number of symptoms (symptom scoring) presented by each participant was used. However, analyses were replicated using severity scoring, and few differences were observed. Additionally,

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[18] For example, six items of the Beck Depression Inventory (Beck, Steer, & Brown, 1993), a known symptom severity scale, assess feelings of guilt and worthlessness. Thus one can score up to 18, a score which exceeds most suggested cut-off scores, only by feeling highly guilty.

[19] As an example, the item evaluating insomnia is: (0) I was not sleeping less than normal; (1) I occasionally had slight difficulty sleeping; (2) I clearly didn't sleep as well as usual; (3) I slept about half my normal amount of time; (4) I slept less than two hours per night.

two modifications were made to the IDD-L. First, as this instrument was designed when a previous version of the DSM was in use, three items referring to non-diagnostic symptoms (sexual drive and anxiety) had to be excluded from the final scoring. The final version of the IDD-L thus comprises 19 items. Second, the Time 3 IDD-L was modified to refer to the week in which participants felt the most depressed during the school year (see Appendix A for a copy of this instrument).

Controlled Variables[20]

The MADDP was designed specifically to study the mechanisms implicated in depression development. Most available variables therefore represented potential risk factors for depression. Among those, every variable which was not used to verify the research question and which represented a known predictor of depression development (see the preceding section) was used as a potential control. It should be noted that all controlled variables were measured at Time 1. The source, number of items, sample items, answer choices and internal consistency of the questionnaires used in this study are reported in Table I.

Demographic information. Gender and age (at October 1st, 2000) of the participants were obtained from school records.

Personal background characteristics. Measures of participants' personal background characteristics included their levels of *neuroticism* (or emotional instability), *anxiety*, *self-esteem*, and *body image satisfaction*, the frequency with which they exhibited socially deviant behaviors in the past year (*behavioral disorders*), and their levels of *pubertal development*. The behavioral disorder scale originally comprised 12 items, 2 of which referred to school misbehaviors. These 2 items were retrieved to be included in another subscale (see below).

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[20] Additional controls were tested for inclusion in this study (parental education, familial rules, time spent with family members, time spent with peers, trust in peers). However, as these variables did not even demonstrate a simple linear relationship with depression development, they are not described here for the sake of brevity.

Table I

Description of the measurement instruments used in the present study

Questionnaires	Authors	Items	Sample item	Answer choices	Consistency[21]
Controls: Personal background characteristics					
Neuroticism	Eysenk & Eysenk, (1963), LeBlanc (1998)	22	"Are your feelings easily hurt "	Yes/no	0.78
Self-esteem	Rosenberg (1965), Vallières & Vallerand (1990)	10	"I feel that I have a number of good qualities"	1- strongly disagree; 2- disagree; 3- agree; 4- strongly agree.	0.75 to 0.83
Body image	Marsh (1990), Ayotte et al. (2003)	8	"I am good looking"	1- false; 2- mostly false; 3- mostly true; 4- true	0.88 to 0.90
Anxiety	Beck, & Steer (1993), Freeston et al. (1994)	21	"Terrified," "Difficulty breathing"	0- not at all; 1- mildly; 2- moderately; 3- severely	0.89 to 0.91
Behavioral disorders	LeBlanc (1998)	10	In the last 12 months, did you: "Use hashish or marijuana," "Refuse to obey your parents"	1- never; 2- one or two times; 3- many times; 4- very often	0.77 to 0.79
Pubertal development	Petersen, Crockett, Richards, & Boxer (1988), Héroux (1997)	7	Generic (3 items): height Girls (2 items): menarche Boys (2 items): voice change	1 to 4 rating scale with body change descriptors.	Girls: 0.70 to 0.73 (0.79 to 0.81) Boys: 0.73 to 0.75 (0.81 to 0.83)
Controls: Life event exposure					
Stressful life events	Newcomb, Huba, and Bentler (1981), Baron, Joubert, & Mercier (1991)	25	In the last 12 months, did the following things happen to you: "I had acne eruptions," "I fell deeply in love,"	Treated as yes/no	Not applicable (NA)
Past difficulties	MADDP[22]	2	"Were you ever forced to participate in sexual acts without your consent," "Were you ever hospitalized for more than 20 days because you were very sick or had an accident"	Treated as yes/no	NA
Controls: Out-of school socialization experiences					
Parental monitoring	LeBlanc (1998)	2	"Your parents know where you are when you're not at home"	1- always; 2- often; 3- from time to time; 4- never	0.95*
Familial rules legitimacy	LeBlanc (1998)	1	"Do your parents create unfair rules?"	1-never; 2- from time to time; 3- many times; 4- often	NA
Parental punishment	LeBlanc (1998)	4	"Do your parents hit you when they want to punish you?"	1- never; 2- from time to time; 3- many times; 4- often	0.82*
Familial attachment	LeBlanc (1998)	10	"Do your parents let you know what they think and how they feel about things"	1- often, 2- many times, 3- from time to time, 4- never	0.83*
Familial instability	MADDP	5	"Did you move within the last year," "Are your parents still together?"	Treated as yes/no	NA
Familial daily hassles	MADDP	3	Do the following elements stress you: "your parents/ stepparents"	1- not at all; 2- a bit; 3- somewhat; 4- a lot	0.68 to 0.63 (0.82 to 0.85)
Communication with friends	LeBlanc (1998)	4	"Do you discuss the problems you have at home with your best friend"	1- often; 2- sometimes; 3- seldom; 4- never	0.83*

[21] For most scales, we report the internal consistency coefficients (α, KR-20) found in this study. However, for the scales included in LeBlanc (1998) SIQ (Social Inventory Questionnaire) and used integrally in this study, we report internal consistency coefficients taken from the SIQ manual. These coefficients are marked by * in Table 1. Additionally, as internal consistency is affected by the number of items, coefficients of shorter scales were adjusted (in parentheses) to eight equivalent items (Nunnally & Bernstein, 1994).

[22] Some questionnaires were developed specifically for the MADDP. Additional information on these questionnaires is available upon request from the first author (see Appendix B for a copy of these questionnaires).

Questionnaires	Authors	Items	Sample item	Answer choices	Consistency[21]
School-related psychological characteristics					
Academic self-efficacy	Skinner (1995), Janosz et al. (2001)	4	"I can get good grades at school when I want to"	1- false; 2- mostly false; 3- mostly true, 4- true	0.60 to 0.81 (0.86 to 0.94)
Academic involvement	Skinner (1995), Janosz et al. (2001)	6	"I study or do my homework everyday"	1- false; 2- mostly false; 3- mostly true, 4- true	0.52 to 0.80 (0.81 to 0.94)
Extracurricular involvement	Skinner (1995), Janosz et al. (2001)	2	"I spent many hours a week in extracurricular activities"	1- false; 2- mostly false; 3- mostly true, 4- true	0.59 to 0.71 (0.85 to 0.91)
School misbehaviors	LeBlanc (1998)	6	"Did you intentionally disturb your class"	1- never; 2- one or two times; 3- many times; 4- often	0.79*
Academic delay	LeBlanc (1998)	1	"Since you began elementary school, did you ever have to repeat a grade?"	1- never; 2- one time; 3- two times; 4- three times; 5- four times	NA
Academic achievement	MADDP	2	"What are your actual grades in French?," "What are your actual grades in mathematics"	1- Less than 60%; 2- 60 to 69%; 3- 70 to 79%; 4- 80 to 89%; 5- 90% +	NA
Parental school-related educative practices					
Parental support	Janosz et al. (2001)	6	"I can count on my parents when I have school difficulties"	1- false; 2- mostly false; 3- mostly true, 4- true	0.61 to 0.66 (0.68 to 0.72)
Parental pressure	MADDP	4	"When I get low grades, my parents make me feel guilty,"	Idem	0.72 (0.84)
In-school peer relationships					
Loneliness at school	Asher, Hymel and Renshaw (1984), Vitaro, Pelletier, Gagnon, & Baron (1995)	5	"I feel lonely at school"	1- not true; 2- a little bit true; 3- somewhat true; 4- very true	0.82 to 0.85 (0.88 to 0.90)
Peer-related daily hassles	MADDP	2	Do the following elements stress you: "My friends," "My classmates"	1- not at all; 2- a bit; 3- somewhat; 4- a lot	0.54 to 0.61 (0.82 to 0.86)
Transitional difficulties	MADDP	2	1) "How easy is it to make new friends at school?" 2) "Are you satisfied with the number of friends you have?"	1) 1- very easy; 2- easy; 3- hard; 4- very hard 2) 1-very satisfied; 2- satisfied; 3- unsatisfied; 4- very unsatisfied	0.57 to 0.61 (0.84 to 0.86)
Friends' school adaptation	Janosz, Rondeau, and Lacroix (1998)	8	"My best friends often talk about dropping out of school"	1- false; 2- mostly false; 3- mostly true, 4- true	0.71 to 0.74
Minor victimization	Janosz (2000)	5	Since the beginning of the school year: "An adult insulted or humiliated you at school"	1- never; 2- one time; 3- two times; 4- three times; 5- four times and more	0.57 (0.68)
Major victimization	Janosz (2000)	6	Since the beginning of the school year: "Students physically attacked you"	1- never; 2- one time; 3- two times; 4- three times; 5- four times and more	0.69 (0.75)
Sexual victimization	Janosz (2000)	3	Since the beginning of the school year: "My boyfriend or girlfriend shook, hit, or squeezed me"	1- never; 2- one time; 3- two times; 4- three times; 5- four times and more	0.70 (0.86)
Socialization experiences involving school adults					
School-related daily hassles	MADDP	6	Do the following elements stress you: "Your teachers," "Homework"	1- not at all; 2- a bit; 3- somewhat; 4- a lot	0.80 (0.84)
Warm and supportive teacher-student relations	Pianta and Steinberg (1992), Larose, Bernier, Soucy, & Duchesne (1999)	6	"I share warm and friendly relationships with my teachers"	1- not at all; 2- not really; 3- neutral; 4- somewhat; 5- very much	0.76 (0.81)
Conflictual teacher-student relations	Pianta and Steinberg (1992), Larose et al. (1999)	7	"Sometimes, I feel that I am unfairly treated by my teachers"	1- not at all; 2- not really; 3- neutral; 4- somewhat; 5- very much	0.85 (0.87)
Dissatisfaction: discipline	MADDP	3	"severity of school rules"	The elements are: not enough (1), enough (0), or too much (1) present	0.69 (0.86)

Questionnaires	Authors	Items	Sample item	Answer choices	Consistency[21]
Dissatisfaction: academic control	MADDP	2	"amount of homework and exams"	The elements are: not enough (1), enough (0), or too much (1) present.	0.57 (0.84)
Dissatisfaction: help	MADDP	3	"availability of help services for personal problems"	Should these elements be more present : yes/no	0.67 (0.84)
Dissatisfaction: encouragement	MADDP	2	"teachers' efforts to be motivating and interesting"	Should these elements be more present : yes/no	0.55 (0.83)
Perceived school climate					
Relational: inter-student	Janosz (2000)	6	"In this school, students can count on each other"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.76 (0.81)
Relational: teacher-student	Janosz (2000)	7	"In this school, teachers treat students with respect,"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.61 to 0.64 (0.83 to 0.86)
Bonding	Janosz (2000)	5	"Students feel at home in this school"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.78 (0.85)
Justice	Janosz (2000)	4	"The rules of this school are fair"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.75 (0.86)
Educational	Janosz (2000)	5	"What we learn in this school is important"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.75 (0.83)
Security	Janosz (2000)	5	"There are places in this school were students are afraid to go."	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.67 to 0.73 (0.88 to 0.89)
Perceived school problems					
Minor violence	Janosz (2000)	6	Since the beginning of the school year, how often did you observe: "thefts," "insults," "vandalism."	1- never; 2- a few times during the school year; 3- a few times per months; 4- a few times per week; 5- nearly every day	0.79 (0.83)
Major violence	Janosz (2000)	4	Since the beginning of the school year, how often did you observe: "fights," "attacks of adults by students"	1- never; 2- a few times during the school year; 3- a few times per months; 4- a few times per week; 5- nearly every day	0.67 (0.80)
School-related	Janosz (2000)	5	Since the beginning of the school year, how often did you observe: "cheating," "truancy," "classroom disturbance"	1- never; 2- a few times during the school year; 3- a few times per months; 4- a few times per week; 5- nearly every day	0.79 (0.86)
Perceived school practices					
Disciplinary practices	Janosz (2000)	11	"School rules are easy to understand," "Teachers apply school rules as prescribed"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.61 to 0.64 (0.83 to 0.86)
Student consultation	Janosz (2000)	2	"Students' opinions regarding school operations are taken into consideration "	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.64 (0.88)
Classroom management	Janosz (2000)	16	"We seldom see teachers yelling at students," "Teachers explain why new subject matters are important"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.64 to 0.80 (0.80 to 0.83)
Extracurricular activities	Janosz (2000)	3	"Extracurricular activities are interesting"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.64 (0.83)
Support mechanisms	Janosz (2000)	3	"In this school, the different help services/resources can really help students who encounter academic or personal problems"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.76 (0.89)
School-family collaboration	Janosz (2000)	5	"Parents often participate in school committees or activities"	1- totally agree; 2- agree; 3- disagree; 4- totally disagree	0.71 (0.80)

Life event exposure. Participants' exposures to generic stressful life events during the past year as well as to *past personal difficulties* were evaluated. The *generic stressful life events* questionnaire originally included 39 items. Due to theoretical and practical considerations, five items had to be excluded from the current questionnaire.

Out-of-school socialization experiences. Measures of participants' out-of-school socialization experiences include *parental monitoring*, or parental knowledge of adolescents' friends and activities; *legitimacy of familial rules*, or the perceived legitimacy of the rules imposed on adolescents by their parents; *parental punishment*, or the use of punishment practices by parents; *familial attachment*, or the affective quality of parent-adolescent contacts, communication, affective bonding and mutual acceptance; *familial instability*, or the amount of changes experienced within the participants' families; *familial daily hassles*, or participants' perceptions of their families' stressfulness ; and *communication with friends*, or the degree to which participants feel free to discuss personal matters and problems with their peers.

Predictors

All the predictors used in the present study were measured at Time 2. Three dimensions of school life were tested as potential predictors of depression development: students' *school-related psychological characteristics*, *school-based socialization experiences*, and *perceptions of their school environment*. Students' *school-based socialization experiences* were further divided into three sub-dimensions: *parental school-related educative practices*, *in-school peer relationships*, and *socialization experiences involving school adults*. Finally, students' *perceptions of their school environment* were also divided into the three sub-dimensions from Janosz, Georges *et al.*'s (1998) theoretical model: *school climate*, or the generic atmosphere permeating the school environment (values, feelings, and attitudes shared by students); *school problems*, or aspects of students' behaviors that may negatively impact on the overall quality of the school environment and on students' adaptation;

and *school practices*, or school attempts to regulate, facilitate, and influence students' behaviors. These three sub-dimensions were evaluated with Janosz' (2000) SEQ (Socio Educational Questionnaire), a questionnaire directly based on this theoretical model and validated in a large sample of Quebec adolescents.

School-related psychological characteristics. Measures of participants' school-related psychological characteristics included participants' *academic self-efficacy*, or feelings of academic competency and of personal control over academic tasks; *academic involvement*, or levels of involvement (efforts) in academic tasks, attitudes toward school, and academic aspirations; *extracurricular involvement*, or degree of participation in extracurricular activities; *school misbehaviors*, or the frequency with which they exhibit school misbehaviors; *academic delay*, or the number of times students had to repeat a school year; and *academic achievement*, or participants' average grades in French (main language) and mathematics. It should be noted that whereas we had access only to students' self-reports of academic achievement at Time 2, we also received school records of students' grades at Time 3. Time 3 self-reported measures of academic achievement could thus be compared with official records. The correlation observed between students' self-reports and their report cards varied between $r = 0.671$ ($p = 0.000$) and $r = 0.798$ ($p = 0.000$), which confirms the validity of students' self-reports at Time 2.

Parental school-related educative practices. Measures of parental school-related educative practices include *parental academic support*, or parental provision of help and support to students regarding academic tasks; and *parental pressure*, or the degree to which parents expose their children to achievement pressure.

In-school peer relationships. Measures of in-school peer relationships include *loneliness*, or the degree to which students feel lonely at school; *peer-related daily hassles*, or participants' perceptions of the stressfulness of relationships with peers and classmates; *transitional difficulties*, or participants' friendship difficulties due to school transition; *friends' school adaptation*, or participants' perceptions of the

degree of school involvement, adjustment and valorization of their peers; and *minor, major and sexual victimization*, or the frequency with which participants were victims of minor, major or sexual acts of violence at school since the beginning of the year.

Socialization experiences involving school adults. Measures of participants' socialization experiences involving school adults include *school-related daily hassles*, participants' perceptions of the stressfulness of their school life; *warm and supportive teacher-student relationships*, or the degree to which participants' relationships with their teachers were characterized by warmth and support; *conflictual teacher-student relationships*, or the degree to which participants' relationships with their teachers were characterized by conflict. Four additional subscales assessed students' personal dissatisfaction with their school's *disciplinary control practices* (rules, punishments, etc.), *academic control practices* (amount of homework, exams, etc.), *help services* (academic and personal), and *encouragement practices* (encouragement offered by teachers).

Perceived school climate. Measures of perceived school climate include *inter-student relational climate*, or the degree to which interactions between students at school are characterized by warmth, trust, and respect; *teacher-student relational climate*, or students' respect for their teachers, teachers' respect toward students, and teachers' warmth and support toward students; *bonding climate*, or the general feeling of school belongingness as perceived by students; *justice climate*, or the degree to which students perceive their school environment as equitable and respectful of individual differences; *educational climate*, or students' perceptions of how much education is emphasized and valorized within their school; and *security climate*, or students' perceptions of teachers' and students' feelings of security within school.

Perceived school problems. Measures of perceived school problems include perceived frequency of *minor* and *major violence problems* and of *school-related problems.*

Perceived school practices. Measures of perceived school practices include *quality of school disciplinary practices*, or the implementation quality of school rules, students' knowledge of school rules, and consistent application of school rules; *student consultation* (regarding school rules and operations); *teachers' classroom management practices*, or the quality of teachers' pedagogical and behavioral management practices, and teachers' motivation; *school extracurricular activities* (quality and availability); *quality and availability of school support mechanisms* (to help students with academic or personal problems); and *quality of school-family collaboration* (information, involvement in decisions, etc.).

Analytical Strategy

Missing Data Replacement

To reduce the amount of missing data, two complementary strategies were used. First, variable scores were computed allowing for 25% to 33% of missing values on scale items. Once this strategy was applied, 0% to 14.82% of the participants still had missing values on the variables studied (M = 5.40%; SD = 4.19%), although few of them had recurring patterns of missing data. Missing data were replaced with variable means to which a random "number" was added according to the variables' standard deviations[23]. This procedure allows for the correction of the variance restriction problem inherent in simple mean-replacement strategies (Little & Rubin, 2002). For further precision in the missing data replacement process, variable means and standard deviations were calculated separately for 32 participant subgroups on the basis of gender (male, female), age (11-12.49, 12.5-12.99, 13-13.99, 14+), and school[24]. This strategy was inspired by the Hot-Deck method described by Allison (2001).

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[23] This was accomplished through the following SPSS 10.0 function: IF (MISSING (variable)) variable = RV.NORMAL (MEAN,SD) .

[24] The total number of subgroups was reduced to 32 instead of 40 (gender X age X schools = 2 X 4 X 5 = 40) because no participants from school 5 were over 13 years old and no participants from schools 1 and 2 were over 14 years old.

Choosing The Control Variables

Statistical controls were identified using a sequential strategy in which the main objective was to maximally reduce the number of variables included in the analyses. We sought to limit the number of variables included in the analyses to maximize statistical power and to limit potential multicollinearity and model specificity problems[25]. First, separate linear regressions were conducted to confirm the predictive role of the controls regarding Time 3 depressive symptoms. Second, these analyses were replicated while controlling for Time 1 depressive symptoms. Some predictors thus became non-significant and were excluded from the remaining analyses. Finally, all of the remaining predictors were entered together as a block in a hierarchical multiple regression analysis in which Time 1 depressive symptoms were controlled. This analysis allowed us to retain only the most significant predictors of Time 3 depressive symptoms as controls.

Answering The Research Question

As the definition of school life used in the present study implied many interrelated predictors, the analytical strategy was again designed to limit the number of variables entered simultaneously in the analyses. Once more, the objective of this strategy was to limit multicollinearity and model specificity problems and to maximize statistical power. Additionally, this strategy allowed us to interpret more clearly the effects of all variables. Whereas it could be argued that variables could have been grouped into higher order factors[26], a more exploratory approach was preferred. The main objective of the present study is to guide school-based preventive interventions. In this context, the fact that two predictors share common variance and that the effects of one are, statistically, completely explained by the effects of the other does not mean that both are not valid targets for preventive programs.

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[25] As a preliminary attempt to limit the number of variables, exploratory factor analyses were conducted on the potential controls. However, the observed groupings were few and inconsistent, confirming the relative independence of the variables.

[26] Once again, preliminary exploratory factor analyses were conducted on the various predictors and the observed groupings were few and sometimes hard to interpret and inconsistent.

Analyses were thus conducted separately to verify the predictive role of the seven dimensions of school life (school-related psychological characteristics, parental school-related educative practices, in-school peer relationships, socialization experiences involving school adults, perceived school climate, perceived school problems, and perceived school practices) evaluated in the present study regarding Time 3 depressive symptoms. First, separate regressions were conducted to evaluate the effects of each predictor on Time 3 depressive symptoms once Time 1 depressive symptoms were taken into account. Second, the remaining significant predictors were entered together as a block in a hierarchical multiple regression analysis in which only Time 1 levels of depressive symptoms were controlled. Once again, non-significant predictors were excluded from subsequent analyses. Third, the remaining significant predictors were entered together as a block in a hierarchical multiple regression analysis in which Time 1 depressive symptoms and background controls were included. Once these analyses were conducted separately for each of the seven sub-dimensions of school life, they were replicated at the dimensional level (school-related psychological characteristics, school-related socialization experiences, and perceived school environment). In these analyses, significant predictors from the preceding analyses were entered together as a block in a hierarchical multiple regression analysis in which Time 1 symptoms and controls were included.

Answering Sub-Questions

In practice, evaluating moderating relationships implies demonstrating that the interaction term composed by the product of both predictors significantly adds to the model over and above the main effects of both variables (Aiken & West, 1991; Baron & Kenny, 1986; Cohen & Cohen, 1983; Holmbeck, 2002; Jaccard & Turisi, 2003; West, Aiken & Krull, 1996). As interaction terms are obtained through the multiplication of subjects' scores on both variables, which are already included in the model, multicollinearity problems may result from this procedure. To prevent this, all independent variables used in this study were converted to deviation score form by subtracting the variable mean from each individual score (i.e. centered at the mean).

This procedure, which is known to considerably reduce multicollinearity problems, may also serve to clarify interaction effects (Aiken, & West, 1991; Cohen & Cohen, 1983; Jaccard & Turisi, 2003; Kreft, Leeuw & Aiken, 1995; West *et al.*, 1996). To further limit multicollinearity, interaction effects were tested separately, in independent regressions. In these regressions, the interaction term was entered last, following the main effects of both predictors. In the evaluation of gender-based interactions, students' Time 1 depressive symptoms were controlled in the analyses[27].

To achieve maximal clarity in decomposing the significant interactions, the effects of each predictor (P) interacting with gender and/or Time 1 depressive symptoms were tested separately at different levels of the moderators (M) (Aiken & West, 1991; Jaccard & Turisi, 2003). Briefly, in multiple regressions in which two-way interaction terms (M * P) are entered after the main effects of both the predictor and the moderator (M and P), the *b* coefficient associated with each predictor (P) represents the slope of the Y on P regression equation when the moderator (M) equals zero. As each variable was centered at the mean, the *b* coefficient associated with P in regressions, including both M and M * P terms, represents the effect of P on Y at the mean value of M. To obtain an estimate of the effects of P at different values of M, one only has to add or subtract constants to M so that zero represents different values and to compare the relative strength of the resulting *b* coefficients to interpret the interaction. In the decomposition of gender-based interactions, regressions were thus replicated twice to estimate the effect of each predictor in males (males coded 0 and females coded 1) and females (reversed coding: males = 1 and females = 0). In these regressions, only Time 1 levels of depressive symptoms were controlled. For the interactions involving Time 1 depressive symptoms, a similar strategy was employed, but no controls were included in the analyses. In the decomposition of these interactions, the effects of the predictors were evaluated at three different levels of Time 1 depressive symptoms, following Kessler's (1997) suggestion (see previous

[27] The analyses in which the interactions were tested and decomposed were replicated three times, adding additional controls (1- Time 1 depressive symptoms; 2- Time 1 depressive symptoms and background controls; 3- Time 1 depressive symptoms, background controls, and other variables from the sub-dimension). The results did not change across these replications but were easier to interpret with minimal controls. Therefore, only the results from the first analyses are reported.

section): (1) asymptomatic (zero reflects the absence of symptoms); (2) symptomatic (one to four symptoms: zero reflects the mid-point, 2.5 symptoms); (3) or clinical (five or more symptoms: zero reflects five symptoms). For the last group, the label "clinical" was preferred to "diagnostic" to account for the fact that full diagnostic criteria were not applied to the delineation of this group.

The Final Model

The most robust predictors of depression development were finally identified in three separate hierarchical multiple regressions, one for each dimension of school life. In these regressions, four blocks of predictors were entered sequentially: (1) Time 1 depressive symptoms; (2) background controls; (3) significant predictors (main effects) from the preceding analyses; and (4) significant interaction terms from the preceding analyses. The significant predictors from these three separate analyses were then entered together in a final regression to estimate the total contribution of school life to depression development.

RESULTS

Normality and Multivariate Outliers

Inspection of the skewness and kurtosis of the different variables revealed that few of them showed normal distributions (i.e., most values were over twice their associated standard errors). However, this assumption is seldom respected in large samples in which the standard error of skewness and kurtosis are often reduced (Tabachnick & Fidell, 1996). Additionally, due to the central limit theorem, in large samples, deviation from normality seldom significantly affects the results of multivariate analyses (de Vaus, 2002; Lewis-Beck, 1980; Tabachnick & Fidell, 1996). Nevertheless, variables with skewness and/or kurtosis values over 1 were transformed and the analyses were replicated with and without these transformations. With one

exeption, these replications showed no significant difference; variables were thus kept in their original state. The only exeption was for the loneliness at school scale, for which the inverse transformation yielded significantly different results (Tabachnick & Fidell, 1996). This scale was thus transformed by inversion before its inclusion in the following analyses. The sign of the *r*, *b*, ß, and *t* coefficients associated with this variable thus has to be reversed before interpretation. Furthermore, the fact that the residuals from all analyses produced normal distributions clearly indicates that the multivariate normality assumption of multiple regressions was respected even without transforming the other variables (Tabachnick & Fidell, 1996)[28].

Multivariate outliers were identified by examining bivariate scatterplots of subjects' Cooks' Distances, Leverages, and Mahalnobis' D^2 (Tabachnick & Fidell, 1996). This procedure identified 14 potential multivariate outliers. Additional analyses revealed that these subjects differed from the others by exhibiting a more severe pattern of psychosocial adaptation problems. As more seriously affected students were already lost through the attrition process, we decided to keep the multivariate outliers in the analyses. However, most analyses were first replicated with and without these subjects and the results did not significantly change, thus confirming the decision to keep these subjects in the analyses.

Choosing the Control Variables

Results from the analyses designed to reduce the number of control variables are reported in Table II. The first set of univariate analyses confirmed that all of the proposed control variables represented significant predictors of depression development. It should also be noted that the relationship between Time 1 and Time 3 depressive symptoms was quite strong (ß = 0.460, $t = 17.698$, p = 0.000, $R^2 = 0.211$). When the analyses were replicated while controlling for previous levels of depressive symptoms, most of the family-related variables, with the exception of familial

[28] Additional inspection of expected versus predicted residual scatterplots and of normal probability plots of the residuals from each of the following regression analyses indicates that the homoscedasticity and linearity assumptions of multiple regression were also respected.

Table II

Relationships between controlled variables and Time 3 depressive symptoms

	Univariate effects			Univariate effects, depression control			Multivariate effects, depression control		
	Beta	t	p	Beta	t	p	Beta	t	p
Age	0.095	3.257	0.001	0.083	3.189	0.001	0.023	0.796	0.426
Gender	0.112	3.864	0.000	0.041	1.551	0.121	0.002	0.059	0.953
Neuroticism	0.304	10.883	0.000	0.150	5.390	0.000	0.029	0.898	0.369
Self-esteem	-0.271	-9.611	0.000	-0.176	-6.728	0.000	-0.107	-3.570	0.000
Body image	-0.187	-6.510	0.000	-0.109	-4.139	0.000	-0.044	-1.568	0.117
Anxiety	0.294	10.494	0.000	0.136	4.859	0.000	0.064	2.113	0.035
Behavioral disorders	0.152	5.261	0.000	0.087	3.320	0.001	0.012	0.430	0.667
Pubertal development	0.162	5.604	0.000	0.102	3.906	0.000	0.049	1.667	0.096
Stressful life events	0.202	7.030	0.000	0.108	4.100	0.000	0.031	1.091	0.275
Past personal difficulties	0.062	2.110	0.035	0.031	1.181	0.238			
Parental monitoring	-0.083	-2.852	0.004	-0.034	-1.295	0.196			
Rules legitimacy	-0.116	-4.001	0.000	-0.037	-1.389	0.165			
Parental punishment	0.090	3.089	0.002	0.016	0.617	0.537			
Familial attachment	-0.118	-4.069	0.000	-0.050	-1.896	0.058			
Familial instability	0.087	2.991	0.003	0.070	2.705	0.007	0.023	0.879	0.380
Familial daily hassles	0.257	9.073	0.000	0.158	5.982	0.000	0.088	3.192	0.001
Communication with friends	0.201	7.008	0.000	0.108	4.075	0.000	0.086	3.100	0.002
R^2 change									0.065

instability and daily hassles, became non-significant predictors of depressive symptoms. Most of the other variables, however, remained significant predictors of later levels of depressive symptoms, with the sole exception of exposure to past personal difficulties. It should be noted that although gender became a non-significant predictor of Time 3 depressive symptoms when Time 1 symptoms were controlled in the analyses, we decided to keep this variable as a control in subsequent analyses due to later testing of gender-based interactions. Finally, when all of the previously identified significant predictors were considered together in a multiple hierarchical regression analysis, only four of the purported control variables still predicted Time 3 depressive symptoms: anxiety, self-esteem, familial daily hassles, and communication with friends. Together, these variables explained 6.5% of Time 3 depressive symptoms' variance. When this analysis was replicated including only the significant predictors, the resulting model explained 6% of depressive symptoms' variance.

Correlations Among Predictors and Controls

The correlations between the variables used as controls and predictors in the present study are reported in Table III. An analysis of these correlations confirms the adequacy of the selected controls, including Time 1 depressive symptoms, as these variables all shared significant relationships with various aspects of students' school life. These correlations also confirm the highly interrelated character of the different aspects of students' school life. Indeed, most of the school life variables shared low to moderate correlations with each other. However, these correlations are generally low enough to justify their separate consideration in the analyses. The only exception was found among some aspects of students' perceptions of their school environment where 12 of the observed correlations were higher than 0.5 (only three of these correlations were higher than 0.6 and none exceeded 0.7, which would have indicated a potential multicollinerarity problem). Since such interrelations were already postulated in Janosz, Georges *et al.*'s (1998) theoretical model, since validation analyses of the SEQ confirmed the existence of distinct and interrelated factors, and since different forms of preventive interventions would be needed to act on these

Table III

Zero-order correlations among predictors and controls

	COMF	ANX	SE	FDH	D	AA	AINV	ASE	EXI	ISB	FAMS	FAMP	TP	LS (I)
ANX	0.125*													
SE	-0.018	-0.259*												
FDH	0.118*	0.248*	-0.243*											
D	-0.030	0.067*	-0.217*	0.095*										
AA	0.105*	-0.026	0.205*	-0.084*	-0.267*									
AINV	0.014	-0.060*	0.237*	-0.171*	-0.241*	0.245*								
ASE	0.009	-0.057	0.254*	-0.103*	-0.209*	0.376*	0.294*							
EXI	-0.015	0.037	0.050	-0.030	-0.023	0.030	0.162*	0.026						
ISB	0.041	0.101*	-0.181*	0.185*	0.339*	-0.308*	-0.428*	-0.257*	-0.052					
FAMS	-0.016	-0.064*	0.177*	-0.158*	-0.149*	0.064*	0.413*	0.184*	0.103*	-0.290*				
FAMP	0.040	0.086*	-0.069*	0.165*	-0.014	-0.080*	-0.081*	-0.110*	0.020	0.058*	-0.188*			
TP	-0.016	0.125*	-0.235*	0.060*	-0.030	-0.032	-0.083*	-0.051	-0.080*	-0.035	-0.070*	0.074*		
LS (I)	0.062*	-0.130*	0.215*	-0.103*	-0.029	0.083*	0.074*	0.063*	0.073*	0.000	0.081*	-0.071*	-0.521*	
PDH	0.069*	0.224*	-0.230*	0.218*	0.072*	-0.041	-0.142*	-0.029	-0.019	0.119*	-0.089*	0.108*	0.295*	-0.378*
FSA	0.061*	-0.059*	0.203*	-0.177*	-0.345*	0.280*	0.467*	0.256*	0.111*	-0.412*	0.321*	-0.095*	-0.036	0.077*
MINV	0.097*	0.208*	-0.167*	0.151*	0.063*	-0.068*	-0.098*	-0.106*	-0.018	0.228*	-0.065*	0.101*	0.164*	-0.204*
MAJV	0.031	0.131*	-0.152*	0.150*	0.144*	-0.073*	-0.132*	-0.170*	0.034	0.345*	-0.143*	0.014	0.024	-0.089*
SEXV	0.052	0.091*	-0.101*	0.129*	0.132*	-0.109*	-0.106*	-0.158*	0.018	0.325*	-0.089*	-0.037	-0.039	0.000
SDH	0.093*	0.301*	-0.267*	0.279*	0.118*	-0.157*	-0.282*	-0.184*	-0.055	0.304*	-0.176*	0.133*	0.144*	-0.165*
TS	0.088*	0.074*	0.047	-0.011	-0.072*	0.167*	0.269*	0.123*	0.147*	-0.140*	0.171*	-0.073*	-0.008	-0.035
TC	0.116*	0.100*	-0.177*	0.189*	0.124*	-0.146*	-0.362*	-0.253*	-0.103*	0.421*	-0.295*	0.204*	0.062*	-0.052
DSH	-0.048	0.095*	-0.144*	0.046	0.124*	-0.127*	-0.064*	-0.133*	0.057	0.115*	-0.106*	0.095*	0.075*	-0.065*
DSE	0.029	0.131*	-0.020	0.050	-0.015	-0.051	-0.071*	-0.062*	0.013	0.080*	-0.040	0.053	0.067*	-0.037
DSAC	0.103*	0.074*	-0.033	0.055	-0.051	-0.009	-0.164*	-0.100*	-0.084*	0.108*	-0.114*	0.027	0.063*	-0.009
DSDC	0.076*	0.063*	-0.069*	0.089*	0.070*	-0.126*	-0.256*	-0.152*	-0.073*	0.248*	-0.144*	0.108*	-0.018	0.017
BC	0.012	-0.093*	0.171*	-0.105*	-0.108*	0.169*	0.345*	0.173*	0.130*	-0.304*	0.203*	-0.077*	-0.131*	0.140*
BSRC	0.057	-0.138*	0.257*	-0.118*	-0.231*	0.135*	0.263*	0.173*	0.115*	-0.264*	0.206*	-0.057	-0.250*	0.277*
TSRC	-0.033	-0.129*	0.162*	-0.082*	-0.177*	0.132*	0.321*	0.163*	0.100*	-0.336*	0.286*	-0.075*	-0.088*	0.073*
EC	0.008	-0.049	0.190*	-0.093*	-0.184*	0.149*	0.407*	0.240*	0.118*	-0.392*	0.289*	-0.057	-0.076*	0.038
JC	-0.035	-0.053	0.133*	-0.101*	-0.144*	0.137*	0.358*	0.224*	0.071*	-0.323*	0.246*	-0.149*	-0.034	0.021
SC	0.028	-0.139*	0.191*	-0.093*	-0.203*	0.191*	0.217*	0.261*	0.057	-0.290*	0.217*	-0.055	-0.067*	0.130*
MINP	0.081*	0.171*	-0.146*	0.148*	0.137*	-0.127*	-0.191*	-0.056	0.001	0.342*	0.114*	0.043	-0.013	-0.050
MAJP	0.036	0.129*	-0.192*	0.154*	0.292*	-0.229*	-0.280*	-0.195*	-0.024	0.484*	-0.226*	0.018	-0.054	0.004
SCOP	0.052	0.137*	-0.164*	0.088*	0.177*	-0.191*	-0.253*	-0.072*	-0.019	0.343*	-0.132*	0.056	0.013	-0.048
SDP	-0.038	-0.083*	0.123*	-0.140*	-0.051	0.026	0.232*	0.142*	0.051	-0.254*	0.254*	-0.098*	-0.090*	0.076*
SSC	-0.027	-0.042	0.025	-0.018	0.093*	-0.031	0.057*	-0.030	0.052	-0.028	0.029	-0.032	-0.081*	0.042
TCMP	-0.038	-0.085*	0.217*	-0.128*	-0.163*	0.152*	0.337*	0.257*	0.065*	-0.337*	0.330*	-0.129*	-0.087*	0.049
EXAC	0.006	-0.057	0.112*	-0.099*	-0.089*	0.052	0.251*	0.131*	0.245*	-0.243*	0.220*	-0.022	-0.084*	0.079*
SHP	0.036	-0.104*	0.140*	-0.074*	-0.062*	0.040	0.192*	0.112*	0.032	-0.179*	0.156*	-0.094*	-0.093*	0.102*
PSC	0.010	-0.107*	0.103*	-0.089*	-0.073*	0.059*	0.208*	0.087*	0.094*	-0.229*	0.234*	-0.068*	-0.081*	0.026
IDD-L1	0.213*	0.388*	-0.225*	0.234*	0.007	0.024	-0.038	-0.001	0.006	0.083*	-0.093*	0.062*	0.155*	-0.166*

Table III (continued)

	PDH	FSA	MINV	MAJV	SEXV	SDH	TS	TC	DSH	DSE	DSAC	DSDC
FSA	-0.152*											
MINV	0.220*	-0.089*										
MAJV	0.094*	-0.172*	0.415*									
SEXV	0.075*	-0.146*	0.258*	0.583*								
SDH	0.392*	-0.283*	0.230*	0.103*	0.127*							
TS	0.012	0.257*	0.003	0.057	-0.035	-0.135*						
TC	0.155*	-0.341*	0.208*	0.119*	0.089*	0.416*	-0.264*					
DSH	0.084*	-0.121*	0.069*	0.063*	0.072*	0.146*	-0.031	0.096*				
DSE	0.074*	-0.045	0.042	-0.022	0.028	0.202*	-0.059*	0.192*	0.459*			
DSAC	0.066*	-0.081*	0.086*	0.013	0.029	0.228*	-0.123*	0.225*	0.086*	0.164*		
DSDC	0.048	-0.244*	0.105*	0.056	0.087*	0.196*	-0.247*	0.328*	0.076*	0.115*	0.322*	
BC	-0.170*	0.295*	-0.236*	-0.101*	-0.076*	-0.279*	0.282*	-0.342*	-0.118*	-0.159*	-0.161*	-0.188*
BSRC	-0.254*	0.269*	-0.264*	-0.237*	-0.163*	-0.210*	0.097*	-0.184*	-0.108*	-0.108*	-0.057	-0.04
TSRC	-0.165*	0.283*	-0.220*	-0.150*	-0.129*	-0.262*	0.183*	-0.335*	-0.116*	-0.160*	-0.126*	-0.107*
EC	-0.102*	0.349*	-0.219*	-0.179*	-0.176*	-0.291*	0.205*	-0.382*	-0.148*	-0.170*	-0.159*	-0.174*
JC	-0.060*	0.261*	-0.216*	-0.126*	-0.079*	-0.227*	0.220*	-0.403*	-0.081*	-0.149*	-0.193*	-0.372*
SC	-0.136*	0.268*	-0.231*	-0.225*	-0.182*	-0.201*	-0.044	-0.214*	-0.221*	-0.128*	-0.012	-0.013
MINP	0.156*	-0.177*	0.341*	0.119*	0.052	0.257*	-0.016	0.264*	0.101*	0.085*	0.079*	0.118*
MAJP	0.126*	-0.300*	0.243*	0.306*	0.279*	0.234*	-0.034	0.264*	0.144*	0.036	0.006	0.103*
SCOP	0.157*	-0.219*	0.221*	0.047	0.010	0.246*	-0.054	0.252*	0.116*	0.138*	0.053	0.098*
SDP	-0.102*	0.229*	-0.218*	-0.205*	-0.125*	-0.250*	0.086*	-0.281*	-0.075*	-0.104*	-0.140*	-0.179*
SSC	-0.067*	0.028	-0.153*	0.056	0.048	-0.137*	0.107*	-0.131*	0.061*	-0.067*	-0.084*	-0.114*
TCMP	-0.165*	0.304*	-0.221*	-0.175*	-0.128*	-0.297*	0.227*	-0.452*	-0.173*	-0.234*	-0.184*	-0.201*
EXAC	-0.093*	0.211*	-0.118*	-0.094*	-0.033	-0.173*	0.170*	-0.295*	-0.053	-0.144*	-0.162*	-0.145*
SHP	-0.135*	0.142*	-0.176*	-0.139*	-0.114*	-0.191*	0.066*	-0.225*	-0.147*	-0.164*	-0.101*	-0.137*
PSC	-0.103*	0.205*	-0.144*	-0.075*	-0.041	-0.176*	0.173*	-0.248*	-0.149*	-0.194*	-0.125*	-0.154*
IDD-L1	0.212*	-0.004	0.161*	0.042	0.012	0.265*	0.033	0.142*	0.039	0.077*	0.103*	0.085*

Table III (continued)

	BC	BSRC	TSRC	EC	JC	SC	MINP	MAJP	SCOP	SDP	SSC	TCMP	EXAC	SHP	PSC
BSRC	0.493*														
TSRC	0.587*	0.570*													
EC	0.629*	0.449*	0.565*												
JC	0.435*	0.245*	0.422*	0.457*											
SC	0.220*	0.275*	0.310*	0.288*	0.221*										
MINP	-0.367*	-0.198*	-0.364*	-0.323*	-0.250*	-0.330*									
MAJP	-0.275*	-0.241*	-0.351*	-0.354*	-0.262*	-0.411*	0.613*								
SCOP	-0.375*	-0.200*	-0.379*	-0.302*	-0.244*	-0.300*	0.694*	0.533*							
SDP	0.329*	0.313*	0.385*	0.417*	0.416*	0.175*	-0.166*	-0.195*	-0.126*						
SSC	0.259*	0.156*	0.197*	0.186*	0.213*	-0.095*	-0.063*	0.030	-0.105*	0.267*					
TCMP	0.554*	0.383*	0.558*	0.583*	0.514*	0.347*	-0.275*	-0.313*	-0.304*	0.401*	0.214*				
EXAC	0.406*	0.288*	0.352*	0.395*	0.320*	0.179*	-0.164*	-0.141*	-0.139*	0.389*	0.230*	0.412*			
SHP	0.393*	0.319*	0.372*	0.408*	0.343*	0.149*	-0.112*	-0.124*	-0.113*	0.397*	0.240*	0.405*	0.338*		
PSC	0.480*	0.298*	0.404*	0.457*	0.365*	0.119*	-0.208*	-0.178*	-0.217*	0.422*	0.340*	0.439*	0.504*	0.399*	
IDD-L1	-0.086*	-0.094*	-0.064*	-0.033	-0.048	-0.029	0.104*	0.028	0.087*	-0.072*	-0.060*	-0.078*	-0.054	-0.064*	-0.062*

Legend. * Correlation is significant at the 0.05 level (2-tailed); **COMF**: Communication with friends; **ANX**: anxiety; **SE**: Self-esteem; **FDH**: Familial daily hassles; **D**: Academic delay; **AA**: Academic achievement; **AINV**: Academic involvement; **ASE**: Academic self-efficacy; **EXI**: Extracurricular involvement; **ISB**: School misbehaviors; **FAMS**: Parental academic support; **FAMP**: Parental academic pressure; **TP**: Transitional difficulties; **LS (I)**: Loneliness at school (inversed); **PDH**: peer-related daily hassles; **FSA**: Friends' school adaptation; **MINV**: Minor victimization; **MAJV**: Major victimization; **SEXV**: sexual or romantic victimization; **SDH**: School-related daily hassles; **TS**: Warm and supportive teacher-student relationships; **TC**: Conflictual teacher-student relationships; **DSH**: dissatisfaction with school help mechanisms; **DSE**: dissatisfaction with school encouragement mechanisms; **DSAC**: dissatisfaction with school academic control; **DSDC**: dissatisfaction with school disciplinary control; **BC**: Bonding climate; **BSRC**: Inter-student relational climate; **TSRC**: teacher-student relational climate; **EC**: Educational climate; **JC**: Justice Climate; **SC**: Security climate; **MINP**: Perceived frequency of minor violence problems; **MAJP**: Perceived frequency of major violence problems; **SCOP**: Perceived frequency of school-related problems; **SDP**: School disciplinary practices; **SSC**: Student consultation practices; **TCMP**: Teachers' classroom management practices; **EXAC**: Extracurricular activities' quality and availability; **SHP**: School help practices; **PSC**: School-family collaboration mechanisms; **IDD-L1**: Time 1 depressive symptoms.

different variables, the decision was made to keep them separate in subsequent analyses. The selected strategy ensured that no problems of multicollinearity resulted from this decision[29].

Relationships Between School Life and Depression

School-Related Psychological Characteristics

Results from the regressions evaluating the effects of school-related psychological characteristics on Time 3 depressive symptoms are reported in Table IV.

Main effects. Results from the first set of regression analyses indicated that most of the variables studied did predict Time 3 depressive symptoms, even when previous levels of depressive symptoms were taken into account. In fact, only participants' levels of extracurricular involvement were found to be unrelated to depression development. These results indicate that students who exhibit higher levels of school misbehaviors or academic delays tend to present higher levels of depressive symptoms at Time 3, whereas students with higher levels of academic achievement, involvement, and self-efficacy tend to present lower levels of depressive symptoms at Time 3. However, when these variables were considered simultaneously in the analyses, only academic self-efficacy and school misbehaviors remained significant predictors of Time 3 depressive symptoms. Furthermore, when controls were partialled out in the analyses, only participants' levels of school misbehaviors still predicted Time 3 depressive symptoms.

Moderating role of gender. Among all the gender-based interactions evaluated, only the school misbehaviors (ß = 0.083; t= 2.519; p = 0.012) and academic involvement (ß = -0.078; t= -2.167; p = 0.030) interactions appeared significant. The decomposition of these interactions indicates that participants' levels of school

[29] For each regression performed in the present study, colinearity indicators were found to be in the acceptable range: none of the variance inflation indicators exceeded 5, none of the tolerance indicators was under 0.2, and none of the condition indexes exceeded 30 (Tabashnick & Fidell, 1996).

Table IV

Relationships between school-related psychological characteristics and Time 3 depressive symptoms

	Univariate effects, depression control			Multivariate effects, depression control			Multivariate effects, all controls			Final model for the dimension, all controls		
	Beta	t	p	Beta	t	p	Beta	t	p	Beta	t	p
School-related psychological characteristics												
Academic delay	0.073	2.807	0.005	0.010	0.352	0.725						
Academic achievement	-0.057	-2.199	0.028	0.020	0.709	0.479						
Academic involvement	-0.105	-4.053	0.000	-0.028	-0.956	0.339						
Academic self-efficacy	-0.101	-3.916	0.000	-0.061	-2.162	0.031	-0.029	-1.091	0.276			
Extracurricular involvement	-0.050	-1.909	0.056									
School misbehaviors	0.170	6.621	0.000	0.145	4.837	0.000	0.124	4.706	0.000	0.130	5.047	0.000
R^2 change			NA			0.033			0.016			0.016

misbehaviors represent a more potent predictor of depression development for girls (a = 1.978; b = 0.226; p = 0.000) than for boys (a = 1.658; b = 0.111; p = 0.000), while academic involvement predicted Time 3 depressive symptoms among girls only (girls: a = 1.954; b = -0.733; p = 0.000; boys: a = 1.678; b = -0.252; p = 0.099).

Moderating role of Time 1 depressive symptoms. Among the depression-based interactions, only the effects of participants' levels of extracurricular involvement were moderated by Time 1 depressive symptoms (ß = -0.090; t= -3.471; p = 0.001). The decomposition of this interaction revealed that participants' levels of extracurricular involvement did not predict depressive symptom development among asymptomatic (a = 0.527; b = 0.141; p = 0.161) and symptomatic (a = 1.613; b = -0.082; p = 0.216) participants but were negatively related to Time 3 depressive symptoms among previously clinical students (a = 2.698; b = -0.305; p = 0.000).

Final model for the dimension. When students' levels of school misbehaviors were entered alone, following controls, in a regression analysis to predict Time 3 depression symptoms, they explained 1.6% of depressive symptoms variance. Adding both of the preceeding significant interaction terms (i.e., gender * school misbehaviors, gender * academic involvement, and depression * extracurricular involvement) to this regression explained an additional 1% of Time 3 depressive symptoms variance. However, among the interactions, only the Time 1 depressive symptoms by extracurricular involvement interaction remained significant (ß = -0.074; t= -2.978; p = 0.003).

School-Related Socialization Experiences

Results from the regression analyses evaluating the effects of student's school-related socialization experiences on Time 3 depressive symptoms are reported in Table V.

Main effects of parental school-related educative practices. Results from the first set of regression analyses indicate that parental academic support and pressure both predicted Time 3 depressive symptoms, even when previous levels of depressive symptoms are taken into account. More precisely, students exposed to a higher level

Table V

Relationships between school-related socialization experiences and Time 3 depressive symptoms

	Univariate effects, depression control			Multivariate, depression control			Multivariate effects, all controls			Final model for the dimension, all controls		
	Beta	t	p	Beta	t	p	Beta	t	p	Beta	t	p
Parental school-related educative practices												
Parental academic support	-0.059	-2.253	0.024	-0.049	-1.838	0.066						
Parental academic pressure	0.064	2.452	0.014	0.055	2.077	0.038	0.034	1.321	0.187			
R^2 change			NA			0.006			0.001			
In-school peer relationships												
Transitional difficulties	0.089	3.380	0.001	0.015	0.502	0.615						
Loneliness at school (inv.)	0.107	4.073	0.000	-0.064	-2.063	0.039	-0.073	-2.653	0.008	-0.084	-3.235	0.001
Peer-related daily hassles	0.151	5.770	0.000	0.086	3.040	0.002	0.049	1.763	0.078			
Friends' school adaptation	-0.098	-3.779	0.000	-0.063	-2.417	0.016	-0.044	-1.684	0.092			
Minor victimization	0.176	6.800	0.000	0.134	4.672	0.000	0.109	4.010	0.000	0.106	4.010	0.000
Major victimization	0.090	3.470	0.001	-0.026	-0.797	0.425						
Sexual victimization	0.098	3.807	0.000	0.065	2.083	0.037	0.033	1.283	0.200			
R^2 change			NA			0.055			0.029			
Socialization experiences involving school adults												
School-related daily hassles	0.183	6.905	0.000	0.148	5.170	0.000	0.093	3.168	0.002	0.074	2.542	0.011
Teacher support	-0.006	-0.222	0.824									
Conflicts with teachers	0.141	5.449	0.000	0.084	3.011	0.003	0.066	2.384	0.017	0.059	2.116	0.035
Dissatisfaction: help	0.019	0.721	0.471									
Dissatisfaction: encouragement	0.027	1.017	0.309									
Dissatisfaction: academic control	0.008	0.305	0.760									
Dissatisfaction: discipline	0.028	1.060	0.289									
R^2 change			NA			0.037			0.015			0.034

of parental academic pressure tended to present more symptoms of depression at Time 3, while those who received higher levels of parental academic support tended to present fewer symptoms. However, when both of these variables were entered together in the analyses, only parental academic pressure remained a significant predictor of depressive symptoms. Finally, following the inclusion of additional controls in the analyses, the effects of parental academic pressure on depression also became non-significant.

Main effects of in-school peer relationships. In the first set of analyses, most aspects of participants' in-school peer relationships predicted Time 3 depressive symptoms. More precisely, these results indicate that students exposed to higher levels of transitional problems, loneliness at school (inversed), peer-related daily hassles, and minor, major, and sexual victimization tended to present more depressive symptoms at Time 3, while students whose friends presented higher levels of adaptation to school exhibited less symptoms. In the next set of regressions, the simultaneous consideration of these variables in the analysis resulted in the disappearance of two of the previously identified effects: transitional problems and major victimization became non-significant predictors of Time 3 depressive symptoms. Finally, only two variables still predicted Time 3 depressive symptoms following the inclusion of background controls in the analyses: loneliness at school (inversed) and minor victimization.

Main effects of socialization experiences involving school adults. Among the varied dimensions of participants' socialization experiences involving school adults, only two were found to significantly predict Time 3 depressive symptoms: school-related daily hassles and conflictual relationships with teachers. Students who perceived their school experiences as more stressful and who had more conflictual relationships with their teachers tended to present higher levels of depressive symptoms at the end of the study. Furthermore, these relationships were unaffected by the simultaneous consideration of both variables and by the inclusion of background controls in the analysis.

Moderating role of gender. The evaluation of gender-based interactions revealed that the effects of four aspects of students' school-related socialization experiences on Time 3 levels of depressive symptoms were moderated by gender: minor victimization (ß = 0.132; t= 4.052; p = 0.000), school-related daily hassles (ß = 0.133; t= 4.011; p = 0.000), conflictual relationships with teachers (ß = 0.092; t= 2.704; p = 0.007), and dissatisfaction with school disciplinary control (ß = 0.110; t= 3.218; p = 0.001). Additionally, the interaction between gender and major victimization was also found to be marginally significant (ß = 0.055; t= 1.775; p = 0.076). The decomposition of these interactions revealed that the first three variables, as well as major victimization, represented more potent predictors of depressive symptoms in girls (minor victimization: a = 1.990; b = 0.257; p = 0.000; major victimization: a = 1.962; b = 0.216; p = 0.000; daily hassles: a = 1.909; b = 1.212; p = 0.000; conflicts with teachers: a = 1.950; b = 0.592; p = 0.000) than in boys (minor victimization: a = 1.663; b = 0.084; p = 0.002; major victimization: a = 1.673; b = 0.087; p = 0.025; daily hassles: a = 1.683; b = 0.395; p = 0.003; conflicts with teachers: a = 1.675; b = 0.228; p = 0.011), while students' dissatisfaction with school disciplinary control predicted Time 3 depressive symptoms only among girls (girls: a = 1.905; b = 0.684; p = 0.002; boys: a = 1.700; b = -0.228; p = 0.219).

Moderating role of Time 1 depressive symptoms. Time 1 depressive symptoms were found to moderate the impact of several variables on depression development: transitional difficulties (ß = 0.065; t= 2.494; p = 0.013), minor victimization (ß = 0.062; t= 2.393; p = 0.017), major victimization (ß = -0.084; t= -3.236; p = 0.001), conflictual relationships with teachers (ß = 0.069; t= 2.672; p = 0.008), and dissatisfaction with school encouragement practices (ß = 0.059; t= 2.273; p = 0.023). Additionally, the interaction between Time 1 depressive symptoms and sexual victimization was also found to be marginally significant (ß = -0.048; t= -1.839; p = 0.066). The decomposition of these interactions revealed that experiencing transitional difficulties and conflictual relationships with teachers predicted Time 3 depressive symptoms only among previously symptomatic (transitional difficulties: a

= 1.595; b = 0.242; p = 0.013; conflict: a = 1.599; b = 0.317; p = 0.000) and clinically depressed (transitional difficulties: a = 2.641; b = 0.466; p = 0.000; conflict: a = 2.626; b = 0.487; p = 0.000) participants rather than among previously asymptomatic students (transitional difficulties: a = 0.548; b = 0.017; p = 0.910; conflict: a = 0.571; b = 0.146; p = 0.172). Similarly, exposure to minor victimization became more strongly associated with Time 3 depressive symptoms as participants' Time 1 symptoms increased (asymptomatic: a = 0.592; b = 0.080; p = 0.020; symptomatic: a = 1.602; b = 0.128; p = 0.000; clinical: a = 2.611; b = 0.176; p = 0.000) and students' levels of dissatisfaction with school encouragement practices only appeared to predict depressive symptoms among previously clinically depressed adolescents (asymptomatic: a = 0.513; b = -0.253; p = 0.311; symptomatic: a = 1.600; b = 0.116; p = 0.484; clinical: a = 2.688; b = 0.488; p = 0.025). Finally, the effects of major (asymptomatic: a = 0.535; b = 0.247; p = 0.000; symptomatic: a = 1.620; b = 0.142; p = 0.000; clinical: a = 2.704; b = 0.037; p = 0.350) and sexual (asymptomatic: a = 0.528; b = 0.356; p = 0.000; symptomatic: a = 1.613; b = 0.236; p = 0.000; clinical: a = 2.698; b = 0.116; p = 0.162) victimization on Time 3 depressive symptoms were limited to previously asymptomatic and symptomatic students, while they were non-significant among previously asymptomatic youths.

Final model for the dimension. Four variables related to students' school-related socialization experiences were found to significantly and positively predict Time 3 depressive symptoms: loneliness at school (inversed), minor victimization, school-related daily hassles and conflictual relationships with teachers. When these four variables were entered together in a multiple regression analysis, following controls, all remained significant predictors of depression development and explained 3.4% of Time 3 depressive symptom variance. Adding the significant interaction terms from the school-related socialization experience dimension[30] to this regression explained an additional 3.1% of Time 3 depressive symptom variance. Among the previously

[30] Five of these interactions involve gender: minor and major victimization, school-related daily hassles, conflictual relationships with teachers, and dissatisfaction with school disciplinary control. The other six interactions involve Time 1 levels of depressive symptoms: transitional difficulties, minor, major and sexual victimization, conflictual relationships with teachers, and dissatisfaction with school encouragement practices.

described interactions, six remained significant predictors of Time 3 depressive symptoms in this final model. Three of these interactions involved gender: minor victimization (ß = 0.076; t= 2.143; p = 0.032), school-related daily hassles (ß = 0.075; t= 2.064; p = 0.039), and dissatisfaction with school disciplinary control (ß =0.069; t= 2.012; p = 0.044). The other three interactions involved Time 1 levels of depressive symptoms: minor victimization (ß = 0.074; t= 2.628; p = 0.009), major victimization (ß = -0.086; t= -2.890; p = 0.004), and conflictual relationships with teachers (ß = 0.071; t= 2.700; p = 0.007).

Perceived School Environment

Results from the regression analyses evaluating the effects of student's perceptions of their school environments on Time 3 depressive symptoms are reported in Table VI.

Main effects of perceived school climate. In the first set of regressions, the six aspects of students' school climate perceptions were found to share significant negative relationships with Time 3 depressive symptoms. In the next set of regressions, the simultaneous consideration of these variables in the analysis resulted in the disappearance of four of these effects. Indeed, only students' perceptions of school justice and security climates remained significant predictors of depression development. The inclusion of background controls in the analysis did not change these results.

Main effects of perceived school problems. In the first set of regression analyses, students' perceptions of the frequency of minor violence, major violence and school-related problems at school were found to share a positive relationship with Time 3 depressive symptoms. When these dimensions were considered together in the analyses, only participants' perceptions of the frequency of minor violence problems remained a significant predictor of their later levels of depressive symptoms. This relationship remained unaffected by the inclusion of additional controls in the analyses.

Table VI
Relationships between perceived school environment and depression development

	Univariate effects with depression control			Multivariate effects (sub-dimensions), depression control			Multivariate effects (sub-dimensions), all controls			Final model for the dimension, all controls		
	Beta	t	p	Beta	t	p	Beta	t	p	Beta	t	p
Perceived school climate												
Bonding	-0.097	-3.726	0.000	-0.021	-0.582	0.561						
Relational (inter-student)	-0.102	-3.930	0.000	-0.050	-1.526	0.127						
Relational (teacher-student)	-0.100	-3.865	0.000	-0.004	-0.104	0.917						
Educational	-0.093	-3.575	0.000	0.004	0.104	0.917						
Justice	-0.115	-4.471	0.000	-0.076	-2.527	0.012	-0.074	-2.870	0.004	-0.039	-1.282	0.200
Security	-0.118	-4.584	0.000	-0.083	-3.025	0.003	-0.068	-2.579	0.010	-0.044	-1.588	0.113
R^2 change			NA			0.026			0.012			
Perceived frequency of school problems												
Minor violence	0.133	5.145	0.000	0.119	3.033	0.002	0.090	3.503	0.000	0.057	2.103	0.036
Major violence	0.092	3.538	0.000	0.015	0.463	0.643						
School-related	0.097	3.748	0.000	0.007	0.198	0.843						
R^2 change			NA			0.018			0.008			
Perceived quality of school practices												
Disciplinary practices	-0.110	-4.232	0.000	-0.064	-2.091	0.037	-0.053	-1.931	0.054	-0.040	-1.401	0.162
Student consultation	-0.056	-2.167	0.030	-0.020	-0.721	0.471						
Classroom management	-0.127	-4.931	0.000	-0.096	-3.098	0.002	-0.065	-2.343	0.019	-0.023	-0.737	0.461
Extracurricular activities	-0.060	-2.314	0.021	0.010	0.326	0.744						
Support mechanisms	-0.096	-3.700	0.000	-0.040	-1.329	0.184						
School-family collaboration	-0.063	-2.425	0.015	0.023	0.700	0.484						
R^2 change			NA			0.022			0.010			0.016

Main effects of perceived school practices. When they were considered alone in analyses in which Time 1 depressive symptoms where controlled, the six aspects of students' perceptions of school practices negatively predicted their levels of depressive symptoms at the end of the school year. The simultaneous inclusion of these variables in the analyses considerably reduced the number of statistically significant effects. Indeed, only students' perceptions of school disciplinary practices and teachers' classroom management practices still predicted Time 3 depressive symptoms. Moreover, the effects of school disciplinary practices became only marginally significant following the inclusion of additional controls in the analyses.

Moderating role of gender. The evaluation of gender-based interactions revealed that the effects of four aspects of students' perceptions of their school environment were significantly moderated by gender: security climate (ß = -0.085; t= -2.482; p = 0.013), minor violence problems (ß = 0.127; t= 3.584; p = 0.000), major violence problems (ß = 0.097; t= 2.843; p = 0.005), and school-related problems (ß = 0.109; t= 3.070; p = 0.002). The decomposition of these interactions indicated that these four variables predicted Time 3 depressive symptoms only, or mostly, among girls (security climate: a = 1.955; b = -0.632; p = 0.000; minor violence: a = 1.922; b = 0.114; p = 0.000; major violence: a = 1.936; b = 0.176; p = 0.000; school-related: a = 1.890; b = 0.091; p = 0.000) rather than boys (security climate: a = 1.678; b = -0.221; p = 0.041; minor violence: a = 1.690; b = 0.024; p = 0.178; major violence: a = 1.691; b = 0.032; p = 0.340; school-related: a = 1.698; b = 0.011; p = 0.535).

Moderating role of Time 1 depressive symptoms. The evaluation of Time 1 depressive-symptom-based interactions revealed that the effects of several aspects of students' perceptions of their school environment on Time 3 depressive symptoms were moderated by their previous levels of depression: bonding climate (ß = -0.091; t= -3.535; p = 0.000), inter-student relational climate (ß =- 0.063; t= -2.426; p = 0.015), justice climate (ß = -0.087; t= -3.362; p = 0.001), minor violence problem frequency (ß = 0.059; t= 2.275; p = 0.023), school disciplinary practices (ß =-0.069; t= -2.691; p = 0.007), student consultation practices (ß = -0.062; t= -2.382; p =

0.017), teachers' classroom management practices (ß = -0.070; t= -2.719; p = 0.007), extracurricular activity quality and availability (ß = -0.062; t= -2.376; p = 0.018), and school-family collaboration mechanisms (ß = -0.079; t= -3.035; p = 0.002). The decomposition of these interactions revealed that the effects of three of these variables were related to Time 3 depressive symptoms only, or mostly, among students who were clinically depressed at Time 1: student consultation practices (asymptomatic: a = 0.534; b = 0.036; p = 0.711; symptomatic: a = 1.605; b = -0.119; p = 0.070; clinical: a =2.676; b = -0.273; p = 0.001), extracurricular activity quality and availability (asymptomatic: a = 0.530; b = 0.043; p = 0.743; symptomatic: a = 1.605; b = -0.156; p = 0.074; clinical: a = 2.681; b = -0.355; p = 0.001), and school-family collaboration mechanisms (asymptomatic: a = 0.539; b = 0.096; p = 0.506; symptomatic: a = 1.609; b = -0.187; p = 0.053; clinical: a = 2.668; b = -0.471; p = 0.000). Similarly, the predictive effects of six other variables were limited to symptomatic and clinically depressed students at Time 1: bonding climate (asymptomatic: a = 0.548; b = 0.029; p = 0.828; symptomatic: a = 1.603; b = -0.285; p = 0.000; clinical: a = 2.649; b = -0.598; p = 0.000), inter-student relational climate (asymptomatic: a =0.552; b = -0.123; p = 0.430; symptomatic: a = 1.603; b = -0.367; p = 0.000; clinical: a = 2.655; b = -0.611; p = 0.000), justice climate (asymptomatic: a = 0.548; b = -0.050; p = 0.687; symptomatic: a = 1.606; b = -0.316; p = 0.000; clinical: a = 2.665; b = -0.582; p = 0.000), minor violence problem frequency (asymptomatic: a = 0.557; b = 0.032; p = 0.104; symptomatic: a = 1.604; b = 0.060; p = 0.000; clinical: a = 2.650; b = 0.088; p = 0.000), school disciplinary practices (asymptomatic: a = 0.540; b = -0.149; p = 0.468; symptomatic: a = 1.603; b = -0.509; p = 0.000; clinical: a = 2.667; b = -0.868; p = 0.000), and teachers' classroom management practices (asymptomatic: a = 0.551; b = -0.196; p = 0.245; symptomatic: a = 1.604; b = - 0.488; p = 0.000; clinical: a = 2.657; b = -0.781; p = 0.000).

Final model for the dimension. Five variables related to students' perceptions of their school environment were found to significantly predict Time 3 depressive symptoms: justice and security climates, perceived frequency of minor violence

problems, disciplinary practices, and teachers' classroom management practices. When these five variables were entered together in a multiple regression analysis, following controls, they explained a total of 1.6% in Time 3 depressive symptom variance. However, only perceived frequency of minor violence problems remained a significant predictor of depression development. When this regression was replicated including only this variable and background controls, perceived frequency of minor violence problems explained only 0.8% of the variance in Time 3 depressive symptoms. Adding the significant interaction terms from the perceived school environment dimension[31] to this regression explained an additional 2.6% of Time 3 depressive symptom variance, although none of the interactions terms remained significant.

Contribution of School Life to Depression Development

A final regression analysis was conducted to estimate the total contribution, in terms of percentage of explained variance, of school life to depression development. When all significant predictors from the previous analyses (final models from each of the three dimensions) were entered together following controls, they explained a total of 4.1% of Time 3 depressive symptom variance. In this regression, Time 1 depressive symptoms and background controls explained 21.2% and 6.0% respectively of Time 3 depressive symptom variance. Among the predictors entered, four remained significant predictors of Time 3 depressive symptoms: school misbehaviors (ß = 0.091; t= 3.199; p = 0.001), loneliness at school (inversed: ß = -0.093; t= -3.579; p = 0.000), minor victimization (ß = 0.089; t= 3.260; p = 0.001), and school-related hassles (ß = 0.060; t= 2.061; p = 0.040). The effect of the other two variables – conflictual relationships with teachers (ß = 0.027; t= 0.919; p = 0.358) and perceived frequency of minor violence problems (ß = 0.021; t= 0.756; p = 0.450) – thus became

[31] Four of these interactions involve gender: security climate and perceived frequency of minor violence, major violence, and school-related problems. The other nine interactions involve Time 1 levels of depressive symptoms: bonding, relational (inter-students), and justice climates, perceived frequency of minor violence problems, school disciplinary practices, student consultation practices, teachers' classroom management practices, extracurricular activity quality and availability, and school-family collaboration mechanisms.

non-significant. When this regression was replicated including only the four significant predictors and background controls, they were found to explain 4% of the Time 3 depressive symptom variance. Adding the significant interaction terms from the preceding analyses to this regression explained an additional 3.4% of Time 3 depressive symptom variance. The final model thus explained a grand total of 34.6% of Time 3 depressive symptom variance. Among the interactions included, six remained significant predictors of Time 3 depressive symptoms in this final model. Two of these interactions involved gender: school-related daily hassles (ß = 0.069; t= 2.070; p = 0.039) and minor victimization (ß =0.066; t= 2.078; p = 0.044). The other four interactions involved Time 1 levels of depressive symptoms: extracurricular involvement (ß = -0.061; t= -2.508; p = 0.012), minor victimization (ß = 0.080; t= 3.013; p = 0.003); major victimization (ß = -0.093; t= -3.569; p = 0.000), and conflictual relationships with teachers (ß = 0.056; t= 2.236; p = 0.026). Only the previous levels of depression * dissatisfaction with school disciplinary control interaction became non-significant in this final model (ß = 0.058; t= 1.763; p = 0.078).

DISCUSSION

Relationships Between School Life and Depression

The main objective of this study was to evaluate the nature of the relationship between school life and depression development. A summary of the results obtained in this study is presented in Table VII. Perhaps the most important of these results is that the majority of the school life characteristics evaluated did represent significant predictors of depression development among high school students when their effects were considered separately. The fact that many of these factors became non-significant predictors of depression development when they were considered simultaneously does not mean that they should not be targeted in preventive interventions. Indeed, this study suggests that improving various aspects of students'

Table VII
A summary of the results

	Main effects					Gender-based variations				Depression-based variations				
	U_CD	M_CD	M_AC	FMD	FM	M	F	FMD	FM	A	S	C	FMD	FM
School-related psychological characteristics														
Academic delay	↑	---	---	---	---									
Academic achievement	↓	---	---	---	---									
Academic involvement	↓	---	---	---	---	---	↓	---	---					
Academic self-efficacy	↓	↓	---	---	---									
Extracurricular involvement	---	---	---	---	---					---	---	↓	↓	↓
School misbehaviors	↑	↑	↑	↑	↑	↑	↑↑	---	---					
School-related socialization experiences : *Parental school-related educative practices*														
Parental academic support	↓	---	---	---	---									
Parental academic pressure	↑	↑	---	---	---									
School-related socialization experiences : *In-school peer relationships*														
Transitional difficulties	↑	---	---	---	---					---	↑	↑↑	---	---
Loneliness at school	↑	↑	↑	↑	↑									
Peer-related daily hassles	↑	↑	---	---	---									
Friends' school adaptation	↓	↓	---	---	---									
Minor victimization	↑	↑	↑	↑	↑	↑	↑↑	↑	↑	↑	↑↑	↑↑↑	↑	↑
Major victimization	↑	---	---	---	---	↑	↑↑	---	---	↑↑	↑	---	↑	↑
Sexual victimization	↑	↑	---	---	---					↑↑	↑	---	---	---
School-related socialization experiences : *Socialization experiences involving school adults*														
School-related hassles	↑	↑	↑	↑	↑	↑	↑↑	↑	↑					
Teacher support	---	---	---	---	---									
Conflicts with teachers	↑	↑	↑	↑	---	↑	↑↑	---	---	---	↑	↑↑	↑	↑
Dissatisfaction: help	---	---	---	---	---									
Dissatisfaction: encour.	---	---	---	---	---					---	---	↑	---	---
Dissatisfaction: acad. control	---	---	---	---	---									
Dissatisfaction: discipline	---	---	---	---	---	---	↑	↑	↑					

Table VI (continued)

	Main effects					Gender-based interactions				Depression-based interactions				
	S_CD	M_CD	M_AC	FMD	FM	M	F	FMD	FM	A	S	C	FMD	FM
***Perceived school environment**: Perceived school climate*														
Bonding	↓	---	---	---	---					---	↓	↓↓	---	---
Relational (students)	↓	---	---	---	---					---	↓	↓↓	---	---
Relational (teachers-students)	↓	---	---	---	---									
Educational	↓	---	---	---	---									
Justice	↓	↓	↓	---	---					---	↓	↓↓	---	---
Security	↓	↓	↓	---	---	↓	↓↓	---	---					
***Perceived school environment**: Perceived frequency of school problems*														
Minor violence	↑	↑	↑	↑	---	---	↑	---	---	---	↑	↑↑	---	---
Major violence	↑	---	---	---	---	---	↑	---	---					
School-related	↑	---	---	---	---	---	↑	---	---					
***Perceived school environment**: Perceived quality of school practices*														
Disciplinary practices	↓	↑	↑	---	---					---	↓	↓↓	---	---
Students' involvement	↓	---	---	---	---					---	↓	↓	---	---
Classroom management	↓	↑	↑	---	---					---	↓	↓↓	---	---
Extracurricular activities	↓	---	---	---	---					---	↓	↓	---	---
School support	↓	---	---	---	---									
Parental implication	↓	---	---	---	---					---	↓	↓	---	---

Legend. U_CD: univariate (U) effect of the variable on Time 3 level of depression while controlling (C) for previous levels of depression (D); **M_CD**: effect of the variable on Time 3 level of depression in a multivariate analysis including all predictors from the dimensions and controlling (C) for previous levels of depression (D); **M_AC**: effect of the variable on Time 3 level of depression in a multivariate analysis including all predictors from the dimensions including all (A) controls (C); **FMD**: effect of the variable in the final (F) model (M) for the dimension (D); **FM**: effect of the variable in the final (F) model (M); **M**: effect of the variable among males; **F**: effect of the variable among females; **A**: effect of the variable among asymptomatic subjects at Time 1; **S**: effect of the variable among symptomatic subjects at Time 1; **C**: effect of the variable among clinically depressed subjects at Time 1; ↑: positive significant relationship (higher levels of the predictor are associated with higher levels of depression); ↓: negative significant relationship (lower levels of the predictor are associated with higher levels of depression); ↓ or ↑: marginally significant effect; ↑, ↑↑ and ↑↑↑ (or reversed): comparative importance of the effects; ---: non significant effect.

school-related psychological characteristics, in-school socialization experiences and perceptions of their school environment would likely reduce their risk of developing depression following high school transition.

However, the simultaneous consideration of multiple aspects of students' school life in the prediction of depression development clearly indicated that some of these variables may be more potent predictors of depression development than others. This result may be due to the interrelated character of the multiple facets of school life. For instance, whereas most aspects of students' perceptions of school climate individually predicted their later levels of depressive symptoms, the simultaneous consideration of these aspects in the analyses left only justice and security climates as significant predictors of depression development. A parsimonious interpretation of this result could be that the effects of the other aspects of school climate (inter-student and teacher-student relational climates, bonding climate, and educational climate) on students' levels of depression only represented an artifact of their relationships with justice and security climates. Again, this result does not mean that improving, for example, school bonding climates would not help to prevent depression development since such an improvement would likely result in the simultaneous enhancement of school justice and security climates. However, more systematic evaluations of the relationships between school life characteristics would be needed to test the plausibility of this hypothesis.

Alternatively, the fact that most aspects of school life appear to be worthy targets for preventive interventions does not mean that they also provide worthy explanations for depression development. Indeed, in the preceding example, most aspects of students' perceptions of school climates were described as potential targets for prevention programs due to their intertwined character. Nevertheless, only justice and security climates represent potential "causes" of depression development as the results suggest that, no matter how problematic other aspects of school climate may be viewed by students, theses perceptions may not increase students' risk of developing depression if they perceive their schools' justice and security climates positively.

Another important result from this study is the fact that the inclusion of students' personal and social background characteristics as controls in the analyses only minimally affected the observed relationships. Indeed, the effects of only five of the predictors of depressive symptoms identified became non-significant once these variables were controlled in the analyses: the relationships between depression development and students' levels of academic self-efficacy, parental academic pressure, perceived stressfulness of students' relationships with friends and classmates, friends' school adaptation, and sexual/romantic victimization may thus represent an artifact of students' background characteristics. More precisely, students' background characteristics may influence their exposure to these specific in-school factors as well as their risk of developing depression, and thus explain the observed statistical associations. However, a far more important implication of this finding is that school life effects on depression development may be relatively independent from the effects of students' lives outside of school. Youths' lives in and out of school should therefore be seen as complementary targets for preventive interventions rather than as mutually exclusive targets. The present results clearly suggest that neither form of intervention is likely to be sufficient to prevent depression.

Among all the school life characteristics evaluated, few may be seen as exerting a determining impact on depression development: students' levels of school misbehaviors, students' feelings of loneliness at school, students' exposure to minor victimization and to conflictual relationships with teachers, perceived stressfulness of students' school life, and students' negative perceptions of the quality of their schools' justice and security climates, of the quality of the disciplinary and classroom management practices used in their schools and of the frequency with which minor violence problems occur at their schools. Furthermore, in a more integrated predictive model, it appears that the main effects of school life on depression development may only result from the action of four variables: students' levels of school misbehaviors, students' feelings of loneliness at school, students' exposure to minor victimization, and perceived stressfulness of students' school lives. The disappearance of the other

effects should not be surprising since it is highly plausible that these remaining variables may act as complete mediators of the relationships between the other variables and depression development. For instance, students' negative perceptions of the quality of their schools' security climates, of the quality of the disciplinary and classroom management practices used in their schools, and of the frequency with which minor violence problems occur at their schools all refer to school violence or to school efforts to reduce violence. In this context, it is possible that these variables all converge to augment students' risk of being victimized, and that victimization represents the proximal determinant of depression development involved in their effects. In a related way, students' negative perceptions of their schools' justice climates and exposure to conflictual relationships with teachers may both represent potential contributors to students' perceptions of their schools as stressful places, which may in turn mediate their effects.

Some of these results are highly consistent with those from previous studies. For instance, previous studies also generally failed to find significant relationships between depression development and academic achievement (Bandura *et al.*, 1999; Cole *et al.*, 1996; Lewinsohn *et al.*, 1994; Reinherz *et al.*, 1993), parental school-related educative practices (Hilsman, & Garber, 1995; Lewinsohn *et al.*, 1994), and educative climate (Kasen *et al.*, 1990; Roeser & Eccles, 1998; Roeser, Eccles, & Sameroff, 1998). Previous studies similarly found that students frequently exhibiting school misbehaviors (Austin, & Joseph, 1996; Kaltiala-Heino *et al.*, 1999, 2000; Lewinsohn *et al.*, 1994; Nansel *et al.*, 2001), bullied and/or lonely students (Austin & Joseph, 1996; Hodges & Perry, 1999; Kaltiala-Heino *et al.*, 1999, 2000; Kiesner, 2002; Reinherz *et al.*, 1993; Stein *et al.*, 1996), students feeling more stressed by their school environment (Siddique & D'Arcy, 1984; Turner & Cole, 1994), students with more negative perceptions of their school justice climate (Resnick *et al.*, 1997; Roeser, Eccles, & Sameroff, 1998), and students negatively evaluating the disciplinary and classroom management practices used in their schools (Eccles *et al.*, 1997; Roeser & Eccles, 1998) presented a higher risk of developing depression.

In some cases, our results may appear inconsistent with those found in previous studies. For instance, levels of academic self-efficacy (Bandura *et al.*, 1999; Hilsman & Garber, 1995; Lewinsohn *et al.*, 1994), involvement in extracurricular activities (Gore *et al.*, 2001; Mahoney *et al.*, 2002), students experiencing stressful peer relationships (Brendgen *et al.*, 2001; Jaffe *et al.*, 2002), affiliation with peers presenting high levels of school adaptation problems (Cantin *et al.*, 2002) and supportive teacher-student relationships (Roeser and Eccles, 1998; Sim, 2002) were generally found to predict depression development in previous studies. However, it should be noted that this study represents, to our knowledge, the first attempt to systematically evaluate the impact of the different facets of school life in a single study while providing adequate controls for intake of personal, familial, and friendship characteristics. Since it is well known that personal and familial characteristics exert an impact on an individual's exposure to specific environmental characteristics and on the choice of specific school environments and peer groups, it is highly possible that some of the previously found effects of school life dimensions were in reality only an artifact of the lack of control of all relevant variables in the analyses (Mortimore, 1995; Rutter, 1999). Moreover, due to the intertwined character of school life characteristics, it is also possible that some of the effects found in previous studies reflect their general failure to simultaneously consider the full reality of school life. The analytical strategy used in this study allowed us to partially confirm these hypotheses. Indeed, whereas most of the aspects of school life studied were found to predict depression development when they were considered alone in the analyses, many of these effects disappeared altogether when other aspects of school life were entered in the analyses and/or when background characteristics were controlled.

Finally, two more specific results are worth considering. First, many of the most significant predictors identified in the present study refer, directly or indirectly, to school violence (e.g., school misbehaviors, security climate, minor victimization, minor violence problem frequency) or school efforts to reduce violence (e.g., justice climate, disciplinary practices, classroom management practices). This result lends

strong support to the recent societal and scientific claims that school violence prevention programs should be seen as a key priority for modern societies (Gottfredson, 2000; Gottfredson & Gottfredson, 1985). Second, the effects of three forms of victimization on depression development were evaluated. Whereas no relationships were observed between the most severe forms of victimization (major or sexual violence) and depression development, one of the strongest effects found in this study indicates that repeated exposure to minor forms of violence represents a very robust predictor of depressive symptoms. This finding is highly consistent with current victimization theories which state that it is the daily life of victims and the daily pecking they are subjected to rather than the few severe assaults to which they are exposed which would likely exert the most deleterious effects on their development (Cousineau, Gravel, Laverge, & Wemmers, 2003).

Moderating Role of Gender : Differentiated Impact of School Life on Boys and Girls

The fact that, beginning in early adolescence, girls present higher rates of depression than boys is a well-documented phenomenon in developmental research (Bebbington, 1996l Cyranowski *et al.*, 2000; Nolen-Hoeksema, 2002). At the theoretical level, a plausible explanation for this result invokes the fact that, due to their earlier pubertal maturation, girls tend to enter adolescence in a state of biopsychosocial dysregulation (Cyranowski *et al.*, 2000). Puberty indeed represents a highly challenging experience for adolescents and may become even more challenging when it occurs simultaneously with other developmental transitions. Due to their earlier pubertal maturation, girls often tend to simultaneously experience pubertal changes and high school transition (Bebbington, 1996). They may therefore be more severely affected than boys by any form of school-related stress and benefit more from school-based support mechanisms. In the present study, we sought to evaluate whether school life did indeed represent a more significant predictor of girls', rather than boys', depression development. Interestingly, many results appear to support this hypothesis. For instance, many school life characteristics were found to predict depression development among girls only: low levels of academic involvement,

dissatisfaction with school disciplinary control mechanisms, and the perceived frequency of various forms of school problems (minor violence, major violence, and school-related problems). Other factors, such as school misbehaviors, minor and major victimization, perceived school life stressfulness, conflictual relations with teachers, and security climate perceptions appeared to represent stronger predictors of depression development among girls than among boys. Although it remains to be seen whether this differential impact may explain the gender differences in depression prevalence rates, the present results support the plausibility of this hypothesis.

Moderating Role of Previous Depressive Symptoms : Prevention or Intervention Targets?

Following Kessler's (1997) suggestion, the present study also sought to determine whether school life exerted a differential impact on the emergence or aggravation of depressive symptoms. Again, strong evidence was found in favor of such a differentiated impact. Indeed, many aspects of school life were found to represent stronger predictors of depression among previously symptomatic and clinically depressed students rather than among previously asymptomatic students: extracurricular involvement, transitional difficulties, minor victimization, conflictual relationships with teachers, dissatisfaction with school-based encouragement practices, bonding, relational (inter-student) and justice climates, and perceptions of the frequency of minor violence problems and of the quality of school disciplinary practices, student consultation mechanisms, classroom management practices, extracurricular activities, and school-family collaboration mechanisms. These results suggest that these aspects of school life would be worthy targets for school-based treatment programs. Conversely, students' exposure to major and sexual victimization appeared as stronger predictors of depressive symptoms among previously asymptomatic and symptomatic students, rather than clinically depressed ones.Both of these dimensions would appear to be worthy targets for school-based prevention programs. Interestingly, these results clearly appear to sustain a dimensional view of depression.

Total Contribution of School Life to Depression Development

In the final model, the most significant predictors of depression development, which were entered together after background controls, were found to explain a grand total of 7.4% of depressive symptom variance (previous levels of depressive symptoms explained 21.2% and background controls explained 6.0%). Three conclusions could be reached from this result. First, compared with the results from other studies, this percentage is quite low. For instance, Resnick *et al.* (1997) found that 13.1% to 17.6% of the variance in students' levels of depression could be statistically explained by the school-level factors measured in their study. As Resnick *et al.*'s (1997) study was based on a cross-sectional design, their results are hard to compare with the present ones. However, Roeser and Eccles' (1998) study lends support to Resnick *et al.*'s (1997) results. Indeed, these authors found that school life still explained 14% of depression levels once minimal controls (including previous levels of depression) were included in the analysis. Two reasons may explain this apparent discrepancy with the present results: Roeser and Eccles' (1998) study included only a very limited number of controls in the analyses and was based on a longer term follow-up of students. As we showed in this study, adding controls to the analyses diminished the predictive power of some variables. Moreover, it is also possible that, due to the longer follow-up used in their study, Roeser and Eccles (1998) were able to detect effects which were still unstable in the present study due to the recent school transition experienced by the participants. The fact that another one-year follow-up study, in which more than minimal controls were considered, found that school life explained only 3% of the variance in depressive symptoms, lends support to this interpretation (Kuperminc, Leadbeater & Blatt, 2001).

Second, school life was found to contribute as much as students' personal, familial and peer-related background characteristics to depression development. Regardless of the specific strength of this contribution, this result clearly lends support to the design of school-based prevention programs.

Third, significant interaction terms explained as much variance in depressive symptoms (3.4%) as the main effects of school life characteristics (4%). This result strongly suggests that school life effects differ according to students' characteristics and thus lends support to the need to rely more often on person-centered analyses in developmental research (Von Eye & Bergman, 2003). Person-centered analyses are rapidly gaining popularity in developmental research. The main reason for this popularity is that classical variable-centered approaches usually portray relations as they apply to the average individual. As this average individual seldom exists, person-centered approaches were designed to take into account individual variability regarding cross-sectional and longitudinal patterns of risk factor aggregation and symptomatic expressions (Bergman, 2000; Nagin, 1999; Von Eye & Bergman, 2003).

A Note on Controlled Variables

Many of the control variables evaluated were found to represent non-significant predictors of depression development following their simultaneous inclusion in the analyses. Moreover, while the percentage of variance in depressive symptoms explained by previous levels of depression (21.2%) is consistent with what is known about depression stability and continuity (Harrington & Dubicka, 2001; Kessler, 2002; Kessler *et al.*, 2001; Lewinsohn & Essau, 2002), background controls were found to explain only 6% of depressive symptom variance. In other studies, individual and familial characteristics were generally found to explain at least twice as much variance (e.g., Barrera *et al.*, 2002; Chase-Lansdale *et al.*, 1995; Duggal, Carlson, Sroufe, & Egeland, 2001; Ge *et al.*, 1996). Many reasons may explain these results. First, many of the variables that became non-significant represented familial characteristics that could already have been present before the onset of the study or were based on retrospective evaluations (past difficulties, stressful life events, and behavioral disorders). Accordingly, if these variables really represented significant predictors of depression, their predictive power could have been offset by the inclusion of Time 1 levels of depressive symptoms in the analyses, which may have themselves been influenced by these variables.

Second, a high level of intercorrelations was observed between Time 1 levels of neuroticism, anxiety, self-esteem and body image satisfaction (r = - 0.132 to 0.502). This observation, which suggests that these different variables may represent highly overlapping constructs, could explain the disappearance of the effects of neuroticism and body image satisfaction in a multivariate analysis. Previous results strongly support this hypothesis. Indeed, in an article combining a meta-analysis and three studies based on seven samples, Judge, Erez, Bono, and Thoresen (2002) found that self-esteem, neuroticism, locus of control and generalized self-efficacy were so interrelated as to be best represented as a single higher-order construct.

Third, the disappearance of the effects of age and pubertal development could easily be explained by a range restriction artifact. Indeed, the present study was based on a sample of seventh grade students having just experienced high school transition. Consequently, most students were of similar age and pubertal development status.

Fourth, the apparent absence of gender differences in rates of depression may seem harder to explain, given the well-documented character of these differences (Nolen-Hoeksema, 2002). However, the fact that the gender effect disappeared following the inclusion of Time 1 levels of depression in the analyses suggests that these differences could have already been present at the beginning of our study. Additional analyses in which age and gender were used to predict Time 1 depressive symptoms support this hypothesis (β = 0.163, t= 5.591, p = 0.000).

Fifth, it is also possible that the low predictive power of the control variables regarding depression development reflects the fact that school transition is associated with so many social transitions that it provides a window of opportunity for students to develop in ways that are increasingly independent from their own personal, familial, and friendship backgrounds (Rutter *et al.*, 1997). Only further studies will be able to provide clear answers to this question.

As the main objective of this study was not to evaluate the precise role of background characteristics in depression development, we did not seek further answers to these questions. The analytical strategy used in this study was designed to evaluate the contribution of school life to depression development and not to obtain a detailed evaluation of the impact of background characteristics, which were only seen as variables to be controlled in the most parsimonious manner. Alternate analytical strategies may yield different results.

Limits and Directions for Future Research

Although promising, the results from the present study are plagued by at least four important limitations which should be addressed in future studies. First, we did not conduct mediation analyses which could have helped to clarify the causal relationships implicated in the present results. For instance, whereas we found a relationship between the perceived quality of classroom management practices and depression development, the reason for this effect remains unclear. Indeed, classroom management practices could diminish the prevalence of conflictual teacher-student relationships or the frequency of students' school misbehaviors, which were both found to significantly predict depression development. However, the present analytical strategy did not allow us to distinguish whether the non-significant variables identified in this study really exerted no impact on depression development or whether their effects were completely meditated by other variables. For example, it remains possible that students exposed to higher levels of parental academic support and to lower levels of parental academic pressure would exhibit lower levels of school misbehaviors which in turn predict depression development. Hopefully, further studies will provide answers to these questions.

Second, while the present study tried to identify risk factors for adolescent depression development, no attempt was made to evaluate protective factors or other forms of moderating relationships between the different predictors. Thus, although we know that some factors do not predict depression development, we did not evaluate the possibilities that these factors could protect at-risk students from developing

depression or amplify their already elevated risk level. This possibility should be evaluated in further studies. Indeed, previous studies focusing on outcomes other than depression repeatedly found that school-related variables could play a protective role on at-risk students. For example, Fallu and Janosz (2003) found that whereas sharing warm and supportive relationships with teachers did not represent a significant risk factor for school adaptation problems, it did represent a very important protective factor for at-risk students.

Third, the present design did not allow us to evaluate the impact of aggregated and structural characteristics of students' school environments. Ideally, analyzing the impact of aggregated or structural school characteristics on students' development would require the use of multilevel statistical analysis (hierarchical linear modeling) to disentangle the effects of individuals' characteristics on depression development from the effect of generic characteristics of their school environment (Bryk & Raudenbush, 1992; Gottfredson, 2000). As the MADDP did not include a sufficient number of schools to conduct this kind of analysis with sufficient statistical power, this latest aspect of school life could not be considered in the present study. However, some previous results indicate that this bias may be smaller than it appears. Indeed, Roeger, Allison, Martin, Dadds, and Keeves (2001), using multilevel analyses, found that almost none of the variance in students' levels of depression (0.87%) could be explained by differences between the schools ($n = 25$).

Fourth, the research design used in the present study seriously limits the generalizability of the findings. Firstly, this part of the MADDP is based on a short term follow-up of students following high school transition, a period of known developmental instability. Thus, whether the present results can be generalized to the following grades remains unknown and should be evaluated in further studies. Hopefully, the design of the MADDP would allow us to answer this question as more years of data collection become available. Secondly, the present sample is far from representative of the North American population. Indeed, our desire to maximize the organizational differences between the schools selected for the present study led us to over-sample gifted or academically talented students. Moreover, many of the most

problematic students were lost through the attrition process. Fortunately, all subjects had the option to complete, on an in-and-out basis, each of our questionnaires. Consequently, although they were not used in the present analyses, some subjects did complete at least some of the questionnaires, including the last one. Complementary analyses in which pairwise case deletion procedures were used revealed that the attrition process did not induce systematic biases in the present results.

Finally, various measurement-related problems may also have affected the validity of the present results. For instance, given the number of measures included, each instrument had to be kept as short as possible. Consequently, the internal consistency of many of them was close to the lowest limit of the acceptability range (Nunnally & Bernstein, 1994). Although this limit most likely represents a statistical artifact of the succinctness of the instruments, the resulting lessening of the scales' reliability still could have affected some of the observed results. Additionally, all of the predictors and controls used in the present study were measured through the exclusive use of youths' self-reports. Accordingly, through a shared-method variance effect, the relationships among the various independent/control variables and between dependent and independent/control variables could have been artificially inflated. Fortunately, the fact that predictors and controls were evaluated at different measurement points somewhat diminishes the severity of this problem. Similarly, some important information regarding students' school lives may have been accessible only through the use of reports from classmates, peers, teachers, or parents and may thus have been missed in the present study. Lastly, the version of the IDD used to evaluate students' outcome levels of depression retrospectively evaluated participants' symptoms since the first measurement point. Although there is no doubt about either the retrospective validity of the IDD or the importance of estimating depression development through the use of such measures (see the methodological section), this methodological characteristic of the present study also means that Time 3 depressive symptoms encompass a time period which started before the evaluation of the predictors. Consequently, the present study should not be considered as a "real" prospective longitudinal one.

CONCLUSION

Notwithstanding these various limitations, the present study clearly illustrates the need to move beyond single variable designs and ritualistic hypothesis testing (see also Richters, 1997) in depression development research to better accommodate the full richness and complexity of human development. If the present results were to be replicated in designs built to answer the present limitations, they would strongly suggest that prevention and treatment programs for adolescent depression would do well to simultaneously consider background individual and familial risk factors in conjunction with factors directly related to adolescents' lives at school. Among these factors, school violence and loneliness appear to represent particularly valuable targets for such programs, although more global organizational development programs may also indirectly influence depression development through complex mediating relationships involving, among other factors, school violence and loneliness (see also Morin & Chalfoun, 2003). Additionally, the fact that school life appears to exert a stronger impact on girls, who also tend to present higher levels of depression than boys, suggests that schools may not be that adapted to girls after all and that similar factors may affect boys' development of conduct disorders and girls' levels of depressive symptoms. Consequently, programs designed to better students' school lives may potentially directly influence depression (girls) and delinquency (boys) and indirectly affect depression development through their impact on school levels of delinquency. More studies will be needed on this topic but the present results clearly provide stimulating hypotheses.

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APPENDICES

Appendix A

French adaptation of the Inventory to Diagnose Depression – Lifetime version

LA SEMAINE OÙ J'AI ÉTÉ LE PLUS DÉPRIMÉ CETTE ANNÉE

Lis attentivement chaque groupe de phrases. Choisis ensuite la phrase dans chaque groupe qui décrit le mieux la façon dont tu t'es senti(e) lors de <u>la semaine où tu as été le plus déprimé(e) depuis le début de l'année scolaire</u>.

(1)

a) Je ne me suis pas senti(e) triste ou déprimé(e).
b) Je me suis senti(e) triste ou abattu(e) de temps en temps.
c) Je me suis senti(e) triste la plupart du temps mais je pouvais y échapper.
d) Je me suis senti(e) triste tout le temps et je ne pouvais pas y échapper.
e) J'étais si triste et malheureux(se) que je ne pouvais pas le supporter.

(2)

a) Mon niveau d'énergie était normal.
b) Mon niveau d'énergie était de temps en temps un peu plus faible que la normale.
c) Je me fatiguais plus facilement ou j'avais moins d'énergie que d'habitude.
d) Je me fatiguais en faisant presque n'importe quoi.
e) Je me sentais fatigué(e) ou épuisé(e) presque tout le temps.

(3)

a) Je ne me suis pas senti(e) plus nerveux (se) et agité(e) que d'habitude.
b) Je me suis senti(e) un peu plus nerveux (se) ou agité(e) que d'habitude.
c) J'ai été très agité(e) et j'ai eu du mal à rester assis(e) tranquillement.
d) J'ai été extrêmement agité(e) et j'ai marché de long en large un petit peu, presque tous les jours.
e) J'ai marché de long en large plus d'une heure par jour et je n'ai pas pu m'asseoir tranquillement.

(4)

a) Je n'ai pas parlé ou bougé plus lentement que d'habitude.
b) J'ai parlé un peu plus lentement que d'habitude.
c) J'ai parlé un peu plus lentement que d'habitude et il me fallait plus de temps pour répondre aux questions mais je pouvais toujours tenir une conversation normale.
d) Les conversations normales étaient difficiles parce que c'était dur de commencer à parler.
e) Je me suis senti(e) extrêmement ralenti(e) physiquement, comme embourbé(e).

Choisis la phrase dans chaque groupe qui décrit le mieux la façon dont tu t'es senti(e) lors de <u>la semaine où tu as été le plus déprimé(e) depuis le début de l'année scolaire</u>.

(5)
a) Je ne me suis pas désintéressé(e) de mes activités habituelles.
b) J'étais un peu moins intéressé(e) par une ou deux de mes activités.
c) J'étais moins intéressé(e) par plusieurs de mes activités habituelles.
d) J'avais perdu la plupart de mon intérêt pour presque toutes mes activités habituelles.
e) J'avais perdu tout intérêt pour toutes mes activités habituelles.

(6)
a) Je tirais autant de plaisir que d'habitude de mes activités habituelles.
b) Je tirais un peu moins de plaisir d'une ou deux de mes activités habituelles.
c) Je tirais moins de plaisir de plusieurs de mes activités habituelles.
d) Je ne tirais presque aucun plaisir de la plupart des activités auxquelles je prenais habituellement plaisir.
e) Je ne tirais aucun plaisir d'aucune des activités auxquelles je prenais habituellement plaisir.

(7)
a) Je ne me suis pas senti(e) coupable.
b) Je me suis senti(e) coupable de temps en temps.
c) Je me suis senti(e) souvent coupable.
d) Je me suis senti(e) très coupable la plupart du temps.
e) Je me suis senti(e) extrêmement coupable la plupart du temps.

(8)
a) Je ne me suis pas considéré(e) comme un(e) raté(e).
b) L'opinion que j'ai eue de moi-même a, de temps en temps, été un peu médiocre.
c) Je me suis considéré(e) inférieur(e) à la plupart des gens.
d) Je me suis considéré(e) comme un(e) raté(e).
e) Je me suis considéré(e) comme une personne totalement sans valeur.

(9)
a) Je n'ai pas eu de pensées de mort ou de suicide.
b) Il m'est arrivé de temps en temps de penser que la vie ne vaut pas la peine d'être vécue.
c) Il m'est arrivé fréquemment de penser à mourir de façon passive (comme m'endormir et ne pas me réveiller) ou bien que je serais plus à l'aise mort(e).
d) Il m'est arrivé fréquemment de penser à me tuer, mais je ne croyais pas le faire.
e) Je me serais tué si j'en avais eu l'occasion ou j'ai commis une tentative de suicide.

Choisis la phrase dans chaque groupe qui décrit le mieux la façon dont tu t'es senti(e) lors de <u>la semaine où tu as été le plus déprimé(e) depuis le début de l'année scolaire</u>.

(10)
a) Je ne pouvais pas me concentrer aussi bien que d'habitude.
b) Ma capacité à me concentrer était légèrement moins bonne que d'habitude.
c) La portée de mon attention n'était pas aussi bonne que d'habitude et j'avais du mal à rassembler mes pensées, mais cela ne m'a pas causé de problèmes.
d) Ma capacité à lire ou à tenir une conversation n'était pas aussi bonne que d'habitude.
e) Je n'étais plus capable de lire, de regarder la télé ou d'avoir une conversation, sans grande difficulté.

(11)
a) Je prenais des décisions aussi facilement que d'habitude.
b) Prendre des décisions était légèrement plus difficile que d'habitude.
c) C'était plus dur de prendre des décisions, et cela prenait plus de temps, mais j'en prenais quand même.
d) J'étais incapable de prendre certaines décisions.
e) Je ne pouvais prendre aucune décision.

(12)
a) Mon appétit n'était pas au-dessous de la normale.
b) Mon appétit était légèrement moins bon que d'habitude.
c) Mon appétit n'était manifestement pas aussi bon que d'habitude, mais je mangeais quand même.
d) Mon appétit était beaucoup moins bon à ce moment.
e) Je n'avais plus d'appétit du tout et je devais me forcer pour manger même un peu.

(13)
a) Je n'avais pas perdu de poids OU je suivais un régime.
b) J'avais perdu moins de 2 kg (4 livres).
c) J'avais perdu entre 2 kg et 5 kg (4 à 10 livres).
d) J'avais perdu entre 5 kg et 11 kg (10 à 22 livres).
e) J'avais perdu plus de 11 kg.

(14)
a) Mon appétit n'était pas plus grand que la normale.
b) Mon appétit était légèrement plus grand que d'habitude.
c) Mon appétit était manifestement plus grand que d'habitude.
d) Mon appétit était beaucoup plus grand que d'habitude.
e) J'avais faim tout le temps.

Choisis la phrase dans chaque groupe qui décrit le mieux la façon dont tu t'es senti(e) lors de <u>la semaine où tu as été le plus déprimé(e) depuis le début de l'année scolaire</u>.

(15)
a) Je n'avais pas pris de poids.
b) J'avais pris moins de 2 kg (4 livres).
c) J'avais pris entre 2 kg et 5 kg (4 à 10 livres).
d) J'avais pris entre 5 kg et 11 kg (10 à 22 livres).
e) J'avais pris plus de 11 kg.

(16)
a) Je ne dormais pas moins que la normale.
b) J'avais un peu de mal à dormir de temps en temps.
c) Je ne dormais manifestement pas aussi bien que d'habitude.
d) Je dormais environ la moitié de ma durée normale de sommeil.
e) Je dormais moins de 2 heures par nuit.

(17)
a) Je ne dormais pas plus que la normale.
b) Je dormais parfois plus que d'habitude.
c) Je dormais fréquemment au moins 1 heure de plus que d'habitude.
d) Je dormais fréquemment au moins 2 heures de plus que d'habitude.
e) Je dormais fréquemment au moins 3 heures de plus que d'habitude.

(18)
a) Je ne me sentais pas découragé(e) au sujet de l'avenir.
b) Je me sentais de temps à autre un peu découragé(e) au sujet de l'avenir.
c) Je me sentais souvent découragé(e) au sujet de l'avenir.
d) Je me sentais très découragé(e) au sujet de l'avenir la plupart du temps.
e) Je sentais que l'avenir était sans espoir et que les choses ne s'amélioreraient pas.

(19)
a) Je ne me sentais pas irrité (e) ou contrarié (e).
b) Je devenais de temps à autre un peu plus irrité (e) que d'habitude.
c) Je devenais irrité (e) ou contrarié (e) par des choses qui, d'habitude, ne me tracassent pas.
d) Je me sentais irrité (e) ou contrarié (e) presque tout le temps.
e) Je me sentais si déprimé (e) que je n'étais pas irrité du tout par des choses qui me tracassent habituellement.

Appendix B

Questionnaires developped specifically for the MADDP

PAST PERSONAL DIFFICULTIES

Est-ce qu'on t'a déjà forcé à faire des choses sexuelles avec lesquelles tu n'étais pas d'accord?

a) Oui
b) Non

As-tu déjà été très malade ou eu un accident qui t'a amené à être hospitalisé plus de 20 jours?

a) Non
b) Oui et j'avais 11 ans ou plus la première fois que c'est arrivé.
c) Oui et j'avais 9 à 10 ans la première fois que c'est arrivé.
d) Oui et j'avais 6 à 8 ans la première fois que c'est arrivé.
e) Oui et j'avais moins de 6 ans la première fois que c'est arrivé.

FAMILIAL INSTABILITY

As-tu déménagé au cours de l'année dernière?

a) Oui
b) Non

Tes parents sont actuellement:

a) Encore ensemble
b) Divorcés (ou séparés) et n'ont pas de nouveaux conjoints
c) Divorcés (ou séparés) et mon père a une nouvelle conjointe
d) Divorcés (ou séparés) et ma mère a un nouveau conjoint
e) Divorcés (ou séparés) et ils ont tous les deux un nouveau conjoint
f) Mon père est veuf et célibataire
g) Mon père est veuf et a une nouvelle conjointe
h) Ma mère est veuve et célibataire
i) Ma mère est veuve et a un nouveau conjoint
j) Autre

Chez qui habites-tu habituellement (qui est responsable de ta garde) ?

a) J'habite avec mes deux parents
b) J'habite chez ma mère la moitié du temps et chez mon père l'autre moitié du temps.
c) J'habite avec mon père et je vois ma mère de temps en temps
d) J'habite avec ma mère et je vois mon père de temps en temps
e) J'habite seulement chez mon père
f) J'habite seulement chez ma mère
g) Autre

As-tu déjà été séparé de ton père pour une longue période de temps (+ d'un an) ?

a) Non
b) Oui, et j'avais 11 ans ou plus
c) Oui, et j'avais 10 ans
d) Oui, et j'avais 9 ans
e) Oui, et j'avais 8 ans
f) Oui, et j'avais 7 ans
g) Oui, et j'avais 6 ans
h) Oui et j'avais 5 ans
i) Oui et j'avais moins de 4 ans
j) Je n'ai jamais connu mon père

As-tu déjà été séparé de ta mère pour une longue période de temps (+ d'un an) ?

a) Non
b) Oui, et j'avais 11 ans ou plus
c) Oui, et j'avais 10 ans
d) Oui, et j'avais 9 ans
e) Oui, et j'avais 8 ans
f) Oui, et j'avais 7 ans
g) Oui, et j'avais 6 ans
h) Oui et j'avais 5 ans
i) Oui et j'avais moins de 4 ans
j) Je n'ai jamais connu ma mère

DAILY HASSLES (FAMILY, PEERS, SCHOOL)

En te référant à <u>ce que tu vis actuellement</u>, indique à quel point chacun des éléments suivants constitue pour toi <u>une source de stress</u> (ou d'embêtement).

Est-ce que ça te stresse ?	**PAS DU TOUT**	**UN PEU**	**PAS MAL**	**BEAUCOUP**
Tes parents / beaux-parents	**A**	**B**	**C**	**D**
Le reste de ta parenté	**A**	**B**	**C**	**D**
Les obligations dans ta famille	**A**	**B**	**C**	**D**
Tes amis	**A**	**B**	**C**	**D**
Tes professeurs	**A**	**B**	**C**	**D**
Les autres membres du personnel de ton école	**A**	**B**	**C**	**D**
Les devoirs	**A**	**B**	**C**	**D**
Le fait d'être étudiant	**A**	**B**	**C**	**D**
Ton école	**A**	**B**	**C**	**D**
Tes partenaires de classe	**A**	**B**	**C**	**D**
Les examens	**A**	**B**	**C**	**D**

ACADEMIC ACHIEVEMENT

Au cours de cette année scolaire, quelles sont tes notes moyennes en français (au meilleur de ta connaissance) ?

90% et plus	Entre 80 et 89%	Entre 70 et 79%	Entre 60 et 69%	Moins de 60%
A	B	C	D	E

Au cours de cette année scolaire, quelles sont tes notes moyennes en mathématiques (au meilleur de ta connaissance)?

90% et plus	Entre 80 et 89%	Entre 70 et 79%	Entre 60 et 69%	Moins de 60%
A	B	C	D	E

À QUEL POINT CHACUN DES ÉNONCÉS SUIVANTS EST-IL VRAI POUR TOI ?	**FAUX**	**PLUTÔT FAUX**	**PLUTÔT VRAI**	**VRAI**
La seule chose que mes parents font quand j'ai une mauvaise note c'est de me « tomber » dessus et de « chialer »	**A**	**A**	**C**	**D**
Quand j'ai une mauvaise note, mes parents me font sentir coupable	**A**	**A**	**C**	**D**
Pour mes parents, il est très important que je sois parmi les premiers(ères) de ma classe.	**A**	**A**	**C**	**D**
Mes parents sont mécontents lorsque mes notes sont « dans la moyenne »	**A**	**A**	**C**	**D**

Es-tu satisfait du nombre d'ami(e)s que tu as aujourd'hui ?

a) Je suis très satisfait du nombre d'amis que j'ai actuellement.
b) Je suis satisfait du nombre d'amis que j'ai actuellement.
c) Je suis insatisfait du nombre d'amis que j'ai actuellement.
d) Je suis très insatisfait du nombre d'amis que j'ai actuellement.

Est-ce que tu trouves qu'il est facile de se faire de nouveaux ami(e)s dans ta nouvelle école ?

a) Je trouve qu'il est très facile de s'y faire de nouveaux amis.
b) Je trouve qu'il est facile de s'y faire de nouveaux amis.
c) Je trouve qu'il est difficile de s'y faire de nouveaux amis.
d) Je trouve qu'il est très difficile de s'y faire de nouveaux amis.

DISSATISFACTION WITH SCHOOL CONTROL MECHANISMS
(DISCIPLINE AND ACADEMIC)

Les questions suivantes visent à évaluer ton opinion concernant certains aspects de la vie dans ton école.

Selon toi, les règlements de ton école sont-ils ?

a) Trop sévères
b) Assez sévères
c) Pas assez sévères

Selon toi, les différents endroits de ton école (corridors, toilettes, casiers, cour d'école, etc.) sont-ils ?

a) Trop surveillés.
b) Assez surveillés.
c) Pas assez surveillés.

Selon toi, les punitions utilisées dans ton école lorsqu'un élève se comporte mal sont-elles ?

a) Trop sévères.
b) Assez sévères.
c) Pas assez sévères.

En pensant aux devoirs et aux examens que les élèves de ton école ont habituellement à faire, dirais-tu que ?

a) Il y en a trop.
b) Il y en a assez.
c) Il n'y en a pas assez.

En pensant au nombre de règlements qu'il y a dans ton école, dirais tu que ?

a) Il y a trop de règlements.
b) Il y a assez de règlements.
c) Il n'y a pas assez de règlements.

En pensant à tout ce que les élèves de ton école ont habituellement à faire dans une semaine d'école, dirais-tu que, d'habitude ?

a) Il y a trop de choses à faire.
b) Il y a juste assez de choses à faire.
c) Il n'y a pas assez de choses à faire.

DISSATISFACTION WITH SCHOOL SUPPORT MECHANISMS
(HELP AND ENCOURAGEMENTS)

ES-TU D'ACCORD AVEC LES ÉNONCÉS SUIVANTS	OUI	NON
Dans mon école, il devrait être plus facile de recevoir de l'aide lorsque nous (les élèves) avons des problèmes personnels (psychologue, travailleur social, etc.)	A	B
Les enseignants de mon école devraient faire plus d'efforts pour nous motiver et nous intéresser au contenu des cours	A	B
En dehors des cours, les élèves et les enseignants de mon école devraient prendre plus de temps pour parler ensemble	A	B
Les enseignants de mon école devraient nous (les élèves) encourager davantage lorsque nous faisons du travail difficile en classe	A	B
Dans mon école, il devrait être plus facile de recevoir de l'aide lorsque nous (les élèves) avons des difficultés scolaires (récupération, tutorat, etc.)	A	B
En général, les élèves et les enseignants de mon école devraient avoir plus de plaisir à être ensemble	A	B
Les adultes de mon école devraient être plus disponibles pour nous écouter et nous offrir de l'aide lorsque nous en avons besoin	A	B

Chapitre IV

Conclusion

Cette thèse avait pour objectif général d'apporter un début de réponse à deux des principales limites des connaissances actuelles concernant le développement de la dépression chez l'enfant et l'adolescent : (a) le manque d'intégration des connaissances et (b) la méconnaissance du rôle du vécu scolaire. À l'égard ces visées, le présent chapitre propose une synthèse des résultats obtenus et revient sur les principaux défis qui en découlent.

UNE SYNTHÈSE DES CONNAISSANCES

Dans le second chapitre de cette thèse, une recension des travaux produits au cours des 12 dernières années et portant sur l'identification des antécédents psychosociaux intervenant dans le développement de la dépression chez l'enfant et l'adolescent a été effectuée. En s'appuyant sur des critères rigoureux de sélection des études, en vue de maximiser la validité des résultats obtenus, 91 études ont été retenues et analysées.

La première conclusion qui se dégage de cette analyse est qu'une évolution surprenante des connaissances concernant le développement de la dépression s'est produite au cours des dernières décennies. En effet, le rôle de nombreux facteurs de risque et de protection commence maintenant à être clairement cerné. Nos connaissances des mécanismes en jeu dans le développement de la dépression a tant progressé au cours des dernières années qu'il est maintenant difficile de croire que la dépression infantile et adolescente était considérée, il y a si peu de temps, comme une impossibilité (Claes, 1983).

Par ailleurs, cette analyse révèle aussi que ce que nous avons décrit dans le premier chapitre comme un « recours répété à une approche fragmentée d'intégration des connaissances » est bel est bien présent dans ce domaine de recherche. En effet, bien

que l'appel à une plus grande multidisciplinarité dans le domaine de la recherche développementale semble avoir été entendu, il n'est pas clair qu'il ait été clairement écouté. En d'autres termes, bien que la majorité des groupes de recherche développementaux actuels soient de nature multidisciplinaire (médecins, psychologues, sociologues, démographes, etc.), les travaux théoriques et empiriques effectués par ces groupes tendent encore à se limiter à l'étude d'un nombre restreint de facteurs de risque et de protection. Des schèmes méthodologiques et devis analytiques de plus en plus complexes sont maintenant appliqués à des questions de plus en plus circonscrites et limitées, plutôt qu'à des questions de complexité équivalente, mais nécessitant un plus grand niveau d'intégration.

Bien entendu, ce ne sont pas en elles-mêmes l'utilité et la pertinence du premier type de travaux qui posent problème, mais bien l'absence quasi complète d'analyses se situant au second niveau. Si cette lacune peut s'expliquer par les pressions de publication auxquelles font face les chercheurs actuels, cette explication ne devrait pas servir de justification à l'abandon de la seconde approche. Après tout, la physique moderne a bien démontré le caractère complémentaire et inséparable des études portant sur l'infiniment grand et sur l'infiniment petit. En ce qui concerne la compréhension des facteurs actifs dans le développement de la dépression, la conséquence de cette limite est un portrait relativement fragmenté des mécanismes en jeu : les arbres cachent la forêt!

Tout au long du second chapitre de cette thèse, nous nous sommes efforcé de présenter les différents modèles théoriques sous-jacents à l'étude des antécédents associés au développement de la dépression chez l'enfant et l'adolescent, et avons insisté sur le fait que ces modèles n'avaient que trop rarement fait l'objet d'évaluations rigoureuses et systématiques. Ce constat limite donc leur utilité potentielle pour guider l'intégration des connaissances, et cette limite est d'autant plus grande que la majorité de ces modèles ne prennent en considération qu'un nombre fort limité de facteurs de risque et de protection. Heureusement, certains modèles issus de la psychopathologie développementale (Compas, Langrock, Keller, Merchant, & Copeland 2002; Rutter, 1986; Rutter & Sroufe, 2000), tels que les modèles organisationnels (Cicchetti &

Rogosh, 2002; Cicchetti & Schneider-Rosen, 1986), transactionnels (Sameroff, 2000; Sameroff & Chandler, 1975), écosystémiques (Bronfenbrenner, 1977) et du déséquilibre personne-environnement (Eccles, Lord, & Midgley, 1991; Eccles *et al.*, 1993), représentent des outils puissants, susceptibles d'orienter ces efforts d'intégration, bien qu'ils n'aient que rarement été invoqués pour expliquer les mécanismes du développement de la dépression (Cicchetti & Toth, 1998; Cyranowski, Frank, Young, & Shear, 2000; Goodman & Gotlib, 1999, 2002).

Ces différents cadres conceptuels reposent sur trois postulats principaux. Premièrement, une multitude de facteurs de risque et de protection individuels et environnementaux semblent associés au développement de la dépression. Pour emprunter un terme utilisé dans les écrits portant sur les fondements de la psychopathologie développementale, la dépression semble représenter l'équifinalité (ou le *final common pathway*) d'une multitude d'antécédents (Cicchetti & Rogosh, 2002; Rutter, 1986; Rutter & Sroufe, 2000). Deuxièmement, puisque ces différents facteurs sont interreliés, leur effet sur l'émergence de symptômes dépressifs ne se produit pas en vase clos. Il apparaît donc irréaliste d'espérer réussir à isoler les effets individuels de chacun d'entre eux dans le cadre d'études empiriques. En d'autres termes, la dépression résulterait plutôt d'un état de déséquilibre affectant l'homéostasie (ou *gestalt*) des différents systèmes internes et externes de l'individu. Troisièmement, compte tenu du fait que le développement humain résulte d'une série de réorganisations internes et externes successives liées à la nature spécifique des tâches développementales associées à des âges différents, et que ces tâches peuvent elles-mêmes varier en fonction du sexe des individus (Cicchetti & Toth, 1998; Erickson, 1968; Gladstone & Beardslee, 2002; Goodman, 2002; Petersen & Lefert, 1995; Piaget, 1972), il est hautement probable que l'effet des différents facteurs de risque et de protection liés au développement de la dépression varie en fonction de l'âge et du sexe des individus.

La principale limite de ces cadres conceptuels eu égard au dévelopement de la dépression provient de leur manque de spécificité face à d'autres problèmes d'adaptation. En contrepartie, la majorité des facteurs reliés au développement de la

dépression interviennent aussi dans le développement d'autres problèmes d'adaptation. Par exemple, l'exposition à des événements de vie négatifs est également associée au développement de nombreux autres problèmes psychosociaux (Lewinsohn, Gotlib, & Seeley, 1995; Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998; McMahon, Grant, Compas, Thurm, & Ey, 2003). Il est de plus généralement reconnu que le risque d'inadaptation des enfants de parents dépressifs n'est pas limité au développement de la dépression (Berg-Nielsen, Vikan, & Dahl, 2002), et que les mécanismes héréditaires en jeu dans la dépression le soit en outre dans l'émergence d'un ensemble assez vaste d'autres problèmes d'adaptation (Kendler *et al.*, 1995). En résumé, il est probable que ces différents cadres conceptuels puissent pareillement servir de guide à l'étude d'autres types de problèmes. Cette limite apparente est cependant conforme au principe de multifinalité de la psychopathologie développementale, qui postule que deux individus exposés à un ensemble identique de facteurs de risque et de protection peuvent tout de même emprunter des trajectoires développementales fort différentes (Cicchetti & Rogosh, 2002; Rutter, 1986; Rutter & Sroufe, 2000). De surcroît, ce manque de spécificité est également susceptible de représenter plus adéquatement le niveau élevé de comorbidité souvent associé à la dépression. Bien entendu, si ces deux dernières remarques n'enlèvent rien au besoin d'accorder une importance accrue à la spécificité des mécanismes à la base du développement de la dépression, elles soutiennent tout de même la synthèse proposée en soulignant sa conformité aux connaissances actuelles concernant les mécanismes du développement psychosocial normatif des enfants et des adolescents.

LES DÉFIS RELEVÉS

Outre les conclusions précédentes, les résultats exposés dans le second chapitre de cette thèse permettent de circonscrire trois défis majeurs auxquels les travaux futurs devront tenter de faire face. Une description détaillée de ces défis ayant été précédemment présentée, nous nous contenterons ici d'en présenter un résumé et mettrons davantage l'accent sur la manière dont ces défis ont été relevés dans le troisième chapitre.

Le défi méthodologique

Ce premier défi est de nature méthodologique et couvre quatre aspects :

(a) un besoin accru d'études basées sur des devis méthodologiques longitudinaux, rigoureux et systématiques;

(b) la nécessité d'arriver à une opérationnalisation plus parcimonieuse des facteurs individuels actifs dans le développement de la dépression;

(c) le besoin d'accorder une attention accrue à l'opérationnalisation des facteurs sociaux potentiellement en jeu dans le développement de la dépression;

(d) l'importance de distinguer les facteurs de risque et de protection selon que leur effet est limité ou non à l'aggravation ou à l'émergence de symptômes dépressifs.

Dans le cadre de l'étude présentée dans le troisième chapitre, certains de ces aspects ont pu être considérés. En effet, le devis prospectif et longitudinal utilisé, le nombre de sujets suivis et l'inclusion de variables de contrôle multiples font du PMDDA une étude dont la rigueur méthodologique est conforme aux critères exposés dans le second chapitre (a). De plus, le recours à une opérationnalisation claire et détaillée, ancrée dans un cadre conceptuel validé (Janosz, Georges, & Parent, 1998), des différentes facettes du

vécu scolaire des adolescents, représente un premier pas en vue d'en arriver à une opérationnalisation plus systématique des différents facteurs sociaux potentiellement liés au développement de la dépression (c). Les résultats obtenus ont d'ailleurs fourni un appui préliminaire à l'opérationnalisation retenue.

En contrepartie, malgré ses qualités, le devis méthodologique utilisé dans le cadre du PMDDA n'a pas permis d'évaluer le rôle potentiel des caractéristiques agrégées et structurelles de l'environnement scolaire dans le développement de la dépression. En effet, l'évaluation de ces aspects du vécu scolaire aurait nécessité le recours à des analyses statistiques multi-niveaux (*hierarchical linear modeling*) afin de départager les effets des caractéristiques individuelles de ceux des caractéristiques génériques des écoles (Bryk & Raudenbush, 1992; Gottfredson, 2000). Dans de telles analyses, l'évaluation de cette seconde source d'influence repose sur des analyses dans lesquelles chaque école devient un sujet. Le PMDDA incluait un nombre insuffisant d'écoles (n = 5) pour mener de telles analyses avec un pouvoir statistique suffisant.

Finalement, la vérification du rôle modérateur du niveau antérieur de symptômes dépressifs manifestés par les élèves, quant à l'impact des différents facteurs de risques identifiés, a révélé que l'effet de certaines variables liées au vécu scolaire des adolescents était effectivement limité à l'émergence ou à l'aggravation de symptômes dépressifs (d). Plus précisément, l'effet de plusieurs aspects du vécu scolaire des élèves sur le développement de la dépression semble être plus important chez les élèves présentant déjà un niveau modéré ou élevé de symptômes dépressifs : une faible implication parascolaire, un niveau élevé de difficultés liées à la transition primaire-secondaire, la victimisation mineure, les conflits maîtres-élèves, l'insatisfaction des élèves à l'égard des mécanismes d'encouragement utilisés à l'école, une perception négative des climats d'appartenance, relationnel (entre élèves) et de justice de l'école, l'impression que des problèmes mineurs de violence survienent souvent à l'école et une perception négative des pratiques disciplinaires, consultatives, de gestion de classe, parascolaires et de collaboration école-famille. En contrepartie, l'effet de la victimisation majeure et sexuelle semble plus important pour les élèves présentant

antérieurement de plus faibles niveaux de symptômes dépressifs. Ces résultats indiquent clairement que divers aspects du vécu scolaire des élèves sont susceptibles d'exercer un impact différentiel sur l'émergence ou sur l'aggravation de symptômes dépressifs.

Le défi théorique

Le second défi est de nature théorique et comporte quatre éléments :

(a) le besoin d'études systématiques visant à examiner les rôles respectifs des relations avec les pairs, du vécu scolaire et du voisinage dans le développement de la dépression;

(b) la nécessité d'évaluer rigoureusement et systématiquement la validité des multiples propositions théoriques visant à expliquer le développement de la dépression;

(c) l'importance de porter une attention particulière à l'identification des facteurs de protection actifs dans le développement de la dépression et de compléter les analyses traditionnelles centrées sur les variables par des analyses typologiques centrées sur la personne.

(d) le besoin de reproduire de manière indépendante la recension des écrits présentées au second chapitre de cette thèse, afin d'évaluer si des chercheurs guidés par des grilles de lectures différentes arriveraient aux mêmes conclusions.

L'étude décrite au troisième chapitre apporte un début de réponse aux deux premiers des aspects précités (a et b). Ainsi, en plus de permettre une évaluation systématique du rôle du vécu scolaire dans le développement de la dépression, le cadre conceptuel et méthodologique proposé dans cette étude est conforme à certains des postulats de la psychopathologie dévelopementale exposés au second chapitre. En effet, définir le vécu scolaire des élèves en fonction des caractéristiques des écoles fréquentées, des différents microsystèmes qui y interagissent et des caractéristiques psychologiques des élèves qui y

sont directement exprimées illustre bien le caractère interrelié des différentes sources d'influence sur le développement humain postulé dans ce cadre conceptuel. En outre, cette interrelation a aussi été prise en considération dans le choix des variables de contrôle potentielles, ce choix s'appuyant sur le postulat que tout facteur susceptible d'influencer le développement d'un adolescent risque aussi d'avoir des répercussions sur son vécu scolaire. Les résultats obtenus confirment d'ailleurs partiellement ces différentes propositions.

Ainsi, cette étude a clairement révélé que la majorité des aspects du vécu scolaire évalués représentaient des prédicteurs significatifs du développement de la dépression chez les adolescents, et que cette relation était relativement indépendante du niveau de base de dépression manifesté par les participants. En contrepartie, le fait que plusieurs de ces prédicteurs deviennent non significatifs lorsqu'ils sont simultanément considérés dans les analyses suggère qu'ils ne sont pas tous directement en jeu, au sens causal du terme, dans le développement de la dépression. Par exemple, alors que les six dimensions du climat scolaire évaluées représentent individuellement des prédicteurs significatifs du développement de la dépression, leur considération simultanée dans les analyses a révélé que seuls les climats de justice et de sécurité apportent une contribution significative directe à la prédiction de la dépression. Une interprétation prudente de ce résultat serait que l'effet sur la dépression des autres dimensions du climat scolaire ne représente qu'un artefact statistique de leur corrélation avec les climats de justice et de sécurité. Dans ce contexte, seuls les climats de justice et de sécurité représentent des « causes » potentielles de la dépression. D'un autre côté, compte tenu des interrelations observées entre les différentes composantes du climat scolaire, cette conclusion n'enlève rien à l'utilité préventive des autres dimensions du climat scolaire puisque toute amélioration apportée à l'une de ces dimensions est susceptible d'exercer un impact positif sur les climats de justice et de sécurité. Si cette dernière interprétation était vérifiée, elle apporterait donc un soutien direct aux propositions d'interrelations du cadre conceptuel décrit à la fin du second chapitre. Seules des analyses de médiation plus poussées permettront de clarifier ces questions.

En outre, l'inclusion des variables de contrôle (caractéristiques individuelles, familiales et sociales liées au vécu non scolaire des élèves) dans les analyses n'affecte que de façon minimale les résultats obtenus. En effet, seuls cinq des prédicteurs précédemment identifiés comme liés au développement de la dépression deviennent non significatifs lors de cette étape des analyses : le sentiment d'efficacité personelle académique des élèves, la pression académique que leurs parents leur font subir, le caractère stressant de leurs relations d'amitié à l'école et l'adaptation scolaire de leurs amis et leur exposition à des agressions de nature sexuelle ou amoureuse à l'école. Ces derniers résultats suggèrent que la relation précédemment observée entre ces variables et le développement de symptômes dépressifs représente un artefact de leur association avec différents aspects du vécu non scolaire des adolescents, eux-mêmes reliés au développement de la dépression. En contrepartie, le fait que seulement cinq des variables évaluées deviennent non significativement liées à la dépression à cette étape des analyses suggère la relative indépendance des effets des variables scolaires et non scolaires sur le développement de la dépression. Conséquemment, il apparaît qu'une intervention préventive aurait avantage, pour être efficace, à cibler conjointement ces deux facettes du vécu des adolescents. Cette conclusion est d'ailleurs renforcée par le fait que nos analyses finales ont révélé que les variables de contrôle et les variables liées au vécu scolaire des élèves exerçaient une contribution équivalente à la prédiction des symptômes de la dépression manifestés par les participants à la fin de l'étude.

De surcroît, les analyses finales ont révélé que les différents effets d'interaction évalués (rôle modérateur du sexe et du niveau antérieur de symptômes dépressifs) contribuent autant que les effets principaux à la prédiction du développement de la dépression. Une implication très importante de ce résultat est que l'effet du vécu scolaire sur le développement de la dépression varie en fonction des caractéristiques spécifiques des élèves considérés. Ce résultat renforce le besoin précédemment souligné (c) de compléter les résultats d'analyses classiques centrées sur les variables par les résultats d'analyses centrées sur la personne (Von Eye & Bergman, 2003). Ce second type d'analyse, qui permet de tenir compte de la variabilité interindividuelle et du fait que l'individu « moyen » considéré dans les modèles analytiques classiques n'existe pas,

croît rapidement en popularité en recherche développementale (Bergman, 2000; Nagin, 1999; Von Eye & Bergman, 2003). Hélas, cette popularité ne semble pas encore avoir gagné les études portant sur le développement de la dépression.

Finalement, l'objectif de cette étude concernait l'identification du rôle de variables liées à la vie scolaire à titre de facteurs de risque pour le développement de la dépression. Le rôle de ces mêmes variables comme facteurs de protection potentiels pour les élèves à risque n'a pas été examiné et devrait indéniablement faire l'objet d'études ultérieures. Or, des études antérieures ont révélé que, bien que certaines variables scolaires (c.-à-d. l'engagement scolaire et la chaleur des relations maîtres-élèves) ne constituent pas des facteurs de risques pour le développement de différents problèmes psychosociaux (problèmes d'adaptation scolaire, toxicomanie), ces variables représentent d'importants facteurs de protection pour les élèves les plus à risque (Fallu & Janosz, 2003, Fallu, Morin, & Janosz, sous presse).

Le défi de généralisation

Le troisième défi concerne la généralisation des résultats obtenus et souligne l'importance de vérifier la variabilité culturelle ou ethnique, socio-économique, sexuelle et développementale des effets observés.

Dans le troisième chapitre de cette thèse, la décision d'évaluer l'effet modérateur du sexe des élèves sur les relations observées visait à fournir une réponse préliminaire à ce défi. Dans ce contexte, l'observation que l'effet de nombreuses variables scolaires sur le développement de la dépression est limité aux filles, ou plus importante chez ces dernières que chez les garçons, contribue clairement à hausser le potentiel de généralisation des résultats obtenus. En outre, cette observation fournit un soutien important à plusieurs modèles théoriques antérieurs ayant tenté d'expliquer les différences intersexes généralement observées au niveau de la dépression (Bebbington, 1996; Cyranowski *et al.*, 2000; Nolen-Hoeksema, 2002). En effet, ces modèles soutiennent généralement que, parce que les changements pubertaires débutent

généralement plus hâtivement chez elles que chez les garçons, les filles tendent à débuter l'adolescence dans un état de déséquilibre biopsychosocial plus important (Cyranowski *et al.*, 2000). La puberté représentant un événement déstabilisant en soi, elle peut exposer les adolescents à un déséquilibre majeur lorsqu'elle se produit en même temps que d'autres transitions importantes. Or, de nombreux résultats indiquent que, parce que leur maturation pubertaire survient plus tôt, les filles tendent souvent à faire l'expérience simultanée de la puberté et de la transition du primaire au secondaire (Bebbington, 1996), ce qui augmenterait leur risque de développer une dépression (Ge, Conger, & Elder, 1996; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997). Compte tenu du déséquilibre résultant de cette double transition, il est probable que les filles puissent, à ce moment, être plus sévèrement affectées que les garçons par différentes sources de stress liées à leur vécu scolaire, et qu'elles puissent bénéficier davantage du soutien qu'elles reçoivent à l'école.

Les résultats obtenus soutiennent cette hypothèse. En effet, c'est uniquement chez les filles qu'une faible implication scolaire, un niveau plus élevé d'insatisfaction face aux pratiques disciplinaires utilisées dans l'école et une perception plus fréquente de l'occurrence de problèmes de violence mineure, de violence majeure et de problèmes scolaires à l'école ont été identifiés comme des prédicteurs significatifs du développement de la dépression. De même, l'effet d'autres variables sur le développement de la dépression s'est avéré être plus important chez les filles que chez les garçons. Ces variables sont l'inadaptation scolaire, la victimisation mineure et majeure, le stress scolaire, les relations maître-élève conflictuelles et le climat d'insécurité.

En contrepartie, plusieurs caractéristiques du devis méthodologique utilisé posent une limite considérable au potentiel de généralisation des résultats. Premièrement, la partie du PMDDA utilisée dans le troisième chapitre repose sur un suivi à court terme (8 mois) d'élèves venant tout juste de vivre la transition du primaire au secondaire, une période notoire d'instabilité dans la vie des adolescents. Avant que ces résultats ne puissent être reproduits auprès d'échantillons plus âgés, il est risqué de tenter de les généraliser au

reste du parcours développemental des adolescents. Deuxièmement, notre désir de maximiser les différences organisationnelles interécoles nous a amené à suréchantillonner des étudiants doués ou exposés à un milieu scolaire plus encadrant que la moyenne (écoles privées). Les analyses d'attrition effectuées ont en outre révélé que les élèves les plus à risque avaient été perdus au cours de l'étude. Ces deux éléments réduisent sérieusement la représentativité de l'échantillon utilisé.

QUELQUES LIGNES DIRECTRICES POUR LES EFFORTS PRÉVENTIFS

Nonobstant les limites précédemment identifiées, les résultats obtenus fournissent tout de même certaines lignes directrices susceptibles de guider les efforts préventifs. Premièrement, ces résultats suggèrent que la modification de nombreux facteurs, tant individuels que sociaux, puisse faire l'objet de programmes préventifs. En effet, la majorité de ces résultats, bien qu'ils soient issus de méthodologies fort différentes, convergent vers une conclusion principale : le développement de la dépression semble être le fruit de l'action d'une multitude de facteurs de risque et de protection biopsychosociaux, dont l'action globale ne peut être réduite au cumul des effets isolés de chacun de ces facteurs. Par conséquent, toute modification qui serait apportée à l'interrelation entre ces facteurs risquerait d'influencer le risque général d'un individu de développer des symptômes dépressifs. Cette conclusion est d'ailleurs conforme à celle d'une recension des écrits antérieure portant sur la prévention de la dépression, et dans laquelle nous avions montré qu'à ce jour, les programmes préventifs les plus efficaces étaient ceux qui reposaient sur une modification du potentiel d'adaptation des individus ciblés à l'égard de leur environnement, ou sur une modification de la capacité de cet environnement à répondre aux besoins développementaux des individus (Morin & Chalfoun, 2003).

Deuxièmement, les facteurs en jeu dans le développement de la dépression sont si nombreux qu'il est peu probable qu'ils affectent tous un même individu. Par conséquent,

les mécanismes de dépistage utilisés dans le cadre de programmes de prévention ciblés devront être raffinés afin de pouvoir considérer à la fois certains facteurs de risque sévères et non spécifiques, qui créent chez l'individu exposé un niveau élevé de risque de développer un problème psychosocial quelconque (p. ex. l'agression sexuelle), et d'autres facteurs moins sévères, mais spécifiques à la dépression. Cette dernière affirmation repose sur la constatation que la majorité des facteurs associés au développement de la dépression ont un pouvoir prédictif faible et que, par conséquent, le développement de la dépression résulte plus probablement d'interactions complexes entre plusieurs de ces facteurs.

Troisièmement, la prévention de la violence en milieu scolaire semble représenter une avenue de choix pour prévenir l'émergence de symptômes dépressifs. En effet, parmi les facteurs de risque les plus importants identifiés dans le cadre du troisième chapitre, plusieurs font référence à la violence en milieu scolaire. En elle-même, la prévention de cette violence, tout comme celle de la dépression, a maintes fois été identifiée comme une priorité pour la société moderne (voir Gottfredson, 2000; Gottfredson & Gottfredson, 1985). Dans ce contexte, les résultats obtenus suggèrent qu'une même intervention peut représenter un moyen efficace d'atteindre simultanément ces deux cibles prioritaires. Cette possibilité est d'autant plus stimulante qu'il est généralement reconnu que la démonstration des effets bénéfiques d'un programme préventif eu égard au développement de problèmes multiples a pour effet de maximiser le rapport coûts-bénéfices associé au programme et, par le fait même, de favoriser son acceptation sociale et politique (Morin & Chalfoun, 2003).

Quatrièmement, il est admis depuis longtemps que le milieu scolaire, parce qu'il est relativement accessible à la majorité des enfants et des adolescents, représente un milieu de choix pour l'implantation de programmes de prévention de la dépression (voir Morin & Chalfoun, 2003). Les résultats obtenus dans le troisième chapitre de cette thèse permettent de préciser cette affirmation en ajoutant que ce milieu, en plus de représenter un endroit de choix pour l'implantation de programmes de prévention, constitue aussi une cible de choix pour ces programmes. Pour reprendre à notre compte les propos de

Janosz et Leclerc (1993), la prévention de la dépression en milieu scolaire aurait donc avantage à combiner une intervention *sur le milieu* aux méthodes plus classiques d'interventions implantées *dans le milieu.*

Finalement, les résultats des analyses de modération effectuées au troisième chapitre de cette thèse renforcent la suggestion de Kessler (1997) quant au besoin d'évaluer l'impact différentiel potentiel de différents facteurs de risque sur l'émergence et l'aggravation de symptômes dépressifs. En effet, les résultats présentés révèlent clairement que l'impact de certains facteurs de risque diffère en fonction du niveau de symptômes antérieurement manifestés par les participants. Ces résultats ont une implication pratique importante. En effet, un programme de prévention se distingue d'un programme de traitement par le fait qu'il cible généralement des individus n'ayant pas encore développé le problème à prévenir afin de les aider à demeurer asymptomatiques. Conséquemment, un programme de prévention de la dépression a intérêt, pour être efficace, à reposer sur la modification de facteurs de risque et de protection associés à l'émergence de symptômes dépressifs plutôt qu'à l'aggravation de symptômes déjà présents. L'inverse est aussi vrai en ce qui concerne les programmes de traitement de la dépression. Concrètement, ces résultats indiquent qu'il serait risqué de s'inspirer des programmes de traitement de la dépression reconnus comme efficaces afin de guider l'élaboration de programmes de prévention. Cette mise en garde est d'ailleurs renforcée par les résultats de l'étude de Lewinsohn, Allen, Seeley et Gotlib (1999), présentée au second chapitre, qui ont révélé qu'un style négatif d'attribution, soit une cible de choix pour de nombreux programmes de traitement efficaces de la dépression (Hollon, Haman, & Brown, 2002), était surtout associé à l'aggravation d'états dépressifs plutôt qu'à leur émergence. Il est intéressant de constater que cette dernière conclusion converge elle aussi vers les conclusions de la recension de Morin et Chalfoun (2003), qui ont observé que les programmes de prévention directement inspirés des modèles thérapeutiques traditionnels démontraient généralement peu d'efficacité.

COMPRENDRE LES MÉCANISMES EN JEU

Cette thèse révèle assurément qu'une multitude de facteurs, tant individuels que sociaux, interviennent de toute évidence dans le développement de la dépression chez l'enfant et l'adolescent. En contrepartie, le pouvoir explicatif de ces facteurs est généralement faible. S'il est possible d'affirmer que la considération simultanée de plusieurs de ces facteurs puisse augmenter ce pouvoir prédictif, ces facteurs sont si nombreux qu'il est irréaliste de penser qu'ils puissent tous être simultanément actifs dans le développement de chacun des cas individuels de dépression. Par exemple, il est clair que les individus dépressifs n'ont pas tous été précédemment exposés à des événements de vie négatifs, ne présentent pas tous des niveaux élevés de névrotisme et n'ont pas tous des antécédents familiaux de dépression (risque génétique). Dans les deux chapitres centraux de cette thèse, de nombreux résultats ont aussi clairement indiqué que l'effet des facteurs de risque et de protection identifiés variait souvent dans différents sous-groupes de sujets.

Cette observation illustre bien la limite inhérente aux méthodes d'analyse centrées sur les variables utilisées dans les études décrites dans cette thèse. Pour cette raison, il apparaît de plus en plus clair qu'une compréhension intégrée des mécanismes de base ne sera possible que lorsque ces études pourront être complétées par des analyses centrées sur la personne (Bergman, 2000; Nagin, 1999; Von Eye & Bergman, 2003). Déjà, l'ensemble des résultats et des hypothèses explicatives présentés précédemment laisse entrevoir au moins trois trajectoires distinctes susceptibles de mener à la dépression.

La première trajectoire repose vraisemblablement sur un risque héréditaire, s'exprimant en partie par un niveau élevé de névrotisme (instabilité émotionnelle) et par un dérèglement du système biologique de réponse au stress. Ce déséquilibre psychobiologique pourrait alors augmenter le niveau d'anxiété et de réactivité au stress des individus concernés. Toute source de stress supplémentaire risquerait donc de déclencher chez ces individus le développement de symptômes dépressifs.

La seconde trajectoire comporte apparemment l'exposition à des expériences de socialisation inadéquates en bas âge, entraînant chez l'enfant le développement d'un sentiment d'inadéquation et d'une faible estime de soi. L'enfant tendrait par la suite à appliquer les modèles d'interaction ainsi appris à d'autres contextes, se privant de ce fait de sources importantes de soutien et de valorisation et augmentant le risque d'être à nouveau rejeté. Ainsi rejetés, ces individus pourraient subséquemment développer des sentiments d'inadéquation et de désespoir susceptibles de déclencher chez eux le développement de symptômes dépressifs.

Une troisième trajectoire concerne vraisemblablement davantage les filles que les garçons et consiste en l'accumulation d'expériences déstabilisantes au début de l'adolescence. Ainsi, les filles présentant préalablement un niveau plus élevé de dépendance dès l'enfance verraient cette tendance s'exacerber à la suite des transformations pubertaires du début de l'adolescence. Cette tendance accrue les rendrait alors plus sensibles à la multitude de changements sociaux survenant au début de l'adolescence (modification des groupes de pairs, adaptation à un nouvel environnement scolaire, début des fréquentations amoureuses et autonomie accrue face à la famille). Ce n'est cependant que lorsque ces changements multiples surviennent de manière simultanée que les filles risqueraient de développer des symptômes de dépression.

Il est intéressant de constater que, dans une étude portant sur un échantillon de jumelles adultes et intégrant un maximum de prédicteurs potentiels, Kendler, Gardner et Prescott (2002) en sont arrivés à une conclusion fort similaire. Bien entendu, cette proposition demeure pour l'instant hypothétique et devra faire l'objet de recherches plus systématiques dans le futur. La complexité de cette question rend d'ailleurs irréaliste la possibilité qu'une telle hypothèse puisse être vérifiée dans le cadre d'une seule étude exploratoire. Vraisemblablement, un véritable programme de recherche multidisciplinaire, ancré dans de solides assises théoriques et combinant différents types d'analyses (centrées sur les variables et sur les personnes), sera nécessaire à l'atteinte d'une compréhension à la fois intégrée et parcimonieuse des mécanismes responsables du développement de la dépression.

ÉPILOGUE

La compréhension des mécanismes en jeu dans le développement de la dépression chez l'enfant et l'adolescent a considérablement évolué au cours des dernières décennies. En effet, alors qu'il n'y a pas si longtemps on considérait impossible qu'un enfant ou un adolescent puisse souffrir de dépression, ce domaine de recherche est aujourd'hui dynamique et prometteur. Des travaux actuels émergent un ensemble d'hypothèses riches et stimulantes, qui permettent de croire qu'il sera bientôt possible d'agir de façon à enrayer le développement de ce sérieux problème de santé mentale. Pour ce faire, des modèles théoriques et analytiques plus complexes devront être élaborés afin de prendre en considération l'ensemble des facteurs en jeu et l'existence potentielle de trajectoires développementales distinctes. Heureusement, nous croyons fermement que les connaissances actuelles sont prêtes à soutenir les efforts d'intégration qui seront requis pour venir à bout de ce problème.

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