

# Clarifying the Link between Parental Psychological Control and Adolescents' Depressive Symptoms

## Reciprocal versus Unidirectional Models

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Research has demonstrated consistent positive associations between perceived parental psychological control and adolescents' depressive symptoms, but the direction of influence remains unclear. Using a cross-lagged longitudinal design in two samples of late (Study 1,  $N = 396$ ) and middle (Study 2,  $N = 724$ ) adolescents, this study compared three models, that is, a psychological control effects model, an adolescent adjustment effects model, and a reciprocal model. Structural equation modeling analyses generally favored the reciprocal model over each of the unidirectional models. The cross-lagged effects of perceived psychological control remained significant after controlling for two important parenting dimensions (i.e., parental responsiveness and behavioral control; Study 1) and were found in all types of parent-adolescent dyads except for the mother-daughter dyad (Study 2). Implications for the understanding of the mechanisms that underlie the deleterious effects of parents' psychological control on adolescent adjustment are discussed.

Adolescence represents a developmental phase marked by increased vulnerability to depressive symptoms (Ge, Lorenz, Conger, Elder, & Simons,

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*Merrill-Palmer Quarterly*, October 2008, Vol. 54, No. 4, pp. 411–444. Copyright © 2008 by Wayne State University Press, Detroit, MI 48201.

1994), particularly among females (Nolen-Hoeksema & Girgus, 1994). An important challenge for developmental psychologists is to identify factors that halt or exacerbate adolescents' trajectories of depressive symptoms. Although theory and research have generally supported the notion that the family—and parents' rearing style in particular—can contribute to or mitigate a vulnerability to depression (Galambos, Barker, & Almeida, 2003), most research on the role of parenting in depressive symptoms has been cross-sectional in nature. Due to a dearth of well-designed longitudinal studies, the direction of effects in associations between parenting and adolescent depressive symptoms remains understudied. This state of affairs is unfortunate because it has been argued from diverse theoretical perspectives—including interpersonal theories of depression (e.g., Coyne, 1976)—that maladaptive interpersonal processes and depressive symptoms reciprocally reinforce one another across time. The present study focuses on the role of psychologically controlling parenting, as research has consistently demonstrated positive associations between this parenting dimension and child and adolescent internalizing problems (Barber & Harmon, 2002). Specifically, the central aim of the present study was to examine the possibility of reciprocal longitudinal associations between perceived parental psychological control and adolescents' depressive symptoms by using a cross-lagged longitudinal design.

### *Psychological Control and Adolescent Adjustment*

Psychological control is characteristic of parents who pressure their children to behave and think in accordance with parental goals and norms through internally controlling and manipulative means. Psychologically controlling parents engage in parenting tactics such as guilt induction, shaming, and conditional approval (Barber, 1996). Although psychological control may be expressed in a rather subtle and covert fashion (e.g., by giving a child the silent treatment), this parenting dimension is thought to have a detrimental impact on children's well-being (Barber & Harmon, 2002; Grolnick, 2003). Psychological control would frustrate children's need for autonomy (Vansteenkiste, Zhou, Lens, & Soenens, 2005) and as such create a vulnerability to maladjustment and to internalizing problems in particular (Barber & Harmon, 2002).

Various cross-sectional studies have shown that psychological control is related to internalizing problems in general and to depressive symptoms in particular (e.g., Barber, Olsen, & Shagle, 1994; Barber, 1996; Soenens, Vansteenkiste, Luyten, Duriez, & Goossens, 2005), even after controlling for other parenting dimensions such as responsiveness and behavioral con-

trol (e.g., Gray & Steinberg, 1999; Herman, Dornbusch, Herron, & Herting, 1997). Studies have demonstrated that the positive associations between psychological control and internalizing problems also hold in non-Western populations (e.g., Barber, Stolz, & Olsen, 2005; Vansteenkiste et al., 2005). Despite the consistency of these findings, the cross-sectional nature of most of these studies precludes any inference about the direction of effects. Although it is typically assumed that psychological control exerts an influence on adolescent adjustment and on depressive symptoms in particular, the possibility also exists that adolescents' adjustment influences parents' use of psychological control or that the relation between psychological control and adolescent adjustment is a reciprocal one.

### *Three Models on the Link between Psychological Control and Adjustment*

An examination of the direction of effects between psychological control and adolescent adjustment requires a longitudinal research design. Unfortunately, the few longitudinal studies that have been conducted to date did not reveal a coherent picture of the nature of these effects. Varying from study to study, evidence has been obtained for one of three models, that is, a psychological control effects model, an adolescent adjustment effects model, and a reciprocal model.

*Psychological control effects model.* In line with the idea that psychological control represents a risk factor for or an antecedent to adolescent maladjustment, some studies have demonstrated that psychological control prospectively predicts maladjustment. For instance, Steinberg, Elmen, and Mounts (1989) demonstrated that psychological control predicted a decrease in adolescents' school grades and psychosocial maturity scores over a one-year period. Using a similar design, Herman, Dornbusch, Herron, and Herting (1997) found that psychological control predicted increases in somatic (but not psychological) symptoms. Conger et al. (1997) found that psychological control predicted increases in internalizing and behavioral problems and decreases in self-confidence, albeit only among boys. Soucy and Larose (2000) found that paternal (but not maternal) psychological control predicted decreasing emotional and social adjustment to college as well as lower grades over the course of a semester.

Together, these studies suggest that psychological control exacerbates rather than simply accompanies adolescents' maladjustment. It should be noted, however, that the stability of psychological control was not controlled for in these studies. Each of these studies examined whether psychological control, as assessed at the onset of the study, predicted later adjustment,

thereby only controlling for earlier adjustment. Although such an analysis gives an indication of the relation between psychological control and over-time changes in adjustment, it does not allow drawing sound inferences about the direction of effects (Burkholder & Harlow, 2003). Any relationship between psychological control at Time 1 (T1) and adjustment at Time 2 (T2) may have been spuriously caused by the stability of psychological control from T1 to T2 and by a significant concurrent association between psychological control at T2 and adjustment at T2. Moreover, any design in which psychological control is only measured at T1 does not allow for an examination of effects of adolescent adjustment on subsequent parental use of psychological control. Hence, these studies did not actually consider the possibility that adolescent maladjustment elicits psychological control over time.

*Adolescent adjustment effects model.* In line with the growing recognition that parenting does not only affect child behavior but that children's behavior also serves to elicit particular parental reactions (e.g., Bell & Chapman, 1986; Stattin & Kerr, 2000), there are indications that aspects of children's earlier adjustment are predictive of parents' later use of psychological control. At least two studies have shown that adolescents' initial levels of problem behaviors predicted more perceived psychological control across time (Albrecht, Galambos, & Jansson, 2007; Rogers, Buchanan, & Winchell, 2003). In these studies, psychological control did not predict later problem behavior. These findings lend support to the notion that poor adjustment in adolescents—and internalizing problems in particular—may be a source of stress for parents, which makes them resort to intrusive parenting. The withdrawn and moody behavior of depressed adolescents may frustrate parental expectations for children's behavior and as such elicit more intrusive parental attempts to make their children behave according to parental goals. It is equally feasible, however, that depressive symptoms lead to biased perceptions of parents rather than to actual changes in parents' behavior, such that depressed adolescents merely view their parents as becoming increasingly controlling.

If perceptions of parental psychological control could be fully accounted for by adolescents' own depressive symptoms, this would imply that parental psychological control is simply a response to or concomitant of children's vulnerability—possibly dispositional—to depression. The adolescent adjustment effects model therefore contradicts Barber and Harmon's (2002) claim that psychological control is at least partly rooted in parents' own functioning and developmental history instead of being a mere consequence of the child's behavior. In line with Barber and Har-

mon's (2002) claim, research indicates that parental psychological control is significantly predicted by parental characteristics such as perfectionism and separation anxiety (Soenens, Vansteenkiste, Duriez, & Goossens, 2006). Given that parental characteristics account for a substantial part of the variability in psychological control, it is unlikely that only child effects would drive the link between psychological control and adjustment. A reciprocal model may provide a better description of this link.

*Reciprocal model.* Reciprocal models are favored within transactional models of socialization (e.g., Magnusson, 1988; Sameroff & Fiese, 2000). Transactional models consider developmental outcomes as the product of a continuous dynamic interaction between parents' and children's behavior and characteristics. With regard to psychological control, transactional theories would predict that parents of less adjusted adolescents would be more likely to rely on psychologically controlling strategies that, in turn, would further increase children's susceptibility to depression. Such a hypothesis is also in line with Coyne's (1976) interpersonal theory of depression, which assumes that depressive individuals' clinging and reassurance-seeking interpersonal behaviors lead to maladaptive and rejecting responses of others (including parents). In an escalating cycle, the depressed person's symptoms would further worsen as a result of parents' negative, intrusive, and critical responses.

To the best of our knowledge, few studies provided evidence for such transactional processes. In one of the most extensive longitudinal studies of psychological control to date, Barber, Stolz, and Olsen (2005) examined cross-lagged effects between psychological control and depression using a four-wave longitudinal design. Through structural equation modeling (SEM), the analyses controlled for stability in both psychological control and depression. Barber et al. (2005) found cross-lagged effects of psychological control on subsequent levels of depression as well as cross-lagged effects of depression on subsequent reports of psychological control. Although these findings seem indicative of a reciprocal relation between psychological control and depression, the study by Barber et al. (2005) did not control for within-time associations between psychological control and depression. In the models that were tested, each data wave included either psychological control (Waves 1 and 3) or depression (Waves 2 and 4). Although such a design allows controlling for prior levels of each construct (i.e., stability effects), it does not allow simultaneously controlling for associations between the constructs within each wave. As a consequence, the cross-lagged paths that were found may have been spuriously caused by the stability in each construct and the (nonobserved) association between the constructs within each wave.

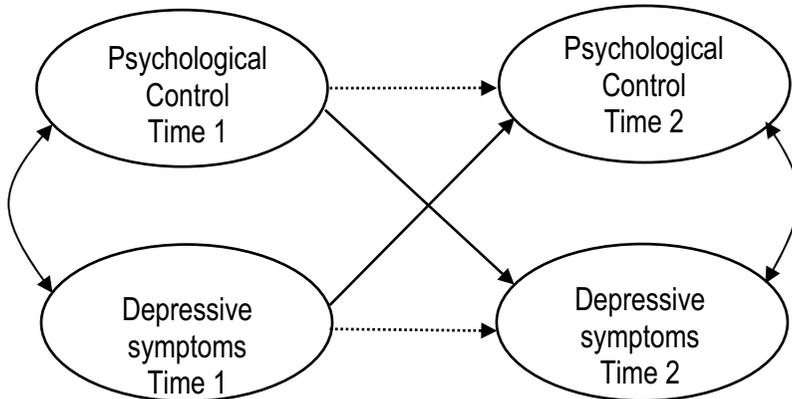
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*The Present Study*

The present study aims to examine the nature of the relation between perceived psychological control and depressive symptoms in two samples of late (Study 1) and middle (Study 2) adolescents. Our review of the extant literature shows that longitudinal studies on psychological control and maladjustment yielded inconsistent evidence. This may be due to a number of differences between studies such as a reliance on different age groups (e.g., middle adolescents versus college students), the diversity of outcomes that was studied (e.g., depressive symptoms versus adjustment to college), and the different spacing between time intervals (e.g., six months versus one year). Most importantly, however, the cited studies differ with respect to (a) the research design that was adopted and (b) the statistical approach to test for longitudinal effects. First, whereas some studies (e.g., Barber et al., 2005) assessed both psychological control and depressive symptoms at multiple measurement waves, other studies (e.g., Herman et al., 1997) measured psychological control only at the onset of the study. Contrary to the former type of study design, the latter approach precludes the possibility of finding reciprocal associations between parental control and depressive symptoms. Accordingly, in the present study both psychological control and adolescent depressive symptoms were systematically assessed at each measurement wave.

Second, previous studies differed in the extent to which they controlled for stability in psychological control and the adolescent outcomes as well as for the within-time correlations between psychological control and adolescent adjustment. To the best of our knowledge, none of the longitudinal studies on psychological control simultaneously controlled (a) for stability in both psychological control and adolescents' adjustment and (b) for within-time associations between psychological control and adjustment. Failing to control for such potential confounds may spuriously inflate the estimates of the cross-lagged paths. The present study relied on a full cross-lagged longitudinal design with annual assessments of both psychological control and depression, as suggested by Rueter and Conger (1998) and Burkholder and Harlow (2003). Figure 1 depicts the conceptual model of this study. As shown in Figure 1, the cross-lagged paths were estimated controlling for both autoregressive (stability) effects and cross-sectional covariances.

The design of this study thus allows for a direct comparison between the three proposed models. The psychological control effects model would receive support if, besides the autoregressive paths and the cross-sectional covariances, only the cross-lagged paths from perceived psychological control to adolescent depression would be significant. Conversely, evidence for



**Figure 1.** Conceptual model of the associations between perceived parental psychological control and adolescent depression. Whereas the dashed arrows depict autoregressive effects, the full arrows depict cross-lagged paths.

the adolescent adjustment effects model would be obtained if only the cross-lagged paths from adolescent depression to perceived psychological control reached significance. Finally, the reciprocal model would be evidenced by significant cross-lagged paths in both directions. Based on transactional theories of development, we hypothesize that the relation between perceived psychological control and adolescents' depressive symptoms is most likely to be a reciprocal one.

In addition to the general aim of examining cross-lagged relations between perceived psychological control and depressive symptoms, we aimed to examine a number of additional issues, including (a) the unique value of the parenting dimension of psychological control in predicting depressive symptoms (i.e., controlling for responsiveness and behavioral control), (b) the consistency of longitudinal relations between psychological control and depressive symptoms across age, and (c) the possible moderating role of parent and adolescent gender in these relations. These three issues are considered in greater detail below.

*Unique predictive value of psychological control.* We aimed to establish whether effects of perceived psychological control on adolescent depressive symptoms, if any, can be attributed specifically to the influence of psychological control. More specifically, Study 1 examined whether perceived psychological control would predict depressive symptoms after controlling for the effects of two other crucial parenting dimensions, namely responsiveness and behavioral control (Gray & Steinberg, 1999). Responsiveness refers to the

degree to which adolescents experience a positive, involved, and warm relationship with their parents, and behavioral control involves the provision of sufficient regulation of children's behavior. Although cross-sectional studies have provided quite consistent evidence for a specialized relation between psychological control and depressive symptoms (e.g., Barber et al., 1994), the present study is among the first to assess this specialized effect using a longitudinal framework (but see Barber et al., 2005).

*Age differences.* We deemed it important to examine the hypothesized longitudinal associations between perceived psychological control and depressive symptoms in two different age groups, that is, middle and late adolescence. It could be argued that the relationship between manipulative and intrusive parental behaviors and depressive symptomatology will decrease as adolescents grow older because the influence of nonparental adults (e.g., teachers and mentors), peers, and romantic partners becomes increasingly important (Steinberg & Silk, 2002). Conversely, one could argue that with increasing age, adolescents' representations of their parents' behavior become increasingly stable and internalized such that these representations continue to affect adolescents' functioning in late adolescence (e.g., Soucy & Larose, 2000). Other theories also assume that associations between parental control and maladjustment are not age-bound. Self-determination theory (SDT) (Deci & Ryan, 2000), for instance, claims that (representations of) inconsistent and controlling parenting will detract from optimal functioning at any given age because such parenting would frustrate the satisfaction of an innate need for autonomy that is essential to optimal functioning across the life span (Grolnick, 2003). In keeping with such reasoning, we could also expect parents to react to adolescents' depressive symptoms with increased control irrespective of adolescents' age because the underlying process that is assumed to trigger parental control (e.g., anxiety and worry about the adolescent's development) is likely to be the same across age.

The present study examines the longitudinal associations between psychological control and depressive symptoms in a sample of late adolescents (i.e., college students; Study 1) and in a sample of middle adolescents (i.e., high school students; Study 2). On the basis of SDT and transactional theories of development, we hypothesize that reciprocal relations between psychological control and depressive symptoms will be found in both samples.

*Gender differences.* Past studies have found small but significant gender differences in psychological control, with males typically reporting somewhat higher levels of psychological control than females (Barber, Bean, & Erikson, 2002). Gender differences are also typically found in depression, with females obtaining higher scores than males (e.g., Galambos, Leadbeater, & Barker, 2004; Leadbeater, Kuperminc, Blatt, & Herzog,

1999). For this reason, we controlled for the possibly confounding influence of adolescent gender in all analyses.

Furthermore, it is important to assess whether the hypothesized longitudinal structural relationships vary by adolescent and parent gender. Rogers et al. (2003) performed the most explicit and detailed examination of this issue to date and hypothesized that the association between psychological control and depressive symptoms would be most pronounced in mother-daughter dyads because mother-daughter relationships have been found to be particularly emotionally intense in regard to both closeness and conflict. However, Rogers et al. did not obtain clear-cut evidence for any of these hypotheses. Similarly, Barber et al. (2005) found that the reciprocal associations between psychological control and depression were generally consistent across adolescent gender. However, the latter study did not formally test for gender differences (e.g., through multigroup analysis). Given the paucity of research on this topic, the present study aimed to contribute to the literature by further evaluating the possible moderating role of gender in Study 2. In sum, we will control for mean-level gender differences in the primary analyses of both Study 1 and Study 2. In Study 2 we will additionally examine gender as a moderating variable in a separate set of analyses.

## Study 1

The aims of Study 1 were (a) to compare the three models of longitudinal associations between perceived psychological control and depressive symptoms using a three-wave cross-lagged design and (b) to assess the unique predictive value of perceived psychological control relative to the two other fundamental parenting dimensions (i.e., responsiveness and behavioral control). These research objectives were addressed in a sample of late adolescents (i.e., college students).

## Method

### *Participants and Procedure*

The data for this study were collected at a large university in Belgium (Europe) in the context of a larger longitudinal project on identity development (Luyckx, Goossens, & Soenens, 2006). The first wave of this study was conducted at the end of 2002. At T1, all participants were freshmen from the Faculty of Psychology and Educational Sciences, consisting of a predominantly female student population. This sample comprised 565 students consisting of 482 women (85.3%). Mean age was 18 years and 8 months (SD

= 7.6 months). These participants were followed with two biannual measurements each year (one in the fall semester and one in the spring semester). Data for the present essay are taken from the first, third, and fifth measurement waves because the measurements of interest to this study were only administered at these three time points. These three measurement waves were one year apart.

Approximately 70% of the initial sample participated in each of the three measurement waves. This longitudinal sample of 396 participants was the sample of interest and consisted of 351 women (88.6%). Eighty-four percent of the participants lived in an intact family with parents being married and/or living together. Thirteen percent had parents being divorced, and 3% had one deceased parent. It is important to note that the large majority of university students in Belgium (i.e., >95%) still live with parents (i.e., commuters) or return home every week during the weekends (see Luyckx et al., 2006). Hence, with few exceptions, Belgian university students still live with their parents and have frequent contact with them.

A logistic regression analysis tested whether sample attrition (dummy coded as dropout = 0 and retention = 1) was predicted by age, gender (dummy coded as female = 0 and male = 1), and all study variables at T1. Age and gender were entered in Step 1. The three parenting dimensions and depression were entered in Step 2. Model  $\chi^2$  for Step 1 was significant ( $\chi^2[2] = 19.56, p < .01$ ). Retention was significantly predicted by being female (odds ratio [OR] = .51,  $p < .01$ ) and being younger (OR = .65,  $p < .01$ ). Step 2 did not significantly add to the multivariate prediction of retention ( $\chi^2[4] = 8.77, p > .05$ ). In short, students who participated at all three waves were more likely to be female and younger than those who participated at only one or two waves, but no substantial differences were found on any of the study variables at T1, demonstrating the aselectivity of our longitudinal sample compared to the initial sample.

Permission to undertake this study was granted by the ethical commission of the researchers' department. The adolescents signed an informed consent form before answering the questionnaire at the first occasion and were informed that they could refuse or discontinue participation at any time. Confidentiality was guaranteed. At each measurement occasion, questionnaires were distributed in lecture halls or by mail, and participants were asked to complete the questionnaires as soon as possible.

### *Measures*

*Parenting style.* Participants completed 21 items derived from the Children's Report on Parent Behavior Inventory (CRPBI) (Schaefer, 1965; Soe-

nens, Vansteenkiste, et al., 2005) and rated these for both parents together on a scale ranging from 1 (totally disagree) to 5 (totally agree). Cronbach's alphas for the psychological control scale (7 items, e.g., "My parents are less friendly to me if I don't see things like they do") were .82, .85, and .86 at T1, T2, and Time 3 (T3), respectively. Past studies have shown that this scale is distinct from other parenting dimensions such as responsiveness and behavioral control yet relates in theoretically predicted ways to these dimensions (e.g., Beyers & Goossens, 1999). Cronbach's alphas for the responsiveness scale (7 items, e.g., "My parents make me feel better after I discussed my worries with them") were .91, .90, and .91 at T1, T2, and T3, respectively. Cronbach's alphas for the behavioral control scale (7 items, e.g., "My parents allow me to do anything I want," reverse coded) were .81, .83, and .84 at T1, T2, and T3, respectively.

*Depressive symptoms.* The Centre for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977) aims to measure depressive symptoms in the general population. As such, it does not provide a clinical diagnosis of depression. Roberts, Lewinsohn, and Seeley (1991) have shown that the CES-D is a reliable and valid instrument to assess depressive symptoms in samples of adolescents. A brief 12-item version of the original 20-item CES-D was developed by Roberts and Sobhan (1992), who found a correlation of .96 between the brief version and the full version of the CES-D. Research with the Dutch translation of the CES-D has demonstrated good reliability and validity as well, both for the 20-item CES-D (Bouma, Ranchor, Sanderman, & van Sonderen, 1995) and the 12-item CES-D (Hooge, Decaluwé, & Goossens, 2000). In this study, we used the 12-item CES-D.

Participants indicated how often they experienced 12 depressive symptoms during the previous week. Ratings were made on a scale ranging from 0 to 3 (0 = rarely or none of the time [less than one day], 1 = a couple of times [1–2 days], 2 = sometimes or regularly [3–4 days], and 3 = most or all of the time [5–7 days]). For each individual, a total depressive symptoms score was calculated by summing the responses. Accordingly, scores could range from 0 to 36. In the current sample, observed scores ranged between 0 and 30 at T1, between 0 and 35 at T2, and between 0 and 34 at T3. Cronbach's alphas were .88, .87, and .88 at T1, T2, and T3, respectively.

To provide a more detailed picture of the distribution of depression scores in this sample, we grouped our participants into three categories according to cutoff scores recently developed by Poulin, Hand, Boudreau, and Santor (2005): (a) minimal depressive symptoms (scores 0–11), (b) somewhat elevated depressive symptoms (scores 12–20), and (c) very elevated depressive symptoms (scores 21–36). Across the three waves, between 69.9% and 76.3% of the participants were in the "minimal"

category, between 17.7% and 24.2% were in the “somewhat elevated” category, and between 5.6% and 6.6% were in the “very elevated” category. It should be mentioned that Poulin et al. (2005) did not use the validated brief CES-D used in this study to develop these cutoff scores. Hence, these percentages should be interpreted with caution. Still, it is clear that although the distribution of depressive symptoms is positively skewed, there is substantial variability in depressive symptoms in this sample.

## Results

### *Preliminary Analyses and Descriptive Statistics*

Correlations among the study variables are presented in Table 1. Stability coefficients of the three parenting constructs were higher (ranging from .67 to .79) compared to stability coefficients of depressive symptoms (ranging from .38 to .48). As expected, psychological control was positively correlated with depressive symptoms both across and within measurement waves. In contrast, parental responsiveness was generally negatively correlated with depressive symptoms. Correlations between behavioral control and depressive symptoms, if any, were slightly positive.

The means and standard deviations of the study variables are presented in Table 1. In order to assess gender differences and mean-level changes in the three parenting constructs and in adolescents' depressive symptoms, a repeated measures ANOVA was performed with gender as a between-subjects variable, measurement time as a within-subjects variable, and each of the study variables as dependent variables. A significant gender difference was obtained in responsiveness ( $F[1, 394] = 15.05, p < .01$ ). At all three waves, female participants reported higher levels of responsiveness (first wave,  $M = 3.77, SD = .83$ ; third wave,  $M = 3.74, SD = .77$ ; fifth wave,  $M = 3.68, SD = .78$ ) than did male participants (first wave,  $M = 3.34, SD = .74$ ; third wave,  $M = 3.39, SD = .69$ ; fifth wave,  $M = 3.40, SD = .64$ ). No other gender differences were observed. No mean-level changes were observed in responsiveness ( $F[2, 788] = .21, p > .05$ ), psychological control ( $F[2, 788] = 2.52, p > .05$ ), and depressive symptoms ( $F[2, 788] = 1.98, p > .05$ ). By contrast, behavioral control was found to linearly decrease across the three measurement points ( $F[2, 788] = 35.53, p < .01$ ), respectively. No time  $\times$  gender interactions were significant.

### *Primary Analyses*

SEM with latent variables was used to examine the study hypotheses. Analysis of the covariance matrices was conducted using LISREL 8.54

**Table 1.** Means, Standard Deviations, and Correlations among All Study Variables (Study 1)

Scale	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	M	SD
1. Psychological control T1	.70***	.67***	-.64***	-.54***	-.51***	.38***	.24***	.31***	.37***	.39***	.27***	1.94	.63
2. Psychological control T2	-	.73***	-.51***	-.60***	-.51***	.32***	.29***	.34***	.36***	.39***	.37***	1.94	.65
3. Psychological control T3	-	-	-.48***	-.49***	-.62***	.34***	.27***	.41***	.32***	.42***	.45***	1.98	.68
4. Responsiveness T1	-	-	-	.78***	.74***	-.19***	-.04	-.08	-.29***	-.29***	-.18***	3.72	.84
5. Responsiveness T2	-	-	-	-	.79***	-.11*	-.08	-.10	-.22***	-.33***	-.20***	3.70	.77
6. Responsiveness T3	-	-	-	-	-	-.19***	-.09	-.21***	-.25***	-.34***	-.31***	3.64	.77
7. Behavioral control T1	-	-	-	-	-	-	.74***	.70***	.09	.17**	.16**	3.12	.69
8. Behavioral control T2	-	-	-	-	-	-	-	.77***	.04	.09	.09	2.93	.70
9. Behavioral control T3	-	-	-	-	-	-	-	-	.09	.18***	.17**	2.81	.72
10. Depressive symptoms T1	-	-	-	-	-	-	-	-	-	.43***	.38***	9.45	6.16
11. Depressive symptoms T2	-	-	-	-	-	-	-	-	-	-	.48***	8.51	6.00
12. Depressive symptoms T3	-	-	-	-	-	-	-	-	-	-	-	8.70	6.16

Note. T1 = Time 1, T2 = Time 2, T3 = Time 3.

\*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ .

(Jöreskog & Sörbom, 1996), and solutions were generated on the basis of maximum-likelihood estimation. In the analyses, each construct was represented by parcels rather than by individual scale items. Parceling has several advantages in the modeling of latent variables, relative to the use of individual items. Parcels are likely to have a stronger relationship to the latent variable, are less likely to be affected by method effects, and are more likely to meet assumptions of normality (Marsh, Hau, Balla, & Grayson, 1998). Three randomly created parcels were computed for each construct, and the same parceling procedure was used to represent the constructs at the three measurement points. Several fit indices were used to evaluate the models. The Satorra-Bentler Scaled chi-square statistic (SBS- $\chi^2$ ) (Satorra & Bentler, 1994) should be as small as possible. The Root Mean Square Error of Approximation (RMSEA) should be less than .06, and the Comparative Fit Index (CFI) should exceed .95 (Hu & Bentler, 1999).

*Psychological control and depressive symptoms.* A first set of models tested longitudinal associations between psychological control and adolescents' depressive symptoms. Before testing the structural models, we conducted a Confirmatory Factor Analysis (CFA) to assess the longitudinal invariance of the measurement model. The baseline model without invariance constraints included six latent variables (i.e., psychological control at three measurement points and depressive symptoms at three measurement points) and 18 observed indicators (i.e., parcels). The measurement errors of the same indicators at different measurement points were allowed to covary (Burkholder & Harlow, 2003). This model showed an adequate fit to the data (SBS- $\chi^2$ [120] = 250.96, CFI = .98, RMSEA = .05). Next, a model was estimated in which the factor loadings were set equivalent across the three measurement points. Compared to the model with freely varying factor loadings, the latter model did not result in a significant loss in model fit (SBS- $\chi^2$ diff[8] = 14.65,  $p > .05$ ), indicating that the measurement model was equivalent across measurement waves. Moreover, all constrained factor loadings were highly significant ( $p < .001$ ), ranging from .66 to .89 (mean  $\lambda = .83$ ). In sum, evidence was obtained for a reliable and longitudinally invariant measurement model, which was used in all subsequent tests of the structural models.

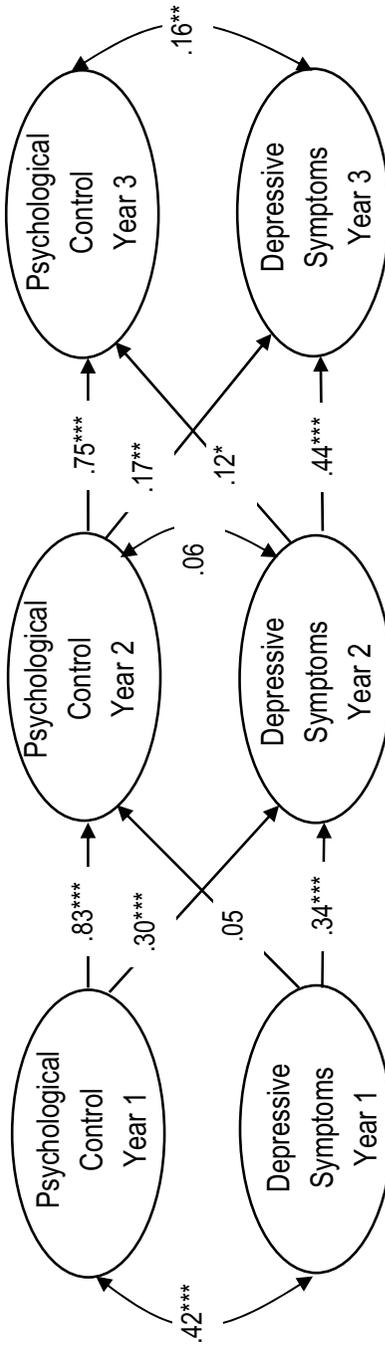
In a first step, we estimated a baseline autoregressive model that specified only autoregressive effects and within-time correlations between psychological control and depressive symptoms. As such, this model assumes that cross-lagged effects do not exist. This model (as well as all subsequent models) included gender as a control variable by allowing paths from gender to each of the six latent constructs. The baseline autoregressive model yielded an acceptable fit (SBS- $\chi^2$ [136] = 367.99, CFI = .97, RMSEA =

.07). In a second step, we estimated the two unidirectional cross-lagged models, that is, the psychological control effects model and the adolescent adjustment effects model. The psychological control effects model (SBS- $\chi^2[134] = 325.46$ , CFI = .98, RMSEA = .06) provided a better fit to the data compared to the baseline autoregressive model (SBS- $\chi^2\text{diff}[2] = 42.76$ ,  $p < .001$ ). Similarly, the adolescent adjustment effect model (SBS- $\chi^2[134] = 350.28$ , CFI = .98, RMSEA = .06) better fitted the data compared to the baseline autoregressive model (SBS- $\chi^2\text{diff}[2] = 17.82$ ,  $p < .001$ ).

In a third and final step, we estimated the reciprocal model that specifies both cross-lagged paths from psychological control to depressive symptoms and vice versa. The reciprocal model (SBS- $\chi^2[132] = 315.85$ , CFI = .98, RMSEA = .06) was found to provide a better fit to the data than either the psychological control effects model (SBS- $\chi^2\text{diff}[2] = 9.31$ ,  $p < .01$ ) or the adolescent adjustment effects model (SBS- $\chi^2\text{diff}[2] = 32.48$ ,  $p < .001$ ). This final model is depicted in Figure 2 and shows that all but one of the cross-lagged structural paths is significant. Whereas the two cross-lagged paths from psychological control to depressive symptoms are significant ( $\beta$ s = .30 and .17,  $p < .01$ , respectively), only the path from depressive symptoms at T2 to psychological control at T3 is significant ( $\beta = .12$ ,  $p < .05$ ), but not the path from depressive symptoms at T1 to psychological control at T2.

As suggested by Burkholder and Harlow (2003), we also tested whether the reciprocal associations between psychological control and depressive symptoms would hold across a two-year interval. This was done by including only the latent constructs of psychological control and depressive symptoms at T1 and T3. It was found that whereas the cross-lagged path from psychological control to depressive symptoms was still significant ( $\beta = .16$ ,  $p < .05$ ), the path from depressive symptoms at T1 to psychological control at T3 was not significant ( $\beta = .04$ ,  $p > .05$ ).

*Unique predictive value of psychological control.* In an additional set of analyses we examined whether psychological control is longitudinally predictive of depressive symptoms after controlling for the effects of parental responsiveness and behavioral control. To this aim, in addition to the paths specified in Figure 2, cross-lagged paths were specified from responsiveness and behavioral control at T1 to depressive symptoms at T2 and from responsiveness and behavioral control at T2 to depressive symptoms at T3. The model also controlled for stability in responsiveness and behavioral control and for within-time associations with both psychological control and depressive symptoms. As in the previous set of models, the factor loadings of the indicators on their respective latent factors were set invariant across the three measurement waves. Estimation of the model



**Figure 2.** Structural model of the associations between perceived parental psychological control and adolescent depression (Study 1). The coefficients shown are standardized path coefficients. For the sake of clarity, the effects of gender are not shown.  
 Note: \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ .

with responsiveness and behavioral control as additional predictors of adolescents' depressive symptoms yielded an adequate fit ( $SBS-\chi^2[577] = 1018.62$ ,  $CFI = .98$ ,  $RMSEA = .04$ ). Whereas the two cross-lagged paths from psychological control to depressive symptoms remained significant ( $\beta = .29$ ,  $p < .01$  and  $\beta = .29$ ,  $p < .01$ , respectively), none of the cross-lagged paths from responsiveness ( $\beta = .02$ ,  $p > .05$  and  $\beta = .11$ ,  $p > .05$ , respectively) or behavioral control ( $\beta = .04$ ,  $p > .05$  and  $\beta = -.04$ ,  $p > .05$ , respectively) to depressive symptoms reached significance.<sup>1</sup>

## Brief Discussion

The results of this study generally favor the reciprocal relationships model, suggesting that perceived parental psychological control and late adolescents' depressive symptoms mutually reinforce one another. Psychological control predicted increases in college students' depressive symptoms between Year 1 and Year 2, between Year 2 and Year 3, and even between Year 1 and Year 3. Moreover, the effects of psychological control on depressive symptoms were found to remain significant after controlling for the effects of two other fundamental parenting dimensions, namely responsiveness and behavioral control. Second, college students' level of depressive symptoms additionally predicted an increase in perceived parental psychological control, although this effect was only obtained between Year 2 and Year 3.

## Study 2

In Study 2, we aimed (a) to further test whether the relationship between perceived psychological control and adolescent depressive symptoms is a reciprocal one using a two-wave cross-lagged design and (b) to explore a number of additional issues that could not be addressed in Study 1. First, as

<sup>1</sup> Within a configurational approach to parenting, it is assumed that the effect of one parenting dimension cannot be considered in isolation from other parenting dimensions (Maccoby & Martin, 1983). It is possible, for instance, that the longitudinal effect of psychological control is moderated by the effect of parental responsiveness. Moderated regression analyses were therefore used to examine whether the parenting dimensions would interact in the prediction of depressive symptoms. In a first regression analysis, depressive symptoms at T2 were regressed on gender and depressive symptoms at T1 (Step 1), the main effects of the parenting constructs at T1 (Step 2), and each of the possible interaction effects between the T1 parenting constructs (Step 3). This analysis was then repeated with depressive symptoms at T3 as the dependent variable and the T2 control variables and parenting dimensions as predictors. None of the interaction terms reached significance (all  $ps < .05$ ), indicating that the main effects of psychological control on future depressive symptoms were not moderated by responsiveness or behavioral control.

Study 1 involved a sample of late adolescents, it is important to examine whether these findings generalize to younger adolescents. Study 2 therefore samples middle adolescents. Second, because the sample of Study 1 was predominantly female, gender differences could not be adequately assessed. Therefore, we sampled a more balanced sample with regard to adolescent gender, which additionally allowed us to examine the moderating role of adolescent gender. Third, whereas Study 1 assessed the overall level of perceived psychological control used by both parents, Study 2 included separate assessments of maternal and paternal psychological control to examine whether the hypothesized reciprocal model replicates across maternal and paternal ratings of psychological control.

## Method

### *Participants and Procedure*

The data for this study were collected in six secondary schools in Flanders (Belgium). The first wave of this study was conducted at the end of 2004. At T1, all participants were in the 10th grade. All students attended a regular high school (academic track). The initial sample consisted of 441 females (49%) and 463 males (51%). Mean age of the participants at the onset of the study was 14.94 years ( $SD = .50$ ). The second measurement wave took place one year later. A total of 724 students (i.e., 80% of the initial sample) participated in the second wave. This longitudinal sample was the sample of interest and consisted of 368 females (51%) and 356 males (49%). A total of 84% of the participants lived in an intact family with parents being married and/or living together, 13% had parents who were divorced, and 3% had at least one deceased parent. Active informed consent was obtained from the participants, and questionnaires were administered during a class period. Anonymity was guaranteed, and participation was voluntary. Students had approximately 45 minutes to complete the survey.

A logistic regression analysis tested whether sample attrition (dummy coded as dropout = 0 and retention = 1) was predicted by age, gender (dummy coded as female = 0 and male = 1), and all study variables at T1. Age and gender were entered in Step 1. The three parenting dimensions and depression were entered in Step 2. Model  $\chi^2$  for Step 1 was significant ( $\chi^2[2] = 7.40, p < .05$ ). Retention was significantly predicted by being younger of age ( $OR = .50, p < .01$ ) but not by gender. Step 2 did not significantly add to the multivariate prediction of retention ( $\chi^2[3] = 2.11, p > .05$ ). In short, as in Study 1, no substantial differences emerged between those who participated and those who dropped out at T1.

## Measures

*Psychological control.* Participants completed a six-item version of the same psychological control scale that was used in Study 1. One item was dropped from the original seven-item scale because it had a low loading on the underlying factor in previous research (Soenens, Vansteenkiste, et al., 2005). Cronbach's alphas for paternal and maternal ratings of psychological control were .82 and .79 and .82 and .79 at T1 and T2, respectively.

*Depressive symptoms.* Participants completed the 12-item version of the Center for Epidemiologic Studies Depression Scale (CESD; Radloff, 1977), which was also used in Study 1. Observed scores ranged between 0 and 36 at T1 and between 0 and 31 at T2. Cronbach's alphas were .88 and .87 at T1 and T2, respectively. A total of 69.3% and 68.5% of the participants were in the "minimal depressive symptoms" category at T1 and T2, respectively; 24.3% and 24.0% were in the "somewhat elevated depressive symptoms" category at T1 and T2, respectively; and 6.4% and 7.5% were in the "very elevated depressive symptoms" category at T1 and T2, respectively.

## Results

### *Preliminary Analyses and Descriptive Statistics*

As shown in Table 2, all correlations among maternal psychological control, paternal psychological control, and depressive symptoms within and across time points were positive and significant. As in Study 1, a repeated measures ANOVA was performed with gender as a between-subjects variable, measurement time as a within-subjects variable, and each of the study variables as dependent variables. A significant gender difference was obtained in maternal psychological control ( $F[1, 659] = 7.77, p < .01$ ). Males reported higher levels of maternal psychological control (T1,  $M = 2.21, SD = .84$ ; T2,  $M = 2.50, SD = .86$ ) than did females (T1,  $M = 2.12, SD = .84$ ; T2,  $M = 2.27, SD = .87$ ). No other gender differences were observed. No mean-level changes were observed in depressive symptoms ( $F[1, 659] = .66, p > .05$ ). By contrast, both maternal psychological control ( $F[1, 659] = 16.50, p < .01$ ) and paternal psychological control ( $F[1, 659] = 16.46, p < .01$ ) were found to increase between T1 (maternal psychological control,  $M = 2.16, SD = .84$ ; paternal psychological control,  $M = 2.05, SD = .97$ ) and T2 (maternal psychological control,  $M = 2.38, SD = .87$ ; paternal psychological control,  $M = 2.27, SD = .79$ ). Time and gender significantly interacted in the prediction of maternal psychological control. Follow-up

**Table 2.** Means, Standard Deviations, and Correlations among All Study Variables (Study 2)

Scale	1.	2.	3.	4.	5.	6.	M	SD
1. Paternal psychological control T1	—	.44*	.26*	.57*	.27*	.25*	2.05	.79
2. Maternal psychological control T1		—	.21*	.26*	.53*	.15*	2.16	.84
3. Depressive symptoms T1			—	.27*	.20*	.49*	9.58	6.20
4. Paternal psychological control T2				—	.39*	.31*	2.27	.74
5. Maternal psychological control T2					—	.24*	2.38	.78
6. Depressive symptoms T2						—	9.58	6.27

Note. T1 = Time 1, T2 = Time 2.

\*  $p < .001$ .

analyses revealed that the increase in perceived maternal psychological control was more pronounced in males than in females. No other interactions between time and gender were significant.

### *Primary Analyses*

As in Study 1, we estimated SEM models with latent variables to examine the study hypotheses. Again, we used parceling to create three observed indicators for each construct in the estimated models (i.e., psychological control and depressive symptoms). The same parceling procedure was used to represent the constructs at the two measurement points.

Analyses were performed separately for maternal and paternal ratings of psychological control. Furthermore, to examine the possible moderating role of adolescent gender, multigroup analyses were performed to compare results for male and female adolescents. Multigroup analysis compares a constrained model (i.e., a model in which the structural coefficients are set equal across gender) to an unconstrained model (i.e., a model in which these coefficients are allowed to vary across gender). Models are compared in terms of the chi-square difference corresponding to the number of degrees of freedom. A significant difference implies that the model differs significantly across gender.

*Paternal psychological control and depressive symptoms.* A first set of models tested longitudinal associations between paternal psychological control and adolescents' depressive symptoms. First, a CFA was performed to test whether the measurement model would be invariant across the two measurement waves and across adolescent gender. This measurement model contained four latent constructs (psychological control at T1 and T2 and depressive symptoms at T1 and T2), each indicated by three parcels. Initially, we tested a measurement model without invariance constraints; that is, factor loadings were freely estimated across the two measurement occasions and across gender. The measurement errors of the same indicators at different measurement points were allowed to be correlated. Estimation of this measurement model yielded an acceptable fit ( $SBS-\chi^2[100] = 127.44$ ,  $CFI = .99$ ,  $RMSEA = .03$ ). Constraining the factor loadings to be invariant across measurement waves or across adolescent gender did not significantly worsen model fit ( $SBS-\chi^2\text{diff}[8] = 16.61$ ,  $p > .05$  and  $SBS-\chi^2\text{diff}[4] = 2.16$ ,  $p > .05$ , respectively), indicating that the measurement model was invariant across measurement waves and across gender. In the final constrained measurement model, all factor loadings were highly significant ( $p < .001$ , mean  $\lambda = .79$ ). These findings suggest that the constructs

of psychological control and depressive symptoms had the same meaning across measurement occasions and for both male and female adolescents.

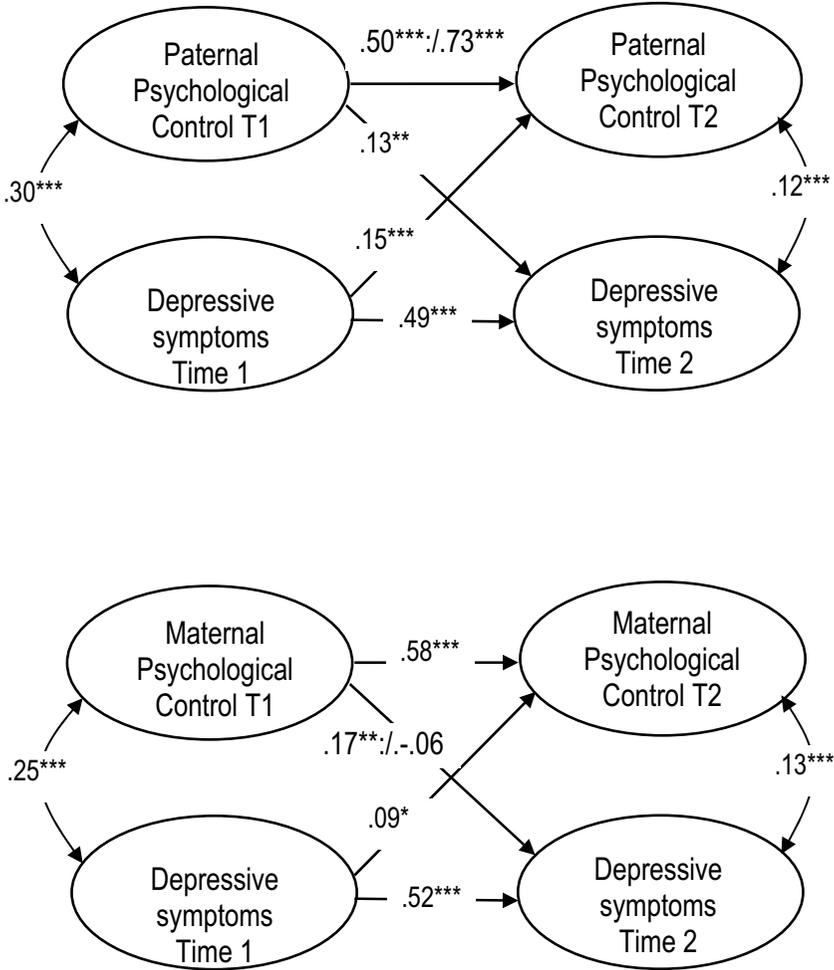
Next, we tested the four structural models of longitudinal associations between paternal psychological control and adolescent depressive symptoms. Initially, these models were tested as constrained models; that is, the structural paths were assumed to be equal for males and females. First, the baseline autoregressive model had an acceptable fit to the data ( $SBS-\chi^2[114] = 172.60$ ,  $CFI = .99$ ,  $RMSEA = .04$ ). However, both the psychological control effects model ( $SBS-\chi^2[113] = 165.74$ ,  $CFI = .99$ ,  $RMSEA = .04$ ) and the adolescent adjustment effect model ( $SBS-\chi^2[113] = 161.51$ ,  $CFI = .99$ ,  $RMSEA = .04$ ) provided a better fit to the data in comparison to the baseline autoregressive model ( $SBS-\chi^2diff[1] = 6.86$ ,  $p < .01$  and  $SBS-\chi^2diff[1] = 11.09$ ,  $p < .01$ , respectively). The reciprocal effects model ( $SBS-\chi^2[112] = 155.66$ ,  $CFI = .99$ ,  $RMSEA = .03$ ), however, provided an even better fit in comparison to either the psychological control effects model ( $SBS-\chi^2diff[1] = 10.08$ ,  $p < .01$ ) or the adolescent adjustment effects model ( $SBS-\chi^2diff[1] = 5.85$ ,  $p < .01$ ), indicating that a reciprocal model provides the best representation of the longitudinal associations between paternal psychological control and adolescent depressive symptoms. In this final model, both the effect of paternal psychological control T1 on depressive symptoms T2 ( $\beta = .13$ ,  $p < .01$ ) and the effect of depressive symptoms T2 on paternal psychological control ( $\beta = .14$ ,  $p < .01$ ) were significant, even when controlling for the stability in paternal psychological control ( $\beta = .62$ ,  $p < .01$ ) and depressive symptoms ( $\beta = .48$ ,  $p < .01$ ) and for the within-time associations between both constructs at T1 ( $r = .30$ ,  $p < .01$ ) and T2 ( $r = .12$ ,  $p < .01$ ).

To test whether this best-fitting model is invariant across adolescent gender, the constrained reciprocal effects model was compared to an unconstrained model in which the structural paths of the model (i.e., the two stability coefficients and the two cross-lagged paths) were set free across adolescent gender. A model in which the stability coefficients were set free across gender fit the data somewhat better than the constrained model ( $SBS-\chi^2diff[2] = 7.81$ ,  $p < .01$ ). This was due to a significant difference in the stability coefficient of paternal psychological control that was more pronounced in females ( $\beta = .73$ ,  $p < .01$ ) than in males ( $\beta = .49$ ,  $p < .01$ ). However, a model in which the two central cross-lagged paths between psychological control and depressive symptoms were allowed to vary by gender did not fit the data better than the constrained model ( $SBS-\chi^2diff[2] = .08$ ,  $p > .05$ ), indicating that paternal psychological control and depressive symptoms are reciprocally related in both male and female adolescents. The final model for paternal psychological control is depicted in Figure 3.

*Maternal psychological control and depressive symptoms.* As for the maternal ratings, we performed a series of analyses similar to the paternal ratings. Initial estimation of a model without invariance constraints yielded an acceptable fit ( $SBS-\chi^2[100] = 131.57$ , CFI = .99, RMSEA = .03). Constraining the factor loadings across measurement waves or across adolescent gender did not significantly worsen model fit ( $SBS-\chi^2\text{diff}[8] = 13.94$ ,  $p > .05$  and  $SBS-\chi^2\text{diff}[4] = 1.67$ ,  $p > .05$ , respectively), indicating that the measurement model for maternal ratings was invariant across measurement waves and across gender. All factor loadings ( $M = .80$ ) of this constrained model were highly significant ( $p < .001$ ).

Next, the four hypothesized structural models were again tested. Initially, the structural parameters of the models were constrained across adolescent gender. The baseline autoregressive model had an acceptable fit to the data ( $SBS-\chi^2[114] = 150.63$ , CFI = .99, RMSEA = .03). Unexpectedly, the psychological control effects model ( $SBS-\chi^2[113] = 150.35$ , CFI = .99, RMSEA = .03) did not provide a comparatively better fit to the data ( $SBS-\chi^2\text{diff}[1] = .28$ ,  $p > .05$ ). The adolescent adjustment effect model ( $SBS-\chi^2[113] = 146.20$ , CFI = .99, RMSEA = .03), however, did fit the data better compared to the baseline autoregressive model ( $SBS-\chi^2\text{diff}[1] = 4.43$ ,  $p < .05$ ). The reciprocal effects model ( $SBS-\chi^2[112] = 146.04$ , CFI = .99, RMSEA = .03), finally, provided a better fit to the data in comparison to the psychological control effects model ( $SBS-\chi^2\text{diff}[1] = 4.31$ ,  $p < .05$ ) but not in comparison to the adolescent adjustment effects model ( $SBS-\chi^2\text{diff}[1] = .16$ ,  $p < .05$ ). These findings suggest that the adolescent adjustment effects model is the best fitting and most parsimonious model for the maternal data, at least when the structural paths are set equal for male and female adolescents. In this model, adolescent depression T1 positively predicted psychological control T2 ( $\beta = .09$ ,  $p < .05$ ) after controlling for the stability in maternal psychological control ( $\beta = .58$ ,  $p < .01$ ) and depressive symptoms ( $\beta = .54$ ,  $p < .01$ ) as well as for the within-time associations between both constructs at T1 ( $r = .26$ ,  $p < .01$ ) and T2 ( $r = .13$ ,  $p < .01$ ).

A multigroup analysis was conducted to examine gender differences in longitudinal associations between maternal psychological control and depressive symptoms. This analysis was conducted on the reciprocal effects model because this model contains all possible structural paths between the constructs. Although we did not find a significant difference between both models regarding the stability coefficients of maternal psychological control and depression ( $SBS-\chi^2\text{diff}[2] = 3.11$ ,  $p > .05$ ), a significant difference did emerge at the level of the cross-lagged paths between both constructs ( $SBS-\chi^2\text{diff}[2] = 6.46$ ,  $p < .05$ ). Follow-up analyses showed



**Figure 3.** Structural models of the associations between perceived parental psychological control and adolescent depression (Study 2). Model 3a depicts associations between paternal psychological control and depression. Model 3b depicts associations between maternal psychological control and depression. Separate coefficients for males and females are provided for paths found to differ significantly by gender. The first coefficient is for males, the second coefficient is for females.

Note: \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ .

that this difference was uniquely due to the path from maternal psychological control T1 to adolescent depression T2, which was significant for males ( $\beta = .17, p < .01$ ) but not for females ( $\beta = -.06, p > .05$ ). In contrast, the significant path from adolescent depression at T1 to maternal psychological control at T2 was not moderated by gender. The final model for maternal psychological control is depicted in Figure 3.

### **Brief Discussion**

The pattern of findings in Study 2 was generally consistent with the findings of Study 1 in that the reciprocal effects model generally provided the best fit to the data. However, a notable difference emerged between the models for paternal and maternal parenting. Evidence was obtained for a reciprocal effects model in associations between paternal psychological control and adolescent depressive symptoms. This model was found to fit the data equally well for male and female adolescents. In contrast, the reciprocal effects model did not provide the best fit to the data for maternal ratings of psychological control. Instead, the adolescent adjustment effects model provided the best fit. A small but significant positive effect of adolescent depression on maternal psychological control was found. However, multigroup analyses indicated that the association between maternal psychological control and depressive symptoms was qualified by adolescent gender. It was found that whereas maternal psychological control predicted increases in boys' depressive symptoms, it did not predict increases in females' depressive symptoms. Overall, then, Study 2 evidences cross-lagged effects of perceived psychological control on future levels of adolescent depressive symptoms in all parent-child dyads except for the mother-daughter dyad. Moreover, cross-lagged effects of adolescent depression on both paternal and maternal psychological control were evident irrespective of parents' and adolescents' gender.

### **General Discussion**

The general aim of the present research was to explicitly test and compare three conceptual models on the relations between perceived parental psychological control and adolescent depressive symptoms. By simultaneously controlling for prior levels of each construct (i.e., stability effects) and for within-time associations between psychological control and depression at each consecutive wave, the present studies provide, to our knowledge, one of

the most rigorous tests of the longitudinal associations between psychologically controlling parenting and depressive symptoms to date.

With one exception in Study 2 (which will be commented upon below), this study provided convincing evidence for significant cross-lagged paths between psychological control and depression. The results suggest that perceived psychological control does not merely correlate with adolescents' depressive symptoms but instead relates to increased levels of depressive symptoms over time. Study 1 additionally demonstrated that these effects remain significant after controlling for the effects of two other fundamental parenting dimensions (i.e., responsiveness and behavioral control), a finding that replicates past demonstrations of the specialized effects of psychological control (Barber & Harmon, 2002) at the longitudinal level. Together, the findings suggest that parents who are perceived to use intrusive socialization techniques such as withdrawing love, inducing shame, and instilling guilt are likely to increase adolescents' symptoms of depression.

Future research may explore the mediating mechanisms of these longitudinal effects of psychological control on adolescent well-being. In a recent cross-sectional study, Soenens, Vansteenkiste, et al. (2005) demonstrated that the association between psychological control and depressive symptoms was accounted for by adolescents' maladaptive (but not adaptive) perfectionism. Children of psychologically controlling parents report high levels of self-criticism and negative self-evaluations that, in turn, relate to internalizing problems. Other recent cross-sectional studies (e.g., Vansteenkiste, Zhou, et al., 2005) show that psychological control relates to more controlled or pressured behavioral regulations that, in turn, relate to lower well-being and achievement. Longitudinal research is needed to establish whether psychological control predicts increases in these mediators (i.e., maladaptive perfectionism and controlled functioning) and whether changes in these mediators serve to explain the longitudinal associations between psychological control and adolescents' depressive symptoms.

Conversely, adolescent depressive symptoms were also found to predict increased levels of perceived psychological control. It should be noted, though, that we obtained somewhat weaker support for the adolescent adjustment effects than for the psychological control effects. For instance, only one of the three possible adolescent adjustment effects (i.e., from T1 to T2, from T2 to T3, and from T1 to T3) in Study 1 was significant. Study 2 did provide more consistent evidence for an effect of adolescent depression on psychological control, but these effects were relatively small. Although adolescent effects were generally somewhat less pronounced than the psychological control effects across the two studies, the adolescent effects observed suggest that adolescents with high levels of depressive symptoms

perceive their parents as becoming increasingly intrusive over time. Due to the self-reported nature of the assessment of psychological control, at least two possible interpretations of these findings can be forwarded. It may be the case that adolescents suffering from depressive symptoms merely perceive their parents as becoming more controlling. Another possibility is that adolescent depression actually fosters a change in parents' behavior such that parents act increasingly controlling toward depressive children. The latter interpretation would imply that adolescent depression represents a predictor of psychological control in addition to other predictors that have been identified in past research, such as parental personality (Soenens et al., 2006).

Future research may examine these two possible explanations in greater detail by including parent reports or observational ratings of parental control. In case adolescent depression causes an actual change in parental behavior, it will be important to tap into the underlying processes explaining this effect. Parents' affective reactions to their adolescents' emotional problems may at least partly explain the use of psychological control (Dix, 1991). Parents of a depressed adolescent may experience disappointment, frustration, worry, or even anxiety. Pressured by their own negative emotions, parents may consider the use of psychological control as the most efficient way to revitalize their children. Depressed adolescents' interpersonal behaviors may provide another explanation of the link between depressive symptoms and increased parental control. According to interpersonal theories of depression (e.g., Coyne, 1976), depressed individuals engage in a clinging and excessively reassurance-seeking interpersonal style. Parents may react to such an interpersonal style by blaming the child, inducing guilt for the child's immature and weak behavior and thus engaging in behaviors that are perceived as controlling by adolescents. As suggested by the current findings, such parenting may even further strengthen a negative vicious cycle of intrusive and controlling parenting and adolescent maladjustment.

Another important aim for future research may be to determine the relative contribution of parental characteristics such as perfectionism and separation anxiety (Soenens, Vansteenkiste et al., 2006) and child characteristics such as vulnerability to depression to the prediction of parental control. Moreover, future research may examine how child and parent characteristics interact to predict psychological control. For instance, it could be hypothesized that although perfectionist parents are generally more likely than non-perfectionist parents to use psychological control, perfectionist parents will be even more likely to engage in intrusive parenting when their child shows symptoms of depression. Perfectionist parents are known to set high

standards for their children and to engage in harsh evaluation of their children's behavior (Soenens et al., 2006). Perfectionist parents may therefore more easily consider depressive symptoms and withdrawn behavior in their child as a signal of failure and worthlessness, which may elicit even stronger attempts to force the child to live up to parental expectations. Unfortunately, given the vulnerable status of depressive children, they are likely to experience such parental interference as even more intrusive, which may in turn further exacerbate their negative emotional state. Such a model positing interactions between child and parent characteristics (in addition to unidirectional effects) is consistent with transactional theories of socialization (e.g., Magnusson, 1988) assuming that neither parents nor children are uniquely responsible for the interactional style they develop but that the combination of both children's and parents' characteristics determines their interactional style and subsequent adjustment outcomes.

The longitudinal associations between psychological control and depressive symptoms were tested and partially replicated across two different age groups. Although a direct comparison between the two samples was not possible due to design-related differences, the reciprocal effects model was generally the best-supported model in both studies. Notably, there were also a number of inconsistent findings across the two samples, including the stronger evidence for adolescent effects in Study 2 (middle adolescence) compared to Study 1 (late adolescence). The relatively consistent evidence for psychological control effects across the two studies is in line with perspectives assuming that intrusive and autonomy-inhibiting socialization poses a threat to children's optimal functioning at any given age because it frustrates a basic need for autonomy (e.g., Barber et al., 2005; Deci & Ryan, 2000; Grolnick, 2003). Given that adolescent adjustment effects were somewhat less consistently evident in Study 1 (late adolescence) compared to Study 2 (middle adolescence), future studies could add to our findings by more directly comparing the strength of longitudinal associations between psychological control and depression across age groups, which might also include early adolescence and childhood (for initial steps in this direction, see Morris et al., 2002). Such research could provide a more stringent test of the idea that psychological control "speaks quite basically to human development" (Barber et al., 2005, p. 114).

Although many studies have found evidence for gender differences in depressive symptoms during middle and late adolescence (e.g., Nolen-Hoeksema & Girgus, 1994), we did not find mean-level differences in depression scores between males and females in our samples. It should be noted that the unbalanced gender distribution in Study 1 did not allow for an adequate test of gender differences. However, the sample of Study 2 did

have a balanced gender distribution and still did not show gender differences in depressive symptoms. Surprisingly, some previous studies also failed to find gender differences in CES-D scores during adolescence (e.g., Santor, Zuroff, Ramsay, Cervantes, & Palacios, 1995). More importantly, some evidence was obtained for a moderating effect of gender on longitudinal associations between psychological control and depressive symptoms in Study 2. Specifically, it was found that psychological control prospectively predicted depression in three out of four parent-child dyads (father-son, father-daughter, and mother-son) but not in the mother-daughter dyad. The reverse effect (from depression to maternal psychological control) was not moderated by adolescent gender, however. Because the evidence for the moderating effect of gender was generally modest, the importance of the one single finding that mothers' psychological control does not predict daughters' depressive symptoms should not be overstated. Still, it is a rather intriguing finding, as it has been argued by some that it is precisely in the mother-daughter dyad that one may anticipate the strongest associations between psychological control and maladjustment. Rogers et al. (2003), for instance, argued that the mother-daughter dyad is characterized by high levels of intense emotional exchanges such that maternal psychological control might have the most pervasive effect among girls. No evidence for this hypothesis was found in their study, and the present study even suggests that psychological control is least predictive of maladjustment in the mother-daughter dyad. Future research is necessary to replicate this finding and to examine the possibility that mechanisms involving maternal psychological control operate differently for boys and girls. Future research may also more directly assess the relative contribution of maternal and paternal control in the prediction of depressive symptoms, for instance, by means of dominance analysis (Barber et al., 2005).

### *Limitations*

Some important limitations of this study need to be mentioned. First, both psychological control and depressive symptoms were assessed through adolescent self-reports, which may increase the likelihood of shared method variance. We attempted, however, to minimize the effect of shared method variance by using SEM with latent variables. Moreover, in previous work on psychological control, it has been demonstrated that using both parent and adolescent reports as indicators of the psychological control construct yields results that are highly similar to the use of adolescent self-reports only (e.g., Soenens et al., 2006). In addition, there are good theoretical reasons to focus on adolescents' own representations of their parents'

use of psychological control. Most likely, it is the degree to which adolescents subjectively experience their parents as intrusive and guilt inducing that will ultimately determine their own development. Despite these arguments, future longitudinal research might do well in using multiple informants to assess the construct of psychological control.

Second, the present study examined the longitudinal effect of psychological control on depressive symptoms only. Depression was chosen as the dependent variable in this study because it has been argued both theoretically and from the empirical literature that psychological control is linked particularly to internalizing problems such as depression (Barber & Harmon, 2002). Nevertheless, it would be worthwhile to examine longitudinal associations between psychological control and a broader range of adjustment variables because recent cross-sectional studies demonstrate that psychological control is related to adverse developmental outcomes in many areas of development, such as academic achievement (Vansteenkiste et al., 2005), externalizing problems (Conger et al., 1997), and social competence (Nelson & Crick, 2002). Such research would help to clarify whether the transactional dynamics evidenced in this article extend to adolescents' general psychosocial and behavioral functioning.

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