Physical activity and depressive symptoms

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Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):
Stavrakakis, N. (2015). *Physical activity and depressive symptoms: is a healthy body necessary for a healthy mind?*. [S.n.].

CHAPTER **II**

Bidirectional Prospective Associations between Physical Activity and Depressive Symptoms.
The TRAILS Study.

"All truly great thoughts are conceived by walking."

Friedrich Nietzsche, *Twilight of the Idols, Or, How to Philosophize with the Hammer*, 1889

"If you would get exercise, go in search of the springs of life."
Henry David Thoreau, *Walking*, 1862

ABSTRACT

Purpose: Low levels of physical activity have been shown to be associated with depression in adults. The few studies that focused on adolescents yielded mixed and inconsistent results. Efforts to examine the direction of this relationship have been inconclusive up to now. The aims of this study were therefore to investigate (1) the direction of the inverse association between physical activity and depressive symptoms over time and (2) whether these associations are specific to particular clusters of depressive symptoms in adolescents.

Methods: Depressive symptoms and physical activity were assessed in a population sample of adolescents (N=2230), who were measured at three waves between age 11 and age 16. Depressive symptoms were measured by the Affective Problems scale of the Youth Self-Report (YSR) and Child Behavior Checklist (CBCL), while physical activity was operationalized as the amount of time spent on physical exercise. Structural Equation Modeling was used to examine bidirectional effects of physical activity and depressive symptoms over time.

Results: We found significant cross-lagged paths from prior physical activity to later depression as well as from prior depression to later physical activity (beta values=-0.033 to -0.047). After subdividing depression into affective and somatic symptoms, the affective symptoms were reciprocally related to physical activity, while the paths between somatic symptoms and physical activity did not reach statistical significance.

Conclusion: An inverse bidirectional association between physical activity and general depressive symptoms was observed. This association was restricted to affective symptoms.

INTRODUCTION

Associations between physical activity (PA) and health have been well documented. Studies have shown that exercise has beneficial effects on medical conditions such as diabetes and cardiovascular diseases (Hu et al., 2004; Wessel et al., 2004), and also lowers the risk of developing a variety of cancers (Howard et al., 2008; Bardia et al., 2006). Associations of PA with mental health, however, have been harder to elucidate. There is some evidence suggesting that PA is inversely related to depression in adults, but it has been difficult to establish causality and the strength of the associations varied from study to study (Harris, Cronkite, & Moos, 2006; Cooper-Patrick, Ford, Mead, Chang, & Klag, 1997).

In observational studies, causality cannot be established, but the temporal order of changes in PA and depression may provide clues about the direction of the effects. It has been very difficult to determine temporality so far, however, due to the cross-sectional nature of most studies. Weyerer (1992) showed a cross-sectional association between low levels of PA and depression, but could not find a prospective association at 5-year follow-up. Since then, only a few studies have assessed the direction of the association between depression and PA, and those were relatively unsuccessful in giving a clear answer (Birkeland, Torsheim, & Wold, 2009; Jerstad, Boutelle, Ness, & Stice, 2010). Intervention trials have yielded ambiguous results as well. Whereas a number of studies indicated that PA reduces feelings of tension, anxiety and anger (Babyak et al., 2000), others could not find such clear effects (Salmon, 2001; Haboush, Floyd, Caron, LaSota, & Alvarez, 2006). In a recent review, Mead and colleagues (2009) showed that, when only trials with blinded outcome assessments, intention to treat and allocation concealment were included in the analysis, effect sizes were weaker and not statistically significant. In adolescents, although cross-sectional and intervention studies have shown a consistent inverse association between PA and depressive symptoms, results from prospective studies have been inconsistent and will be briefly described later.

Why are there so many mixed results regarding associations between PA and depression? Part of the discrepancies can be attributed to methodological limitations such as small sample sizes and, for prospective studies, short follow-up periods. Another reason might be that depression is a heterogeneous disorder, consisting of both affective and somatic symptoms. Instruments that are used to assess depressive symptoms vary widely with respect to the symptom profiles they represent, where some instruments do not include somatic symptoms, while other instruments contain multiple questions on somatic symptoms. It has been shown that somatic and affective symptoms are differentially associated with cardiac autonomic (de Jonge et al., 2006) and HPA axis function (Bosch et al., 2009). Since these two subgroups of symptoms show distinct patterns of association with various physiological measures, they may be differentially related to PA as well.

Despite these discrepancies, several, not necessarily mutually exclusive, hypotheses have been proposed to explain the inverse association between PA and depression:

1) Protection hypothesis (direct causal effect): PA decreases depressive symptoms through biological (elevation of endorphins, serotonin or endocannabinoids) or social (social contact and self-esteem) mechanisms (Allgower, Wardle, & Steptoe, 2001).

2) Inhibition hypothesis (reverse causality): depression negatively influences patterns of PA (Patten, Williams, Lavorato, & Eliasziw, 2009) through symptoms such as lack of energy, anhedonia, low mood and social withdrawal (Struder & Weicker, 2001).

3) Common Cause hypothesis: PA and depression share risk factors, for instance genetic or familial factors such as socioeconomic status (SES), neighborhood and parental rearing style (Stubbe, de Moor, Boomsma, & de Geus, 2007).

In the present study, associations between PA and depressive symptoms were studied in a large prospective population cohort of Dutch adolescents. Studying this association in adolescents is important for at least two reasons. First, evidence regarding the association between PA and depression is even more ambiguous in adolescents than in adults. While cross-sectional and intervention studies in adolescents have shown moderate to strong associations between PA and depression respectively (Monshouwer, ten Have, van Poppel, Kemper, & Vollebergh, 2009; Kirkcaldy, Shephard, & Siefen, 2002), longitudinal studies have yielded mixed results varying from strong effects (Patten et al., 2009; Motl, Birnbaum, Kubik, & Dishman, 2004) to weak (Jerstad et al., 2010; Sagatun, Sogaard, Bjertness, Selmer, & Heyerdahl, 2007) or even no effects at all (Birkeland et al., 2009; Ströhle et al., 2007). Second, a substantial number of adolescents evince sub-threshold depressive symptoms (Pine, Cohen, Cohen, & Brook, 1999). Since adolescent depressive symptoms are thought to predict full-blown depressive disorders later in life (Pine et al., 1999), researching depressive symptoms in adolescence may help to improve the understanding of the etiology of depressive disorders and help design effective interventions for treating affective disorders.

The first aim of this study was to investigate patterns in the association between PA and depressive symptoms, in order to determine whether changes in PA precede, follow or co-occur with changes in depressive symptoms in healthy adolescents. A longitudinal design is the only way to illuminate such patterns. The second aim was to examine the association between PA and two subgroups of depressive symptoms, that is, affective symptoms such as depressed mood and loss of interest, and somatic symptoms such as sleep disturbances and lack of energy. To the best of our knowledge, PA has not been investigated in relation to these symptom groups before.

METHODS

Design

Data for this study were collected as part of the Tracking Adolescents' Individual Lives Survey (TRAILS), a prospective cohort of Northern Dutch adolescents. The study was approved by the Dutch Central Committee on Research Involving Human Subjects. A detailed description of its objectives and main design, as well as of the sample selection procedure and non-response, can be found elsewhere (de Winter et al., 2005).

Data collection took part in three assessment waves (T1, T2, T3), which ran from March 2001 to July 2002; September 2003 to December 2004; and September 2005 to August 2007 respectively. Parental written informed consent was obtained after the procedures had been fully explained. Adolescents gave written informed assent at the second and third assessment wave.

Participants

Of 2935 adolescents initially approached, 2230 (76%; girls = 51%, mean age = 11.11, SD = 0.55) took part in the first assessment wave (T1). The response rate at the second wave (T2) was 96.4% (N = 2149; 51% girls, mean age = 13.65, SD = 0.53), while the response rate was 81.4% (N = 1816; 52.3% girls, mean age = 16.27, SD = 0.73) at the third assessment wave (T3).

Measures

Depressive Symptoms

Depressive symptoms were assessed by the Affective Problems scale of the Youth Self Report (YSR; Achenbach, 1991b), which was completed by the adolescents at school. Parents filled out the parent version of the YSR, the Child Behavior Checklist (CBCL; Achenbach, 1991a) at home. The mean scores of the YSR and CBCL scales were used in the analyses. The YSR and CBCL are composed of a list of problems that are scored on a three-point scale (0 = never or not at all true, 1 = sometimes true and 2 = very often or very true). The Affective Problems scale contains 13 items covering depressive symptoms according to the DSM-IV (Achenbach, Dumenci, & Rescorla, 2003), that is, sadness, loss of pleasure, crying, self-harm, suicidal ideation, feelings of worthlessness, guilt, loss of energy, overtiredness, eating problems and sleeping problems. Because a previous study in the same sample had shown that omission of one sleep item ('I sleep more than most kids') increased the internal consistency of the scale (Bosch et al., 2009), this item was excluded. Scores on the remaining twelve items were averaged to construct a total depressive symptoms scale with an internal consistency (Cronbach's alpha) of 0.75 at T1, of 0.84 at T2 and 0.89 at T3. In addition,

we constructed a somatic symptoms subscale and an affective symptoms subscale, as described by Bosch et al. (2009). The affective symptom subscale included loss of pleasure, crying, self-harm, suicidal ideation, feelings of worthlessness, feelings of guilt and sadness, while the somatic symptoms subscale included lack of appetite, overtiredness, reduced sleep, trouble sleeping and lack of energy. The internal consistency for the affective symptoms scale was 0.72 at T1, 0.82 at T2 and 0.85 at T3, while the consistencies of the somatic symptoms scale were 0.57 at T1, 0.69 at T2 and 0.77 at T3.

Physical Activity

At T1, PA was assessed by the question: "How often do you perform physical exercise (for example, swimming, playing football, horse-riding)?". The question could be answered on a five-point scale (0 = never, 1 = once per week, 2 = two to three times per week, 3 = four to five times per week, and 4 = five to six times per week). At T2 and T3, PA was measured by two questions assessing the time per week the individual engaged in PA during the summer (question 1) and the winter (question 2): "How many days in an average week in the summer/winter do you take part in physical activities?". These questions were rated on an eight-point scale (0 = never, to 7 = seven days per week). The questions on summer and winter were averaged and mean scores were used in the analysis. To achieve a similar metric for the PA measures at all measurement waves, the T1 data (ranging from 0-4) were recoded into an eight-point scale using the monotonic R7 transform recommended by Little (in press).

Covariates

Gender and SES were included as covariates. SES was calculated by averaging five standardized variables (professional occupation and educational attainment for both father and mother, and household income). Three SES groups were created, where the lowest 25% of scores were categorized as 'low SES', the highest 25% as 'high SES' and the remaining scores were grouped as 'intermediate SES'.

Analysis

Imputation of Missing Data

A detailed account of attrition rates within the TRAILS study can be found elsewhere (Huisman et al., 2008). We performed multiple imputations to complete the dataset (i.e., N = 2230 at all three assessment waves) using STATA version 10 (STATA corp., College station, Texas) and created five different datasets (Rubin, 1996), which were subsequently imported into Mplus 5 (Muthén & Muthén, 2007) and used in the analyses.

Cross-Lagged Path Model

Cross-sectional and prospective associations between PA and depressive symptoms were investigated by means of Structural Equation Modeling (SEM), using Mplus 5 (Muthén & Muthén, 2007). Since the depression variables (both total depressive symptoms score and scores on the two subscales) were positively skewed and slightly kurtotic, they were log transformed prior to analysis. A cross-lagged path model was used to investigate cross-sectional and prospective associations between PA and depressive symptoms. Cross-lagged panel designs take into account the time precedence and control for multivariate dependencies of the antecedent predictor variables. Therefore, besides the cross-lagged paths (interpreted as a linkage of the level of one variable at the first wave with a relative change in another variable in the subsequent assessment wave) between depression and PA and vice versa (Figure 1, path c), autoregressive paths (interpreted as relative stability over time) within depression and PA (Figure 1, path b), and cross-sectional covariances (interpreted as correlations at T1 and as correlated change at T2 and T3) between PA and depression (Figure 1, path a) were also estimated. Correlated change and cross-lagged paths reflect longitudinal relationships.

In order to test the predictive relationships between PA and depressive symptoms we developed two cross-lagged models. The first model concerned associations between PA and the total depressive symptoms score, while the second model concerned associations between

Figure 1. Model showing Cross-Sectional Paths (a), Autoregressive (b), and Cross-Lagged Longitudinal Paths (c).
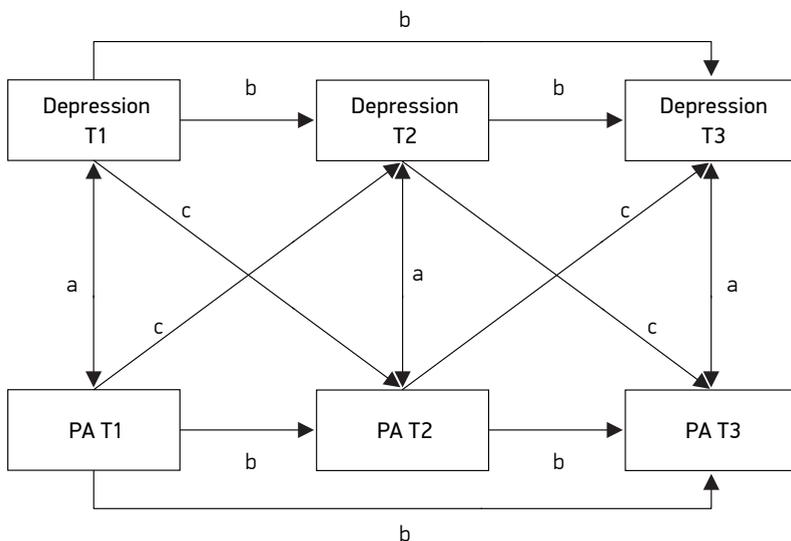
PA = physical activity, T1 = first measurement wave, T2 = second measurement wave and T3 = third measurement wave.

PA and the subscales of somatic and affective symptoms (adjusted for each other). Both models were corrected for gender and SES. As a first step, all cross-lagged and cross-sectional paths were constrained to be equal over time, and the fit indices of the constrained model were compared to those of the unconstrained model. If the fit indices were adequate and not significantly worse than those of the unconstrained model, the more parsimonious, i.e., constrained, model was used. The significance of the cross-lagged paths was established by testing if removal of the paths led to a deterioration of the model fit. To test whether the two subgroups of depressive symptoms (somatic and affective symptoms) differed with regard to their cross-sectional and cross-lagged association with PA, we compared a model where the paths between somatic symptoms and PA and affective symptoms and PA respectively, were constrained to be equal with a model where these paths were allowed to differ.

A good model fit was defined when the Comparative Fit Index (CFI) and the Tucker-Lewis Index (TLI) were greater than 0.95 while the Root Mean Square Error of Approximation (RMSEA) was lower than 0.05. Ideally, the x^2 should be non-significant (p>0.05) as well, but larger samples increase the likelihood of obtaining significant p-values (Bentler, 1990).

RESULTS

Descriptive Statistics

Descriptive statistics of the main variables used in this study are shown in Table 1. Depressive symptoms increased slightly over time, mainly due to a rise in somatic symptoms. PA remained stable over time.

Table 1. Means (Standard Deviations) of the Main Variables.

	T1 (mean age 11.1)	T2 (mean age 13.7)	T3 (mean age 16.3)
Total Depressive Symptoms	0.25 (0.20)	0.25 (0.23)	0.31 (0.29)
Affective Symptoms	0.21 (0.20)	0.19 (0.23)	0.23 (0.28)
Somatic Symptoms	0.32 (0.27)	0.33 (0.30)	0.42 (0.36)
Physical Activity	3.38 (2.09)	3.35 (1.73)	3.45 (1.88)

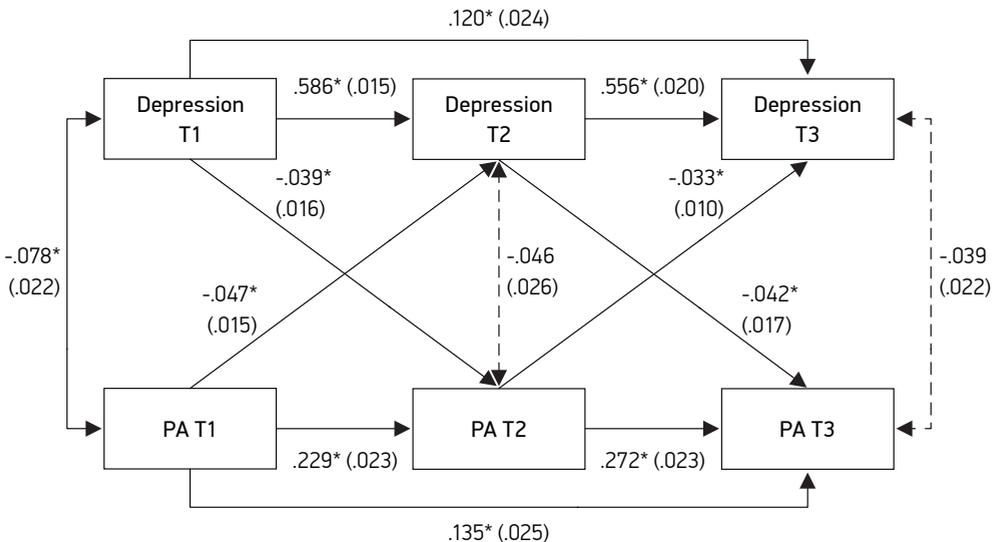N=2230 for all measurements since missing data were imputed.
T1=first measurement wave, T2=second measurement wave and T3=third measurement wave.

Relationships between Depression and PA

Preliminary analyses revealed that cross-lagged paths and correlated changes were constant over time: the model fit parameters of the models where these paths were constrained to be equal across time were adequate and the x^2-values of the constrained models were not significantly worse than the x^2-values of the unconstrained model ($x^2_{difference}$=5.555, df=3, p>0.05 for the model involving total depressive symptoms; $x^2_{difference}$=6.365, df=6, p>0.05 for the model involving depression subgroups). Because the constrained models were more parsimonious we used these models in all subsequent analyses.

Relationships between the total depressive symptoms score and PA are shown in Figure 2 (overall model fit, x^2=13.254, df=6, p<0.05). Fit indices showed an excellent fit of model (CFI=0.997, TLI=0.988, RMSEA=0.022), which was significantly better than the fit of the model without cross-lagged paths from PA to depression ($x^2_{difference}$=8.103, df=1, p<0.05) and the model without cross-lagged paths from depression to PA ($x^2_{difference}$=11.556, df=1, p<0.05). As expected from these results, the cross-lagged paths were all significant. The autoregressive estimate of depression was higher than the autoregressive estimate of PA. The cross-sectional association between PA and depression was significant at T1 but not at later assessment waves.

Figure 2. Associations between Depression (Total Depressive Symptoms Score) and PA over time.
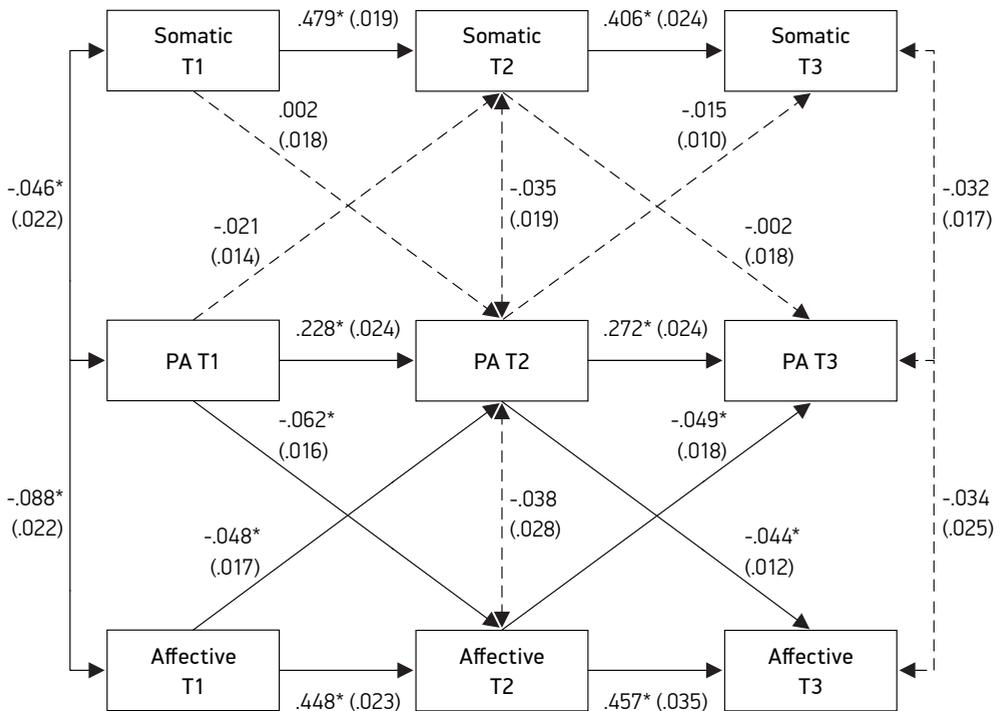

PA=physical activity, T1=first measurement wave, T2=second measurement wave and T3=third measurement wave.
The path coefficients reflect model estimated standardized beta values. Statistical insignificant paths shown are indicated by broken lines. The effects are adjusted for gender and socioeconomic status.
* p<0.05.

The second model, investigating the relationship between somatic symptoms, affective symptoms and PA over time, is shown in Figure 3. Indices of approximate fit showed an excellent fit of model variables ($x^2 = 24.170$, df = 13, p<0.05, CFI = 0.998, TLI = 0.992, RMSEA = 0.020). While cross-lagged relationships between PA and somatic symptoms did not reach statistical significance, affective symptoms both predicted and were predicted by PA. This is reflected in the tests comparing the models with and without these cross-lagged paths: whereas removal of the cross-lagged to and from somatic symptoms did not worsen the model fit significantly ($x^2_{difference} = 2.404$, df = 2, p>0.05), removal of the paths to and from affective symptoms led to a significant decrease of the model fit ($x^2_{difference} = 26.755$, df = 2, p<0.05).

Figure 3. Association between Somatic Symptoms, Affective Symptoms and PA over time.

PA = physical activity, T1 = first measurement wave, T2 = second measurement wave and T3 = third measurement wave.
The path coefficients reflect model estimated standardized beta values. Statistically insignificant paths are indicated by broken lines. The effects are adjusted for gender and socioeconomic status.
* p<0.05. Paths between somatic and affective symptoms although defined in the model (cross-sectional, autoregressive between T1 and T3, and cross-lagged) were omitted from this figure for clarity purposes.

DISCUSSION

The results of this study indicate a bidirectional cross-lagged association between depressive symptoms and PA, in which PA precedes a decrease in depressive symptoms and vice versa. Quite surprisingly, the inverse associations between PA and depressive symptoms concerned affective symptoms such as depressed mood, loss of pleasure and low self-worth in particular. Somatic symptoms such as sleep disturbances, eating problems and lack of energy were not associated with PA. Interestingly, we found that PA remained stable over time, a finding which is inconsistent with some previous studies, which reported a steady decrease of PA over time (Riddoch et al., 2004).

Limitations and Strengths of this Study

This study has a number of notable strengths. First, we used a large, population-based sample of adolescents. Studying adolescents has two main advantages: a) the probability of confounding by prior depressive episodes is low at this age, and b) the prevalence of somatic conditions that may hamper engagement in PA is still relatively low in adolescents compared to older people. Second, the longitudinal design with three measurements across 6 years made it possible to study changes in PA and depressed mood over a long period of time. Third, the model fit was excellent, modeling the relationship between PA and depressive symptoms in our data with great accuracy. A final strength is the fact that we subdivided depressive symptoms into somatic and affective symptoms, which, to the best of our knowledge has not been done before in this context. Depression is a heterogeneous disorder, with different symptoms observed in different individuals. The use of subgroups does justice to this heterogeneity and may help to understand individual differences in the association between PA and depression.

There are also limitations. First, all of the measures used were self-reports and the measurement of PA relied on a single question, unlike validated PA questionnaires (e.g., IPAQ), which include a series of detailed questions of PA involvement. Self-reports tend to be less reliable than more objective measures of PA, such as VO_2 max, heart rate variability and calculation of Metabolic Equivalent of Tasks by, for instance, accelerometers. In addition, at the first measurement wave of this study, PA was assessed slightly different than at the last two waves, in which separate questions for winter and summer, and more response categories were used. Furthermore, we did not measure the nature (aerobic-anaerobic, social-isolated etc.), intensity, or duration of PA, information which may prove useful for a better understanding of the relationship between PA and depressive symptoms. Concerning the nature of PA, for instance, aerobic exercises have been reported to have an effect on depressive symptoms while anaerobic exercises do not (Goodwin, 2003). Similarly, differences in the

intensity and the duration of the activity may be related to depressive symptoms (Galper, Trivedi, Barlow, Dunn, & Kampert, 2006). A final limitation is that the reliability of the somatic symptoms subscale was relatively low, which prevents strong associations with other variables.

The associations observed were bidirectional. The more times per week adolescents engaged in PA, the more likely they were to report a reduction in depressive symptoms. The opposite also held true: the more depressive symptoms adolescents exhibited at some point in time, the more likely they were to report a reduction in PA later on. These findings are in accordance with recent reviews in adults which showed that regular PA decreases the risk for developing depression (Teychenne, Ball, & Salmon, 2008) and that baseline depression might play an important role in the development of an inactive lifestyle or decreased level of PA (Roshanaei-Moghaddam, Katon, & Russo, 2009). In adolescents, longitudinal studies have yielded mixed results. Whereas Jerstad et al. (2010) demonstrated a bidirectional relationship between exercise and depression, Birkerland and colleagues (2009) could not. The bidirectional association between PA and depression found in our study provide support for both the Protection hypothesis and Inhibition hypothesis, which were mentioned earlier in the Introduction.

We also showed that PA is differentially associated with subgroups of depressive symptoms; the effects are stronger for affective than for somatic depressive symptoms. This lack of association between PA and somatic symptoms is unexpected. Although it seems plausible that adolescents who exercise more sleep better, eat better and have more energy, this was not supported by our data. A reason for this lack of association (between PA and somatic depressive symptoms) may be the before-mentioned low reliability of the somatic symptoms scale, which prevents strong relationships. The low reliability implies that there is heterogeneity within the somatic symptom domain. Whether specific somatic symptoms are differentially related to PA was beyond the scope of this paper and remains to be investigated in the future.

The inverse associations found between PA and depression were statistically significant but were relatively weak. This is generally in accordance with previous studies, which rarely yielded strong effects. This could be due to the measures of PA used, which usually reflect only a small portion of activities and do not take into account the nature of the activities (e.g., voluntary, social, competitive or isolated), their duration and their intensity. PA may be beneficial to mental health only after an intensity/duration threshold is reached, which could have weakened the associations. Another likely reason for the weak associations is the fact that both depressed mood and PA are affected by multiple non-shared variables and therefore cannot be expected to explain much variance in each other. Finally, it might be possible that PA and depressive symptoms are not associated in all individuals in the same way; due

to genetic or personality differences, some adolescents may benefit from PA while others do not. An elucidation of the underlying mechanisms might provide clues about which individuals may benefit most from PA.

CONCLUSION

The current prospective study investigated the inverse bidirectional associations between physical activity and depressive symptoms, in particular the two sub-clusters of depressive symptoms (somatic vs. affective), in a population cohort of Dutch adolescents. Physical activity has been shown to be related to depressive symptoms and vice versa but only in relation to affective symptoms. Further research is needed to investigate whether individual symptoms are differentially associated with physical activity, in order to improve the understanding of this complex relation.

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