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Poor peer relations in adolescence, social support in early adulthood, and depressive symptoms in later adulthood—evaluating mediation and interaction using four-way decomposition analysis

Christopher G. Bean, PhD, MPsych *, Ronnie Pingel, PhD, MSocSci, Johan Hallqvist, PhD, MD, Noora Berg, PhD, MSocSci, Anne Hammarström, PhD, MD

Department of Public Health and Caring Sciences, Uppsala University, Uppsala, Sweden

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ABSTRACT

Purpose: Supportive social relations are associated with good mental health, yet few studies have considered the prospective importance of adolescent peer relations for adult mental health and the potential mechanisms involved.
Methods: Participants ($n = 941$) were sourced from the Northern Swedish Cohort, a prospective study comprising school students aged 16 years in 1981. Integrating life course epidemiology with four-way decomposition analysis, this paper considers the controlled direct effect of poor peer relations at age 16 years on depressive symptoms at age 43 years, the pure indirect effect mediated by the availability of social support at age 30 years, and potential interactions between the exposure and the mediator.
Results: After controlling for gender, baseline depressive symptoms, and parental socioeconomic position, poor peer relations at age 16 years were associated with depressive symptoms at age 43 years, largely irrespective of social support at age 30 years. Nonetheless, poor peer relations in adolescence were associated with poorer social support at age 30 years, and mediation accounted for a modest proportion (pure indirect effect 10%) of the association between poor peer relations at age 16 years and depressive symptoms at age 43 years.
Conclusions: Policies to foster constructive peer relations for adolescents at school are encouraged; such policies may promote both the availability of social support and better mental health across the life course.

Introduction

Improving understanding of the etiological pathways associated with depression is a key concern for public mental health [1]. The biopsychosocial perspective recognizes biological [2,3], psychological [4–7], and social factors [8], associated with depression. This paper considers the prospective role of two social factors: poor peer relations in adolescence and social support in early adulthood.

Bronfenbrenner's [9] ecological theory of development has evolved over time [10], still a defining feature remains the importance of considering the interconnected influence of various environmental settings and activities wherein an individual operates [11]. Bronfenbrenner's model labels several levels or systems, which may be seen as circles of influence layered around the individual at the center. The "microsystem," comprises the milieus closest to the individual, including home life, school setting, and peer relations [9]. Interactions between settings within this level constitute the "mesosystem." Another relevant component is the "chronosystem," describing the temporal patterning of ecological events and transitions throughout life. Similarly, life course epidemiology emphasizes the importance of understanding exposures occurring throughout life, which may independently, cumulatively, and/or interactively influence health [12]. Life course epidemiology provides a basis for building and testing theoretical models that link exposures across the life course to later outcomes. The model applied in this paper is "chains of risk," whereby one exposure may increase the risk of subsequent exposures, in addition to having an independent effect on disease risk regardless of the later exposure [12].

Adolescence is a crucial period for individual development where adult patterns of mental illness may be established [13,14].

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* Corresponding author. Department of Public Health and Caring Sciences, Uppsala University, Uppsala, Sweden. Tel.: +46 18-471 6646.
E-mail address: christopher.bean@pubcare.uu.se (C.G. Bean).

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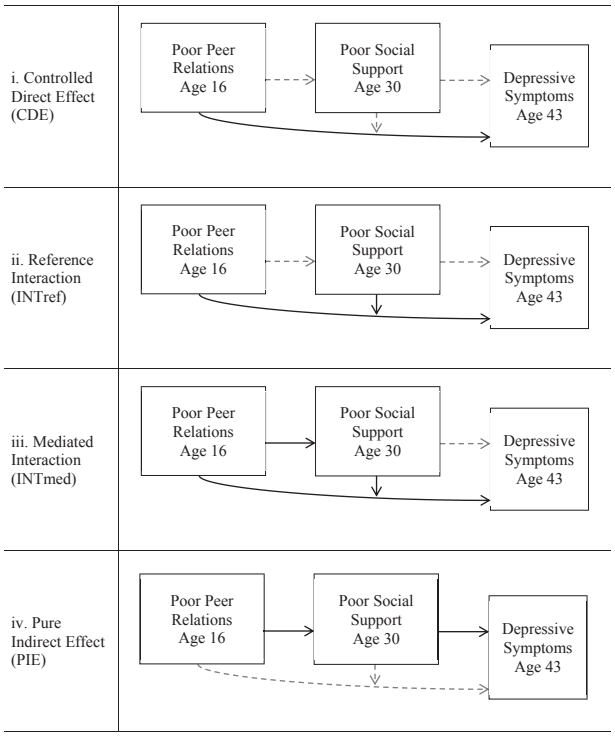
The quality of an individual's peer relations may be indicative of their popularity within a social hierarchy; the degree to which they are liked and accepted by their peers or to which they experience bullying or ostracization [15]. Previous research suggests associations between poor peer relations in adolescence and poorer socioeconomic and health outcomes in later adulthood [15–19]; however, potential intermediary mechanisms along this pathway have received less attention. Social competence–based perspectives suggest that successful relations with peers during this formative time may serve an important function in the refinement of social skills and personal competence, facilitating the ongoing cultivation of social support [20]. Such social support may in turn serve a protective function against poor health, such as depressive symptoms [21–24]. Social support is considered generally beneficial for concurrent psychological well-being [8], as well as from early to midlife adulthood [23,25]; however the potential for social support (or lack thereof) to act as an intermediary factor (i.e., a mediator) specifically between poor peer relations in adolescence and later life depressive symptoms remains unclear.

The concepts of mediation and interaction are useful for disentangling the factors linking exposures and outcomes, helping to explain respectively "how" and "for whom" a cause affects an outcome [26]. In this paper, the mediation hypothesis is that poor peer relations in adolescence may increase the risk of poor social support in early adulthood, which in turn may increase the risk of depressive symptoms in later adulthood. The interaction hypothesis is that poor peer relations may be more strongly associated with depressive symptoms in later adulthood for those who also experience poor social support in early adulthood. There may be instances where both mediation and interaction are present; in such circumstances, the validity and interpretation of traditional mediation analysis are hampered since it is not suited to considering these processes simultaneously or distinguishing the proportionalities of these effects [27]. The need to better understand epidemiologic mechanisms has spurred the development of innovative methods for mediation in recent years [27], such as the unified four-way decomposition of mediation and interaction analysis [28]. This counterfactual approach assesses mediation and interaction simultaneously, providing maximum insight into the nature of the total effect [28]. Figure 1 provides an overview of the conceptual model applied in this paper, demonstrating how the chains of risk model can be developed and integrated with the respective components of the four-way decomposition.

The aim of this paper was to evaluate the importance of poor peer relations in adolescence (exposure) and poor social support in early adulthood (mediator) as determinants of depressive symptoms in later adulthood (outcome) and their mechanistic relations. First, the total effect of the exposure on the outcome is established. Second, the nature of this total effect is decomposed. Consistent with this, the following research questions correspond with the four respective components of the four-way decomposition as depicted in Figure 1:

i. Controlled direct effect (CDE): Are poor peer relations at age 16 years associated with depressive symptoms at age 43 years, excluding the average effect of social support at age 30 years? How much of the total effect is due to just this CDE?
ii. Reference interaction (INTref): Is there a combined effect of poor peer relations at age 16 years and poor social support at age 30 years, for depressive symptoms at age 43 years? How much of the total effect is due to just this interaction?
iii. Mediated interaction (INTmed): Is there a combined effect of poor peer relations at age 16 years and poor social support at age 30 years (which was caused by poor peer relations at age 16 years), for depressive symptoms at age 43 years? How much of the total effect is due to both mediation *and* interaction between poor peer relations at age 16 years and poor social support at age 30 years?
iv. Pure indirect effect (PIE): Is poor social support at age 30 years associated with depressive symptoms at age 43 years, and if so, how much of this is due to the association between poor peer relations at age 16 years and poor social support at age 30 years? How much of the total effect is due to just this mediation?



Solid lines indicate path for the effect of interest for the respective components, dashed lines indicate effects held constant. The Controlled Direct Effect (CDE) describes the effect of the exposure that is due to neither mediation nor interaction. The Reference Interaction (INTref) describes the effect that is attributable to just interaction but not mediation. The Mediated Interaction (INTmed) describes the effect that is due to both mediation and interaction together. The Pure Indirect Effect (PIE) describes the effect that is attributable to just mediation but not interaction.

Fig. 1. Incorporation of the chains of risk model and the components of the four-way decomposition of mediation and interaction analysis.

Methods

Sample

The Northern Swedish Cohort comprises all students ($n = 1083$, 53% male) completing their final year of compulsory schooling in 1981, in Luleå, Sweden. Participants were informed of the study purpose, and participation was voluntary. Aged 16 years at baseline, participants were followed up at ages 30 and 43 years. Detailed questionnaires were completed at each stage, and a summary of data collection items and procedures has been published in the cohort profile [29]. Sample attrition is notably low; of those alive ($n = 1071$) at the latest follow-up in 2008, $n = 1010$ (94%) continued participation. Complete data (i.e., no missing items for any variables in the regression analyses) were available for 941 participants. Ethical approval was provided by the Regional Ethics Vetting Board in Umeå, Sweden.

Measures

Peer relations, age 16 years

As part of a series of questions regarding school environment, students responded to the question "how do you like your classmates?" on a scale from 1 "very good" to 5 "very bad." This question was selected as it has good face validity for the quality of peer relations in the school setting. The distribution of all response options is provided in Table 1. For the exposure of poor peer relations, responses of 1–2 were categorized as "good," while 3–5 were categorized as "poor."

Depressive symptoms, ages 16 and 43 years

A previously validated index was constructed based on six symptoms self-reported at ages 16 and 43 years [30]. The following items enquired about the previous 12 months: poor appetite, general tiredness, concentration difficulties, sleep problems, feeling down/sad, and feeling dejected about the future. Responses to these items were standardized and summed to create a scale from 0 to 2; higher scores indicate worse depressive symptoms. Further details about this index and its psychometric properties are published [30]. In order to preserve statistical power, this variable was treated as a continuous score; age 16 years controlling for baseline, and age 43 years as the study outcome.

Social support, age 30 years

A four-item Swedish version of the "Availability of Social Integration" scale, part of the "Interview Schedule for Social Interaction" (ISSI) [31], was used to quantify social support. Swedish translations of the Interview Schedule for Social Interaction have acceptable psychometric properties [32]. Participants were asked the following: "how many people do you know and have contact with who share your interests?," "how many people do you know that you meet or talk to during a week?," "how many friends do you have who can visit you in your home and feel at home?," and "how many people can you speak openly with?" For each question, response options were: 1 "none," 2 "1–2 persons," 3 "3–5 persons," 4 "6–10 persons," 5 "11–15 persons," and 6 "more than 15 persons." Summed scores were reversed so that higher scores indicate poorer social support. This variable was treated as a continuous score and used as the mediator.

Parental socioeconomic position, age 16 years

Participants reported the most recent occupation of both parents, and these were categorized as "working class" or "nonworking class" [33]. Parental socioeconomic position (SEP) was categorized on a scale from 0 (neither parent "working class") to 2 (both parents "working class") and used as a control variable.

Statistical analyses

Analyses were conducted in Stata v15 utilizing linear regression and the user-written med4way command [34] based on methods described by VanderWeele [28]. The four-way decomposition analysis provides itemized estimates for the four potential contributors as defined below:

i. CDE: The effect that poor peer relations at age 16 years would have had on depressive symptoms at age 43 years, if everyone had received the same amount (e.g., median score) of social support at age 30 years, and the effect of the mediator was excluded. This indicates the extent to which the exposure affects the outcome through pathways that do not require the mediator. This is the portion of the total effect that is due to neither mediation nor interaction.
ii. INTref: The combined effect that poor peer relations at age 16 years and poorer social support at age 30 years would have had on depressive symptoms at age 43 years (for those who have poor peer relations at age 16 years), if having poor peer relations at age 16 years is not in itself necessary for poorer social support at age 30 years. This is the interactive effect when the mediator is left to what it would be in the absence of the exposure and only operates when the mediator is present in the absence of the exposure. This represents the portion of the total effect that is due to interaction only (i.e., interaction but not mediation).
iii. INTmed: The combined effect that poor peer relations at age 16 years and poorer social support at age 30 years (which was caused by poor peer relations at age 16 years) would have had on depressive symptoms at age 43 years (for those who have poor peer relations at age 16 years), if having poor peer relations at age 16 years is in itself necessary for poorer social support at age 30 years. This effect only operates if the exposure has an effect on the mediator. This represents the portion of the total effect that is due to both mediation and interaction.
iv. PIE: The effect that poorer social support at age 30 years would have had on depressive symptoms at age 43 years (for those who have poor peer relations at age 16 years), if having poor peer relations at age 16 years is in itself necessary for poorer social support at age 30 years. This represents the portion of the total effect that is due to just mediation (but not interaction).

For estimating the CDE when using a continuous mediator, the value of the mediator must be fixed at some level. For the decomposition analysis presented here, the mediator was fixed at 13 (median), which is representative of average social support at age 30 years. The choice was made to fix the mediator at the median so that the results would be generally applicable at the population level. When the mediator is fixed at some level, the CDE accrues some of the effect of the interaction between the exposure and the mediator. As such, the specific interpretation of the INTref here is the portion of the total effect due to the interaction between the exposure and the mediator that is not mediated and also not captured by the CDE [28]. The estimation of the INTmed and PIE are not affected by the level at which the mediator is fixed.

Results

Table 1 presents descriptive statistics for the whole sample, as well as stratified by gender and exposure status. Those with poor peer relations at age 16 years reported higher depressive symptoms both at baseline and age 43 years, as well as poorer social support at age 30 years. There were no gender differences in ratings of peer relations or parental SEP at baseline; however, women reported significantly higher depressive symptoms on average compared to men at both baseline and age 43 years. At age 30 years, women reported poorer social support than men.

Table 2 presents crude and adjusted models to evaluate the effect of poor peer relations at age 16 years (i.e., the exposure) on both poor social support at age 30 years (i.e., the mediator) and depressive symptoms at age 43 years (i.e., the outcome; model series 1), the effect of the mediator on the outcome (model series 2), and the effect of both the exposure and the mediator on the outcome (model series 3). Poor peer relations at age 16 years were associated with poorer social support at age 30 years. This association persisted after controlling for gender, baseline depressive symptoms, and parental SEP. Being a woman and having lower parental SEP were each associated with poorer social support at age

Table 1
Descriptive statistics for study variables

Variable	Whole sample ($N = 941$)				Men ($n = 484$)			Women ($n = 457$)			Men−women diff.	Good peer relations ($n = 792$)			Poor peer relations ($n = 149$)			Good−poor peer relations diff.
	M (SD)	Range (actual)	*N*	%	*M* (SD)	*n*	%	*M* (SD)	*n*	%	*M* (Sig.)*	*M* (SD)	*n*	%	M (SD)	*n*	%	*M* (Sig.)*
Age 16 y																		
Peer relations	—	1–5	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Very good (1)	—	—	332	35.28	—	159	32.85	—	173	37.86	—	—	—	—	—	—	—	—
Fairly good (2)	—	—	460	48.88	—	255	52.69	—	205	44.86	—	—	—	—	—	—	—	—
Neither good/bad (3)	—	—	121	12.86	—	60	12.40	—	61	13.35	—	—	—	—	—	—	—	—
Bad (4)	—	—	19	2.02	—	7	1.45		12	2.63	—	—	—	—	—	—	—	—
Very bad (5)	—	—	9	0.96	—	3	0.62		6	1.31	(.108)	—	—	—	—	—	—	—
Dichotomized†		0–1																
Good (0)	—	—	792	84.17	—	414	85.54		378	82.71	—	—	—	—	—	—	—	—
Poor (1)	—	—	149	15.83	—	70	14.46		79	17.29	(.236)	—	—	—	—	—	—	—
Baseline depressive symptoms	0.48 (0.30)	0–2 (0–1.50)	—	—	0.41 (0.29)	—	—	0.56 (0.29)	—	—	−0.14 (<.001)	0.46 (0.29)	—	—	0.61 (0.31)	—	—	−1.57 (<.001)
Parental SEP		0–2																
No working class (0)	—	—	287	30.50	—	143	29.55		144	31.51	—	—	243	30.68	—	44	29.53	—
One working class (1)	—	—	321	34.11	—	162	33.47		159	34.79	—	—	270	34.09	—	51	34.23	—
Both working class (2)	—	—	333	35.39	—	179	36.98	—	154	33.70	(.567)	—	279	35.23	—	54	36.24	(0.955)
Age 30 y																		
Poor social support‡	12.95 (3.92)	4–24 (4–24)	—	—	12.48 (3.92)	—	—	13.46 (3.87)	—	—	−0.98 (<.001)	12.77 (3.88)	—	—	13.94 (4.01)	—	—	−1.17 (<.001)
Age 43 y																		
Depressive symptoms	0.44 (0.36)	0–2 (0–1.83)	—	—	0.38 (0.33)	—	—	0.49 (0.38)	—	—	−0.11 (<.001)	0.41 (0.35)	—	—	0.56 (0.39)	—	—	−0.15 (<.001)

* *P* values from *t* test (two-tail) for continuous and χ^2 for categorical.
† Ratings 1–2 categorized "good," ratings 3–5 categorized "poor.".
‡ AVSI reversed so that higher scores equal worse social support.

Table 2
Models for the mediator and outcome: linear regression for poor social support at age 30 years and depressive symptoms at age 43 years ($n = 941$)

	Effect of exposure on mediator and outcome			Effect of mediator on outcome			Effect of exposure and mediator on outcome					
	Crude 1	Adjusted 1		Crude 2	Adjusted 2		Crude 3		Adjusted 3		Final	
	b (95% CI)	*b* (95% CI)	β	*b* (95% CI)	*b* (95% CI)	β	*b* (95% CI)	β	*b* (95% CI)	β	*b* (95% CI)	β
Model for poor social support, age 30 y (mediator)												
Age 16 y												
Poor peer relations[†]	1.17 (0.49–1.86)**	1.09 (0.40–1.78)**	.10	—	—	—	—	—	—	—	—	—
Gender[‡]	—	0.96 (0.45–0.47)***	.12	—	—	—	—	—	—	—	—	—
Baseline depressive symptoms	—	0.15 (−0.71 to 1.02)	.01	—	—	—	—	—	—	—	—	—
Poor parental SEP[§]	—	0.49 (0.18–0.79)**	.10	—	—	—	—	—	—	—	—	—
	$R^2 = .012$	$R^2 = .037$		—	—	—	—	—	—	—	—	—
Model for depressive symptoms, age 43 y (outcome)												
Age 16, y												
Poor peer relations[†]	0.15 (0.09–0.22)***	0.11 (0.05–0.17) **	.11	—	—	—	0.14 (0.08–0.20)***	.14	0.10 (0.04–0.16)**	.10	0.04 (−0.17 to 0.26)	.05
Gender[‡]	—	0.07 (0.02–0.11) **	.10	—	0.06 (0.12–0.10)*	.08	—	—	0.06 (0.01–0.10)*	.08	0.06 (0.01–0.10)*	.08
Baseline depressive symptoms	—	0.26 (0.18–0.34)***	.22	—	0.28 (0.21–0.36)***	.24	—	—	0.26 (0.18–0.34)***	.22	0.26 (0.18–0.34)***	.22
Poor parental SEP[§]	—	0.02 (−0.01 to 0.05)	.05	—	0.14 (−0.01 to 0.04)	.03	—	—	0.01 (−0.01 to 0.04)	.03	0.01 (−0.01 to 0.04)	.03
Age 30 y												
Poor social support	—	—	—	0.01 (0.01–0.02)***	0.01 (0.01–0.02)***	.13	0.01 (0.01–0.02)***	.14	0.01 (0.00–0.02)***	.12	0.01 (0.00–0.02)**	.11
Interaction												
Poor peer relations × poor social support	—	—	—	—	—	—	—	—	—	—	0.00 (−0.01 to 0.02)	.06
	$R^2 = .025$	$R^2 = .920$	—	$R^2 = .024$	$R^2 = .950$	—	$R^2 = .043$	—	$R^2 = .104$	—	$R^2 = .105$	—

* $P < .05$; **$P < .01$; ***$P < .001$.
† Dichotomized, “poor” coded as 1, “good” coded as 0.
‡ Women coded as 1, men coded as 0.
§ Both parents “working class” coded as 2, one parent “working class” coded as 1, neither parent “working class” coded as 0.

Table 3
Four-way decomposition of effect of peer relations (age 16 years) and social support (age 30 years) for depressive symptoms at age 43 years (final model described in Table 2)

	Decomposition		
	b	95% CI	Effect proportion
Total effect	.108**	0.05–0.17	—
CDE	.094**	0.03–0.16	0.87**
INTref	−.001	−0.00 to 0.00	−0.01
INTmed	.004	−0.01 to 0.02	0.04
PIE	.011*	0.00–0.02	0.10*
N	941	—	—

Adjusted for gender, baseline depressive symptoms, parental SEP.
CDE = controlled direct effect; INTref = reference interaction; INTmed = mediated interaction; PIE = pure indirect effect.
* $P < .05$; ** $P < .01$.

30 years. Both poor peer relations at age 16 years and poor social support at age 30 years were associated with depressive symptoms at age 43 years, and this was observed in both bivariate and mutually adjusted models. These associations persisted after controlling for gender, baseline depressive symptoms, and parental SEP. Being a woman and baseline depressive symptoms were each also associated with depressive symptoms at age 43 years. The final model additionally included an interaction term for poor peer relations at age 16 years and poor social support at age 30 years. In this model, the interaction was not associated with the outcome.

Applying the four-way decomposition, Table 3 provides insight into the composition of the "total effect" of the exposure on the outcome, as identified in Table 2. The total effect indicates that those with poor adolescent peer relations score on average 0.108 points higher on the depressive symptoms scale, compared to those who did not have poor peer relations, given a similar level of baseline depressive symptoms and parental SEP. The CDE was the main contributor with a coefficient (*b*) of .094, representing approximately 87% of the total effect. There was no indication of either a reference or mediated interaction between the exposure and the mediator. A PIE through poorer social support at age 30 years was observed with a coefficient (*b*) of .011 or approximately 10% of the total effect. When the respective components are combined, mediation accounts for approximately 14% of the total effect, while the interaction effect appears negligible.

Discussion

Supporting the relevance of two components within Bronfenbrenner's microsystem (i.e., school peer relations, and social support), we found an association between poor peer relations in adolescence and depressive symptoms in later adulthood (CDE) and a PIE occurring through poorer social support in early adulthood.

There was no support for interactive effects as outlined in our second (INTref) or third (INTmed) research questions. Firstly, poor peer relations in adolescence and poorer social support in early adulthood did not work synergistically in the association with later depressive symptoms. Secondly, while poor peer relations in adolescence were associated with worse social support in early adulthood, this association was not necessary for the association between poor peer relations and later depressive symptoms. As such, the relevance of Bronfenbrenner's mesosystem (i.e., interactions between microsystem settings) was not supported statistically in our example.

With reference to our fourth research question, we found a PIE, whereby the effect of poor peer relations in adolescence on depressive symptoms in later adulthood, was partially mediated by poorer social support in early adulthood. These findings support the relevance of Bronfenbrenner's chronosystem and are consistent with the chains of risk with additive effect model [12], whereby one exposure contributes to further exposures and each adverse experience (i.e., poor peer relations at age 16 years and poorer social support at age 30 years) cumulatively raises the risk of the outcome (i.e., depressive symptoms at age 43 years). Conversely, considering the limited magnitude of the observed mediation, the results are also consistent with previous studies that indicate the association between poor peer relations in adolescence and poorer health in adulthood operates through largely unidentified pathways, seemingly irrespective of a number of putative mediators [15]. While the focus of this paper has been on social processes, another potential mechanism that may contribute to our understanding of how early adversities can predispose individuals to later mental health problems is epigenetic modification (e.g., DNA methylation) [35,36]. However, while there is some early evidence to support the idea of epigenetic modification of this nature (e.g., environmental adversity → DNA methylation → poor mental health), there is a lack of prospective studies capable of testing such mediation empirically [37]. From a psychological perspective, a further potential mechanism that could operate alongside others is the role of individual differences in the appraisal of and responses to the exposures of poor peer relations and poor social support. The cognitive activation theory of stress suggests that negative or maladaptive responses to these experiences may in turn engender a sense of hopelessness (i.e., a person believes they have control but that all outcomes will be negative) or helplessness (i.e., a person believes they have no control over the outcome), thereby contributing to poorer mental health for those who are exposed [38].

Our results provide only limited support for the mechanism suggested by social competence–based perspectives (e.g., successful relations with peers during adolescence facilitate the development of social skills for ongoing efficacious social relations, serving a protective function against poor health [20–22]). Therefore, other mechanisms are likely involved, and these should be explored in future research. Such studies may wish to consider the potential for gender differences as suggested elsewhere [15,23]. Consistent with previous research [39,40], depressive symptoms were notably higher in women compared to men in our sample, at both ages 16 and 43 years. Women in our sample also reported poorer social support compared to men at age 30 years. We controlled for gender in our main analyses since the modest sample size did not allow stratification by gender. Future research with a larger sample size should consider the potential for differential mechanistic relations for men and women.

Strengths and limitations

Strengths of this study include the longitudinal cohort with exceptionally low attrition and the innovative integration of life course epidemiology perspectives with the four-way decomposition method. Limitations include the restricted sample size and the use of self-ratings for the exposure, mediator, and outcome variables; as such, the data may be subject to common-method variance bias. Nonetheless, since these measures were collected at three separate points (i.e., ages 16, 30, and 43 years), this concern is somewhat mitigated [41]. It should be noted that the question addressed in this paper is somewhat specific as motivated by theory and earlier knowledge. The pathways linking environmental influences in adolescence to depressive symptoms in adulthood are likely complex, and the relatively large CDE suggests that poor peer relations at age 16 years are associated with depressive symptoms at age 43 years through pathways that do not require the specific mediator that we have assessed (i.e., poor social support at age

30 years). Future studies may consider other potentially relevant exposures in adolescence and other potentially relevant mediators or sets of mediators in adulthood (see the study by Bellavia and Valeri [42] for discussion on multiple mediators). Furthermore, while we controlled for a basic set of variables that we identified as potential confounders, as with other observational studies, findings should be interpreted with care because of potential unmeasured confounding, including exposure-induced mediator-outcome confounding [43].

Poor school peer relations are likely related to experiences of bullying; however, our measure did not explicitly enquire about this. Future studies may wish to consider both concepts and how they may be interrelated [44]. Furthermore, our use of a single-item question to define the exposure is a limitation since it is unidimensional by nature. On the other hand, there is considerable support for the validity of similar such single-item instruments (e.g., self-rated health status) [45]. Our measure of social support captured the availability of support in terms of the number of people an individual has access to for the provision of support; it did not capture the quality of support. A potential concern may be how our findings generalize to contemporary young people, considering the changed ecological landscape that now includes social media. These technologies may affect both peer relations and the availability of social support for better (e.g., niche interest online communities) or worse (e.g., cyberbullying) [46]. Nonetheless, while the medium may be augmented, the principles of peer relations and social support are likely still relevant.

Policy implications and conclusions

This paper has considered social determinants of health from a socioecological and longitudinal perspective through the lens of life course epidemiology. We have demonstrated that Bronfenbrenner's ecological theory of development can be applied as a contextual guide in studies that extend beyond its original focus on child development. Integrating the chains of risk with additive effect model and the four-way decomposition, we have provided a novel insight into the mechanistic nature of associations between poor peer relations in adolescence, social support in early adulthood, and depressive symptoms in later adulthood. Poor peer relations in adolescence appear to be a risk factor for both poor social support in early adulthood and depressive symptoms in later adulthood. While the association between poor peer relations at age 16 years and depressive symptoms at age 43 years occurred largely through pathways other than poor social support at age 30 years, partial mediation (PIE 10%) was observed. The findings suggest investment in the promotion of good peer relations for adolescents at school may have lasting benefits, potentially improving both the availability of social support and mental health across the life course. Stakeholders should consider the important roles that schools play in fostering social development [20,47] and the potential long-term consequences.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from all individual participants included in the study.

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