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Exploring the developmental impact of internalising problems on hyperactivity/inattention symptoms in adolescence: An analysis of two UK-based longitudinal studies

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Data availability

Data and study materials for MCS and UKHLS are available via UKDS: Homepage - UK Data Service. Code is available at: OSF | Exploring the developmental impact of internalising problems on ADHD symptoms in adolescence: An analysis of two UK-based longitudinal studies.

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The authors have no competing interests to declare

Abstract

Hyperactivity/inattention symptoms and internalising problems such as anxiety and depression commonly co-occur. Research and clinical practice are often based on the premise of hyperactivity/inattention symptoms being causally precedent in this association; however, emerging longitudinal research has suggested potential reciprocal pathways, with internalising problems also exacerbating hyperactivity/inattention symptoms. In this study we therefore used *Understanding Society* and the *Millennium Cohort Study*: two large longitudinal UK samples to explore the reciprocal developmental relations between Hyperactivity/inattention symptoms and internalising problems in adolescence. The analytic sample for *Understanding Society* included n= 13,680 participants (n= 6844 male, n=6825 female) aged 10-15, the majority whom were White (n= 9,370). The analytic sample for MCS included n=13,566 participants aged 11-17 (participants (n= 6,486 male, n=6,683 female), the majority of whom were white (n=11,241). Using a random intercept cross-lagged panel model (RI-CLPM) and RI-CLPM with reciprocal contemporaneous effects, we found evidence that internalising problems exacerbate hyperactivity/inattention symptoms (βs = .10 to .19). Evidence for a negative effect of hyperactivity/inattention symptoms on later internalising problems was inconsistent. Results point to the importance of considering pathways that lead from internalising problems to hyperactivity/inattention over adolescent development.

Keywords: hyperactivity, inattention, internalising problems, anxiety, depression, reciprocal RI-CLPM

Hyperactivity/inattention symptoms and internalising problems such as anxiety and depression are known to commonly co-occur (Choi et al., 2022). For those clinically diagnosed with attention deficit hyperactivity disorder (ADHD), of which inattention and hyperactivity (alongside impulsivity) are characteristic, rates of anxiety and depression exceed those in the general population, while at a dimensional level (reflecting that both ADHD symptoms and internalising problems are on continuua), hyperactivity/inattention symptoms and internalising problems are consistently correlated (Choi et al., 2022; Gundel et al., 2018; Krone & Newcorn, 2015; Murray et al., 2020; Roy et al., 2015). Numerous explanations have been offered for the association, though they predominantly focus mechanisms by which hyperactivity/inattention symptoms might engender the risk for later internalising problems (Antony et al., 2021; Humphreys et al., 2013; Murray et al., 2024; Powell et al., 2020; Roy et al., 2015) or on common causes such as shared genetic risk (Gustavson et al., 2021). However, emerging evidence from longitudinal studies suggest that the relations between hyperactivity/inattention symptoms and internalising problems may be reciprocal (Murray et al., 2020), highlighting the importance of considering the reverse pathway, namely the impact of internalising problems on hyperactivity/inattention symptoms.

As a neurodevelopmental condition, ADHD symptoms are typically thought to manifest earlier in development than internalising problems, at least in terms of forming a diagnosable condition (Kessler et al., 2005, 2007; Rocco et al., 2021; Solmi et al., 2022). As such, explanations for the co-occurrence between hyperactivity/inattention and internalising problems tend to afford causal precedence to the former. Hypothesised mechanisms include hyperactivity/inattention symptoms engendering risk for internalising problems through, for example, peer and academic problems, lower self-esteem, executive dysfunction, emotion dysregulation, and exposure to particular types of parenting (Murray et al., 2024). The idea that hyperactivity/inattention symptoms are causally precedent to internalising symptoms is reinforced by longitudinal evidence of prospective links between hyperactivity/inattention

symptoms and later internalising problems, after adjusting for prior internalising problems (e.g., Antony et al., 2021; So et al., 2022), although there are some examples where this has not been replicated (Baldwin & Dadds, 2008). In further support of this idea, a recent Mendelian randomisation study taking an explicitly causal approach found evidence consistent with a causal effect of ADHD on major depression (Riglin et al., 2021). This was further supported by a study which found evidence consistent with a causal effect of ADHD on major depressive disorder across three different genetically informed designs: a register-based full sibling comparison, a prospective co-twin control analysis, and a two-sample Mendelian randomisation (Garcia-Argibay et al., 2024).

The assumption of causal precedence for hyperactivity/inattention has also been reflected in some clinical guidance and practice in relation to ADHD. For example, for co-occurring ADHD and anxiety, it has been recommended that pharmacological treatments for ADHD be introduced first, introducing those for anxiety only if this fails to resolve anxiety symptoms (Posner et al., 2014). However, it is unclear the extent to which internalising problems respond to ADHD treatment. One meta-analysis reported that methylphenidate treatment was associated with a lower risk of anxiety (Pozzi et al., 2018) and another suggested that stimulant medication use for ADHD was associated with decreased depression (Zhang et al., 2025). However, others have reported no relation between ADHD medications and internalising problems (Bryant et al., 2023). In terms of non-pharmacological interventions, treatment for ADHD with cognitive behavioural therapy (CBT) has been associated with improvements in anxiety, depression and emotional symptoms (Jensen et al., 2016), with reductions in ADHD symptoms predicting improvements in anxiety and depression (Liu et al., 2023).

Despite the predominant focus on the effects of hyperactivity/inattention symptoms on internalising problems, emerging evidence suggests that internalising problems may additionally impact on later hyperactivity/inattention symptoms. Wüstner et al. (2019) found that increases in anxiety over adolescence predicted a stronger increase in ADHD

symptoms over time in the BELLA study. Further, in the same Mendelian randomisation study that found evidence consistent with a causal impact of ADHD symptoms on major depression, the authors also reported evidence consistent with a causal impact of a more broadly defined depression phenotype on ADHD (Riglin et al., 2021). These findings are bolstered by mechanistic explanations which suggest that self-regulation, attention and their development could be disrupted by anxiety or depression (Jarrett & Ollendick, 2008; Schatz & Rostain, 2006). Another recent study explicitly considered the potential reciprocal relations between a measure of hyperactivity, inattention, and impulsivity and internalising problems. Using an autoregressive latent trajectory model with structured residuals (ALT-SR), in the Zurich Project on Social Development from Childhood to Adulthood (z-proso), developmental links between 'ADHD symptoms' and anxiety were identified across age 15 to 17 (though not age 13 to 15) (Murray et al., 2020). These paths were in addition to replicating the more established links between ADHD symptoms and subsequent anxiety. These initial indications of pathways from internalising problems to ADHD symptoms motivate the need to further examine the possibility of bidirectional links between these two symptom domains.

While the co-occurrence of hyperactivity/inattention symptoms and internalising problems is important across the whole life-course, adolescence is arguably a particularly important developmental period for understanding the potential impacts of internalising problems on hyperactivity/inattention symptoms. It is a time of substantial socio-emotional development and a peak period of onset of internalising problems (Kessler et al., 2005; Rapee et al., 2019). There is some evidence that hyperactivity/inattention symptoms also escalate in adolescence for sub-groups of adolescents (Murray, Booth, et al., 2019; Murray, Ushakova, et al., 2021). This includes the emergence of some 'late onset' adolescent ADHD cases for whom symptoms – often subthreshold in childhood – become sufficiently exacerbated in adolescence to cross diagnostic thresholds (Sibley et al., 2017). However, it is unclear the extent to which increases in internalising problems play a role in escalating hyperactivity/inattention symptoms in adolescence.

To further illuminate the potential role of internalising problems in the exacerbation of hyperactivity/inattention symptoms in adolescence, we conducted a longitudinal analysis of two large UK-based datasets. We used a random intercept cross-lagged panel model (RI-CLPM) to separate within- and between-person associations (Hamaker et al., 2015). This is important because without disaggregating these effects, between-person time-stable and within-person effects are conflated and it is unclear whether increases in individuals' internalising problems would be expected to lead to increases in hyperactivity/inattention symptoms. Further, as the time-course of the interplay between hyperactivity/inattention and internalising problems is unclear we additionally fit the recently proposed 'reciprocal model' (Muthén & Asparouhov, 2024). This is recommended as a complement to the traditional RI-CLPM when the measurement lag is longer than the potential time-lag of cross-lagged effects. It includes reciprocal contemporaneous regressions to account for such effects.

Methods

Ethical approval

Ethical approval for the collection and secondary analysis of both datasets was obtained from the relevant institutional review boards and ethical approval for the secondary analysis of the data for the present study was not required. For both datasets informed consent/assent was obtained as relevant.

Participants

Data came from two datasets. First, we used data from the *Understanding Society* youth panel (University of Essex, Institute for Social and Economic Research, 2024). *Understanding Society* is a large nationally representative household panel study, initiated in 2009 and collecting data annually from the UK nations. Those aged 10-15 in participating households are invited to take part in the youth survey forming the youth panel. In the current study, we used all youth panel data available at the time of analysis, comprising waves 1,3,5,7,9,11, and 13. The sample included n= 13, 680 participants overall (n=6,844

male, n=6,825 female and n=5 where sex was inconsistent across waves). The majority of the sample reported their ethnicity as 'British/English/Scottish/Welsh/Northern Irish' (n=9,370), with n= 223 identifying as Irish, n=20 as Gypsy or Irish Traveller, n=235 as 'any other white background', n=245 as white and Black Caribbean, n=89 as white and Black African, n=195 as white Asian, n=252 as 'any other mixed background', n=536 as Indian, n=835 as Pakistani, n=499 as Bangladeshi, n= 29 as Chinese, n=155 as 'any other Asian background', n=228 as 'Caribbean', n=561 as 'African', n=56 as 'any other Black background', n=67 as 'Arab' and n=97 as 'any other ethnic group'. Data on inattention/hyperactivity and internalising problems were available for between n=3,666 (age 10) and n=3,999 (age 14). Given that the measures of hyperactivity/inattention symptoms and internalising problems are administered every two years, to facilitate analysis we divided the sample into two cohorts: the *age 10 baseline cohort* and the *age 11 baseline cohort*. The *age 10 baseline* cohort included n=7156 participants and the *age 11 baseline cohort* included n= 7071 participants.

Second, we used data from the *Millennium Cohort Study*, a large UK-based birth cohort study initiated at the beginning of the 21st century (Connelly & Platt, 2014). The first wave of data collection took place in 2000-2002 when participants were aged 9 months old. Sampling was via a stratified random sampling procedure in which participants were clustered geographically and disproportionately sampled from the three smaller UK nations i.e., Scotland, Wales, and Northern Ireland, disadvantaged areas and ethnic minorities. The present study uses data from the adolescence waves of MCS, namely, when participants were aged 11, 14, and 17. This sample includes n=13,566 participants (n= 6,486 male, n=6,683 female, the remaining missing sex information). A majority of participants had a reported ethnicity of white (n=11,241), with n= 332 Mixed, n=341 Indian, n=526 Pakistani, n=230 Bangladeshi, n=101 Other Asian, n=93 Black Caribbean, n=184 Black African, n=23 Other Black, n=59 Other Ethnic Group (the remaining had missing ethnicity information.

Measures

Hyperactivity/inattention symptoms were measured using the parent-reported *Strengths and Difficulties Questionnaire* (SDQ; Goodman, 1997)*,* hyperactivity/inattention subscale. Parent reports were used in MCS because of their availability across ages 11,14 and 17 and self-reports were used in *Understanding Society*.

The subscale contains five items measuring symptoms covering inattention, hyperactivity and impulsivity: restless, overactive, cannot stay still for long'; 'constantly fidgeting or squirming'; 'easily distracted, concentration wanders'; 'thinks things out before acting'; and 'sees tasks through to the end, good attention span'. The reference frame is the previous six months and responses are on a on a 3-point Likert-type scale with options: *not true* (0), *somewhat true* (1), and *certainly true* (2). Positively worded items were reverse-coded, and item responses were then summed to provide an overall *hyperactivity/inattention* score with a possible range from 0 (lowest symptoms) to 10 (highest symptoms). For reference, previous research has suggested that a score of 6 represents a clinically significant score and 7 represents a borderline score, when validated against DSM-IV diagnostic interview (Riglin et al., 2016). The subscale scores have shown a good correlation with ADHD diagnosis (Algorta et al., 2016; Riglin et al., 2016) and other favourable psychometric including internal consistency reliability and gender, informant and longitudinal (developmental invariance) (Murray, Speyer, et al., 2021a, 2021b).

Internalising symptoms

Internalising symptoms were measured with the emotional problems subscale of the SDQ (described above), again by parent report in MCS and self-report in *Understanding Society*. The emotional problems items refer to: *often complaining of headaches, stomach-aches or sickness; having many worries; being often unhappy, down-hearted, or tearful; being nervous or clingy in new situations; and having many fears, being easily scared*. Items use the same reference frame and response options as the hyperactivity/inattention symptoms subscales. Composite overall *internalising symptoms* scores were created by summation, with a possible range of 0-10. Like the ADHD scores, these scores have

generally shown good psychometric properties in previous research (Kersten et al., 2016; Murray, Speyer, et al., 2021a, 2021b).

Statistical procedure

In line with recommendations for modelling cross-lagged effects we fit several model variants of the random intercept cross-lagged panel model, RI-CLPM (Muthén & Asparouhov, 2024). This is recommended based on the statistical indistinguishability of the models despite having different substantive interpretations and implications.

First, we fit the standard RI-CLPM in which within- and between-person effects can be disaggregated. Random intercept factors were specified for hyperactivity/inattention and internalising symptoms and these were allowed to covary. These capture time-stable individual differences in the overall levels of these two variables and their (between-person) associations. A lag-1 autoregressive and cross-lagged structure was specified between the time-specific residuals after including these random intercept factors. These residuals capture individuals' deviations from their baseline levels of hyperactivity/inattention symptoms and internalising problems at each time point. The autoregressive and cross-lagged effects capture whether increases (or decreases) relative to an individual's baseline are followed by subsequent relative increases (or decreases) relative to their baseline in the same variable or the other variable over time. In other words, they capture within-person autoregressive and cross-lagged effects. Concurrent (residual) covariances between hyperactivity/inattention symptom and internalising problem residuals were also modelled, reflecting the extent to which relative increases (or decreases) in one variable were associated with corresponding increases (or decreases) in the other at the same time. These covariances thus reflect within-person concurrent covariances. The autoregressive and cross-lagged effects over different lags (e.g., 11 to 14 vs 14 to 17) were allowed to differ (i.e., they were not constrained to equality), consistent with the idea that hyperactivity/inattention symptoms and internalising symptoms could differentially impact one another at different stages of adolescence. An example RI-CLPM is shown in Figure 1.

Second, we fit the reciprocal effects cross-lagged panel model in which the within-time residual covariances of the RI-CLPM are replaced with lag0 regressions. An example RI-CLPM with reciprocal contemporaneous effects is shown in Figure 2. Corresponding lag0 regressions were fixed to equality over time for identification purposes i.e., they are assumed to be time-invariant. Further to avoid the dual solution issue highlighted by (Muthén & Asparouhov, 2024) we imposed the constraint that the product of the reciprocal regressions had to be between 0 and 1 and used multiple random starts in the estimation. As recommended by these authors we first fit a model with this constraint then inspected the resulting solution to see if both reciprocal regressions were significant. Under these circumstances the model can be considered supported and interpretable. However, if one or both of the reciprocal regressions is non-significant it (they) can be eliminated from the model. Further, if one of the reciprocal regressions is estimated as 0, this can be taken as evidence that both reciprocal regressions are not supported. If this happens, the parameter can be fixed at 0 (its boundary value) to facilitate correct standard errors for the remaining model parameters.

The RI-CLPM allows for unmeasured time-varying influences on internalising problems and hyperactivity/inattention symptoms via the residual covariances. However, it assumes that the residuals are uncorrelated with the predictor, which will not hold if there are reciprocal effects. That is, if the true model is a reciprocal effects model, the cross-lagged effects in the RI-CLPM will be biased. As such it is valuable to fit both and compare the conclusions drawn across the different approaches.

Models were estimated in Mplus version 8 using pseudo maximum likelihood estimation (PML) when incorporating complex survey design variables (clustering, weights and stratification variables) and robust maximum likelihood estimation (MLR) when not. Specifically, design (attrition, clustering and stratification) adjustment was used in MCS; however, it was not in UKHLS. This was to maximise sample size in the absence of weights for the accelerated cohort that was constructed (and because the construction of the

accelerated cohort means the sample does not align to a single cross-sectional or longitudinal sampling of the population for which weights can be straightforwardly derived).

Results

Full model results are provided at: OSF | Exploring the developmental impact of internalising problems on ADHD symptoms in adolescence: An analysis of two UK-based longitudinal studies. Key results pertaining to the developmental relations between hyperactivity/inattention and internalising problems are provided below.

Understanding Society datasets

Results from the RI-CLPMs models fit to the age 10 and age 11 baseline datasets in Understanding Society datasets are provided in Table 1. The within-person cross-lagged effects RI-CLPMs suggested a detrimental effect of internalising problems on later hyperactivity/inattention symptoms across the age 12 to 14 lag ($\beta = 0.10$, p=.013) 11 to 13 ($\beta = .17$, $p < .001$) and 13 to 15 ($\beta = .11$, p=.008) lags. All within-time (residual) correlations between ADHD and internalising problems were also positive and statistically significant at p<.001 (rs = .28 - .38).

Based on the RI-CLPM model with added contemporaneous time-invariant reciprocal effects (Table 2) fit to the age 10 baseline dataset there were no significant cross-lagged effects. That is, the effect of age 12 internalising problems to age 14 hyperactivity/inattention symptoms from the RI-CLPM was no longer significant. However, neither of the contemporaneous regressions was statistically significant, suggesting that they could be trimmed from the model, leaving the standard RI-CLPM described above and presented in Table 3.

For the age 11 baseline dataset, in the initial estimation the contemporaneous effect of internalising problems on hyperactivity/inattention symptoms was estimated at 0. The model was, therefore, re-estimated with this parameter constrained to 0. Within this model (Table 2), consistent with the RI-CLPM there were significant cross-lagged effects of

internalising problems on hyperactivity/inattention symptoms across the age 11 to 13 (β = .19, *p* <.001) and age 13 to 15 (β = .14, *p*=.001) lags. However, a new effect not seen in the RI-CLPM also emerged in this model: hyperactivity/inattention symptoms had a significant cross-lagged (protective) effect on internalising problems across the age 13 to 15 lag (β = -.09, *p*=.001).

Millennium Cohort Study dataset

Parameters for the RI-CLPM fit to the MCS dataset are provided in Table 3. These suggested that there was a significant cross-lagged effect of internalising problems on ADHD symptoms across the age 14 to 17 lag (β = .100**,** *p*=.005) and of hyperactivity/inattention symptoms on internalising problems across the age 11 to 14 lag (β = .087, *p*=.014), indicative of detrimental effects but no other significant cross-lagged effects. There were, however, significant positive within-time (residual) correlations between hyperactivity/inattention symptoms and internalising problems at all time points.

In the initial estimation run of the RI-CLPM with reciprocal contemporaneous effects, the regression for the contemporaneous effect of hyperactivity/inattention symptoms on internalising problems was not significant and was therefore trimmed from the model. The results from this model are provided in Table 4. The only significant cross-lagged effect in this model was of age 11 hyperactivity/inattention symptoms on age 14 internalising problems (β =.098, *p*=.002). In addition, there was a significant contemporaneous effect of internalising problems on ADHD (β = .28, *p*<.001 at age 17 and β = .22, *p*<.001 at age 14).

Discussion

The purpose of the current study was to examine the developmental associations between hyperactivity/inattention symptoms and internalising problems in adolescence to provide insights into the extent to which their relations over time are bidirectional. We employed variants of RI-CLPM models to disaggregate within- and between-person relations between these symptom domains, including model variants with and without

contemporaneous reciprocal effects to examine the potential implications of short-term interplay between hyperactivity/inattention symptoms and internalising problems. We found evidence for small but significant effects of internalising problems on hyperactivity/inattention symptoms, though these effects were not observed across all lags and nor did they all remain significant in models which included contemporaneous effects. The evidence for an effect of hyperactivity/inattention symptoms on internalising problems was less consistent. There were only two significant cross-lagged effects from hyperactivity/inattention symptoms to internalising symptoms: one suggested a detrimental effect; however, contrary to expectation the other suggested a protective effect.

A key finding was evidence suggestive that internalising problems could worsen hyperactivity/inattention symptoms over development. Specifically, there were significant effects of age 11, 12, and 13 internalising problems on hyperactivity/inattention symptoms at age 13, 14 and 15 respectively in the *Understanding Society* data. Further, there were significant effects of age 14 internalising symptoms on age 17 hyperactivity/inattention symptoms in the *Millennium Cohort Study* dataset, though when adding contemporaneous effects this effect was attenuated to non-significance (while the added contemporaneous effect was significant). These findings are consistent with a previous study that found significant within-person cross-lagged effects of anxiety on ADHD symptoms when using a model similar to the RI-CLPM, namely the ALT-SR (Murray et al., 2020). However, this study extends those findings to two large UK longitudinal studies and additionally explores the role of contemporaneous effects, which recent studies have highlighted as important to examine alongside cross-lagged effects (Muthén & Asparouhov, 2024; Speyer et al., 2025; Yang et al., 2025). Findings suggest that the evidence for an effect of internalising problems on hyperactivity/inattention symptoms is replicable across different datasets and model specifications; however, the time-course of these effects merits further study. It may be that the longer time lags in the MCS data (3 years compared to 2 years in UKHLS) explain why the cross-lagged effect was lost in favour of a contemporaneous effect when this was added

to the model, while the cross-lagged effects in *Understanding Society* remained supported after exploring alternative model specifications. Future multi-timeframe studies and continuous time modelling may be helpful to clarify the time-course of hyperactivity/inattention and internalising symptom interplay (e.g., Dormann & Griffin, 2015; Estrada & Ferrer, 2019). Another contributing factor could be the use of parent/caregiver versus self-reports. Young people may be more aware of and qualified to report on internalising problems (which often relate to internal states) and parents may only become aware of issues once they manifest as more externalised behaviourally-defined hyperactivity/inattention symptoms.

The mechanisms underpinning the effect of internalising problems on hyperactivity/inattention symptoms also merits further study. Several have been proposed or are conceivable, operating over a range of timescales. For example, worry, intrusive thoughts, and attentional biases towards negative stimuli and negative affect could undermine cognitive performance, for example, through consuming attentional resources over short timescales (Eysenck et al., 2007; Jarrett & Ollendick, 2008; Michelini et al., 2015). Developmentally, over longer timescales, it has been proposed that negative emotionality could disrupt the normative acquisition of cognitive control and, in turn, self-regulation (e.g., Nigg et al., 2004, 2020). While this model was originally developed to apply in early development (infancy into childhood), adolescence represents a second critical period of development for self-regulation, characterised for some by a 'developmental imbalance' between cognitive control and reward processing, leading to an escalation in impulsivity (Murray, Mirman, et al., 2021; Shulman et al., 2016; Steinberg et al., 2018). The presence of this second critical period of development in adolescence raises the question of whether the heightened emotionality characteristic of internalising problems could similarly disrupt self-regulation development during this period. Future longitudinal studies including candidate mediators such as measures of cognitive control and reward processing would be valuable to examine this hypothesis further. Finally, the effects may reflect the progression of

internalising symptoms in this developmental period, i.e., the associations may represent a form of heterotypic continuity in which pre-existing internalising problems interact with adolescent development to result in increasingly hyperactivity and inattentive manifestations of distress. This explanation conceptualises inattention/hyperactivity and internalising problems less as distinct domains and more as overlapping manifestations of underlying distress, with particular presentations dominating to different extents at different points in development.

The evidence for an effect of hyperactivity/inattention symptoms on internalising problems was less consistent, conflicting with the idea that hyperactivity/inattention symptoms can lead to an increased risk of anxiety and depression via mechanisms such as peer and academic problems, emotion dysregulation and parenting (Murray et al., 2024; Roy et al., 2015). There are several possible explanations for this divergence. First, it may be that there is genuinely limited impact of hyperactivity/inattention symptoms on internalising problems at the within-person level; however, between-person time-stable effects may confound this cross-lagged effect making it appear that hyperactivity/inattention symptoms influence internalising. In contrast to cross-sectional and cross-lagged analyses with no random intercept factors, the RI-CLPM can separate out these influences and isolate the within-person effects (Hamaker et al., 2015) and there is precedent for the idea that effects identified in traditional CLPMs can disappear or reverse when implementing this disaggregation (Murray, Eisner, et al., 2019). Indeed, there were substantive positive between-person associations between hyperactivity/inattention symptoms and internalising problems in the current study, which would be conflated with any ‘protective’ or null within-person effects when not disaggregated.

A second possibility is that hyperactivity/inattention symptoms offer a mix of protective and risk factors. For example, as well as evidence suggesting that hyperactivity/inattention symptoms can lead to stressful life experiences and ‘failures’ that may impact self-esteem; there is also evidence that they are associated with ‘positive illusory

biases' (e.g., Volz-Sidiropoulou et al., 2016). These may serve to preserve a positive view of oneself and thus protect against anxiety and depression. A third possibility may be that the dynamics of hyperactivity/inattention symptoms and internalising problems vary over development and the effect of internalising problems on hyperactivity/inattention symptoms predominates in adolescence due to the marked emotional development that takes place in this period. All of these speculative explanations will require further investigation in future research.

The fact that internalising problems were associated with subsequent hyperactivity/inattention symptoms, suggest that treating hyperactivity/inattention and internalising problems sequentially and focusing on the former first is not likely to be optimal in adolescence. Rather, results point to the possibility that simultaneous intervention for internalising problems (e.g., using cognitive behaviour therapy (Eddy et al., 2022; Fordham et al., 2021)) could have a double benefit of treating anxiety and depression and potentially also reducing or preventing escalations in hyperactivity/inattention symptoms. These findings also suggest potential value in monitoring for escalations in hyperactivity/inattention symptoms following the onset or increases in anxiety or depression symptoms to facilitate early detection of hyperactivity/inattention symptom changes. To further explore these possibilities it will be valuable to replicate the study in a sample of adolescents with ADHD as the measures of hyperactivity/inattention used in the current study are correlated with but may not be unique to ADHD, and the sample was not clinically diagnosed with ADHD.

Limitations

It is important to acknowledge the limitations of the current study. First, the measures of hyperactivity/inattention symptoms and internalising problems were well-validated but brief owing to the fact that data came from large-scale multi-topic studies. Second, the time lags between waves (2-3 years) will inevitably be too long to miss shorter term hyperactivity/inattention and internalising problems dynamics. Third, while MCS analyses were design-adjusted to allow inference to the general population, UKHLS was used to form

two accelerated cohorts which were not design-adjusted, limiting the generalisability of findings to the underlying UK population.

Future directions

There are several future directions that would be valuable to build on the current finding. First, exploring the relations between hyperactivity/inattention symptoms and internalising problems over multiple timeframes, from day-to-day interplay up to longer-term developmental changes would be valuable. Second, continuing to examine their relations in the transition to adulthood would be important to understand the longer-term implications of their interplay in adolescence and whether similar relations continue into later stages of development. Third, future studies could distinguish specific symptom domains as inattention, hyperactivity and impulsivity could display different dynamics in relation to anxiety and depression. For example, previous research has more consistently supported a relation between inattention and anxiety than hyperactivity and anxiety (Michelini et al., 2015). Fourth, designs calibrated for causal inference could be deployed to examine whether the within-person effects identified are likely to reflect causal effects. While RI-CLPM-type designs can adjust for time-stable between-person confounders they are not built on an explicitly causal framework and typically do not involve identifying, measuring and adjusting for all relevant time-varying confounders (Murray et al., 2025).

Conclusion

Internalising problems may lead to within-person increases in hyperactivity/inattention symptoms in adolescence. This suggests a need for research to consider pathways that link internalising problems to hyperactivity/inattention symptom increases over development, rather than focusing predominantly on pathways from hyperactivity/inattention symptoms to internalising problems.

References

Algorta, G. P., Dodd, A. L., Stringaris, A., & Youngstrom, E. A. (2016). Diagnostic efficiency of the SDQ for parents to identify ADHD in the UK: A ROC analysis. *European Child & Adolescent Psychiatry*, *25*(9), 949–957.

Antony, E. M.-A., Pihlajamäki, M. R., Speyer, L. G., & Murray, A. L. (2021). *Does emotion dysregulation mediate the association between ADHD symptoms and internalizing problems? A longitudinal within-person analysis in a large population-representative study*.

Baldwin, J. S., & Dadds, M. R. (2008). Examining alternative explanations of the covariation of ADHD and anxiety symptoms in children: A community study. *Journal of Abnormal Child Psychology*, *36*(1), 67–79.

Bryant, A., Schlesinger, H., Sideri, A., Holmes, J., Buitelaar, J., & Meiser-Stedman, R. (2023). A meta-analytic review of the impact of ADHD medications on anxiety and depression in children and adolescents. *European Child & Adolescent Psychiatry*, *32*(10), 1885–1898. https://doi.org/10.1007/s00787-022-02004-8

Choi, W.-S., Woo, Y. S., Wang, S.-M., Lim, H. K., & Bahk, W.-M. (2022). The prevalence of psychiatric comorbidities in adult ADHD compared with non-ADHD populations: A systematic literature review. *PloS One*, *17*(11), e0277175.

Connelly, R., & Platt, L. (2014). Cohort profile: UK millennium Cohort study (MCS). *International Journal of Epidemiology*, *43*(6), 1719–1725.

Dormann, C., & Griffin, M. A. (2015). Optimal time lags in panel studies. *Psychological Methods*, *20*(4), 489.

Eddy, L. D., Knouse, L. E., & Safren, S. A. (2022). *26 Adapting CBT for Treating Anxiety Disorders and Depression in Adults with ADHD*.

Estrada, E., & Ferrer, E. (2019). Studying developmental processes in accelerated cohort-sequential designs with discrete-and continuous-time latent change score models. *Psychological Methods*, *24*(6), 708.

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion*, *7*(2), 336.

Fordham, B., Sugavanam, T., Edwards, K., Stallard, P., Howard, R., Das Nair, R., Copsey, B., Lee, H., Howick, J., & Hemming, K. (2021). The evidence for cognitive behavioural therapy in any condition, population or context: A meta-review of systematic reviews and panoramic meta-analysis. *Psychological Medicine*, *51*(1), 21–29.

Garcia-Argibay, M., Brikell, I., Thapar, A., Lichtenstein, P., Lundström, S., Demontis, D., & Larsson, H. (2024). Attention-deficit/hyperactivity disorder and major depressive disorder: Evidence from multiple genetically informed designs. *Biological Psychiatry*, *95*(5), 444–452.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A research note. *Journal of Child Psychology and Psychiatry*, *38*(5), 581–586.

Gundel, L. K., Pedersen, C. B., Munk-Olsen, T., & Dalsgaard, S. (2018). Longitudinal association between mental disorders in childhood and subsequent depression–A nationwide prospective cohort study. *Journal of Affective Disorders*, *227*, 56–64.

Gustavson, K., Torvik, F. A., Eilertsen, E. M., Ask, H., McAdams, T. A., Hannigan, L. J., Reichborn-Kjennerud, T., Ystrom, E., & Gjerde, L. C. (2021). Genetic and environmental contributions to co-occurring ADHD and emotional problems in school-aged children. *Developmental Psychology*, *57*(8), 1359.

Hamaker, E. L., Kuiper, R. M., & Grasman, R. P. (2015). A critique of the cross-lagged panel model. *Psychological Methods*, *20*(1), 102.

Humphreys, K. L., Katz, S. J., Lee, S. S., Hammen, C., Brennan, P. A., & Najman, J. M. (2013). The association of ADHD and depression: Mediation by peer problems and parent–child difficulties in two complementary samples. *Journal of Abnormal Psychology*, *122*(3), 854.

Jarrett, M. A., & Ollendick, T. H. (2008). A conceptual review of the comorbidity of attention-deficit/hyperactivity disorder and anxiety: Implications for future research and practice. *Clinical Psychology Review*, *28*(7), 1266–1280.

Jensen, C. M., Amdisen, B. L., Jørgensen, K. J., & Arnfred, S. M. (2016). Cognitive behavioural therapy for ADHD in adults: Systematic review and meta-analyses. *ADHD Attention Deficit and Hyperactivity Disorders*, *8*, 3–11.

Kersten, P., Czuba, K., McPherson, K., Dudley, M., Elder, H., Tauroa, R., & Vandal, A. (2016). A systematic review of evidence for the psychometric properties of the Strengths and Difficulties Questionnaire. *International Journal of Behavioral Development*, *40*(1), 64–75.

Kessler, R. C., Amminger, G. P., Aguilar-Gaxiola, S., Alonso, J., Lee, S., & Ustun, T. B. (2007). Age of onset of mental disorders: A review of recent literature. *Current Opinion in Psychiatry*, *20*(4), 359.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602.

Krone, B., & Newcorn, J. H. (2015). Comorbidity of ADHD and anxiety disorders. *Attention-Deficit Hyperactivity Disorder in Adults and Children*, *98*.

Liu, C., Hua, M., Lu, M., & Goh, K. K. (2023). Effectiveness of cognitive behavioural-based interventions for adults with attention-deficit/hyperactivity disorder extends beyond core symptoms: A meta-analysis of randomized controlled trials. *Psychology and Psychotherapy: Theory, Research and Practice*, *96*(3), 543–559. https://doi.org/10.1111/papt.12455

Michelini, G., Eley, T. C., Gregory, A. M., & McAdams, T. A. (2015). Aetiological overlap between anxiety and attention deficit hyperactivity symptom dimensions in adolescence. *Journal of Child Psychology and Psychiatry*, *56*(4), 423–431.

Murray, A., Errington, P., Yang, Y., Wright, H., Mirman, D., Obsuth, I., Booth, T., Ribeaud, D., & Eisner, M. (2025). A counterfactual and random intercept cross-lagged panel analysis of the effects

of reading frequency on adolescent mental health in a large longitudinal study. *JCPP Advances*, e70075. https://doi.org/10.1002/jcv2.70075

Murray, A. L., Booth, T., Eisner, M., Auyeung, B., Murray, G., & Ribeaud, D. (2019). Sex differences in ADHD trajectories across childhood and adolescence. *Developmental Science*, *22*(1), e12721.

Murray, A. L., Caye, A., McKenzie, K., Auyeung, B., Murray, G., Ribeaud, D., Freeston, M., & Eisner, M. (2020). Reciprocal Developmental Relations Between ADHD and Anxiety in Adolescence: A Within-Person Longitudinal Analysis of Commonly Co-Occurring Symptoms. *Journal of Attention Disorders*.

Murray, A. L., Dryburgh, K., & Sonuga-Barke, E. (2024). *Identifying candidate mechanisms linking ADHD symptoms and internalising problems in adolescence: An exploratory mediation analysis*. https://www.researchsquare.com/article/rs-4869282/latest

Murray, A. L., Eisner, M., & Ribeaud, D. (2019). Within-person analysis of developmental cascades between externalising and internalising problems. *Journal of Child Psychology and Psychiatry*.

Murray, A. L., Mirman, J. H., Carter, L., & Eisner, M. (2021). Individual and developmental differences in delinquency: Can they be explained by adolescent risk-taking models? *Developmental Review*, (In Press).

Murray, A. L., Speyer, L. G., Hall, H. A., Valdebenito, S., & Hughes, C. (2021a). A Longitudinal and Gender Invariance Analysis of the Strengths and Difficulties Questionnaire Across Ages 3, 5, 7, 11, 14, and 17 in a Large UK-Representative Sample. *Assessment*, 10731911211009312.

Murray, A. L., Speyer, L. G., Hall, H. A., Valdebenito, S., & Hughes, C. (2021b). Teacher versus parent informant measurement invariance of the Strengths and Difficulties Questionnaire. *Journal of Pediatric Psychology*, *46*(10), 1249–1257.

Murray, A. L., Ushakova, A., Speyer, L., Brown, R., Auyeung, B., & Zhu, X. (2021). Sex/gender differences in individual and joint trajectories of common mental health symptoms in early to middle adolescence. *JCPP Advances*, e12057.

Muthén, B., & Asparouhov, T. (2024). Can cross-lagged panel modeling be relied on to establish cross-lagged effects? The case of contemporaneous and reciprocal effects. *Psychological Methods*. https://psycnet.apa.org/record/2024-87605-001

Nigg, J. T., Goldsmith, H. H., & Sachek, J. (2004). Temperament and attention deficit hyperactivity disorder: The development of a multiple pathway model. *Journal of Clinical Child and Adolescent Psychology*, *33*(1), 42–53.

Nigg, J. T., Sibley, M. H., Thapar, A., & Karalunas, S. L. (2020). Development of ADHD: Etiology, Heterogeneity, and Early Life Course. *Annual Review of Developmental Psychology*, *2*(1), 559–583. https://doi.org/10.1146/annurev-devpsych-060320-093413

Posner, J., Kass, E., & Hulvershorn, L. (2014). Using stimulants to treat ADHD-related emotional lability. *Current Psychiatry Reports*, *16*(10), 478.

Powell, V., Riglin, L., Hammerton, G., Eyre, O., Martin, J., Anney, R., Thapar, A., & Rice, F. (2020). What explains the link between childhood ADHD and adolescent depression? Investigating the role of peer relationships and academic attainment. *European Child & Adolescent Psychiatry*, 1–11.

Pozzi, M., Carnovale, C., Peeters, G. G., Gentili, M., Antoniazzi, S., Radice, S., Clementi, E., & Nobile, M. (2018). Adverse drug events related to mood and emotion in paediatric patients treated for ADHD: A meta-analysis. *Journal of Affective Disorders*, *238*, 161–178.

Rapee, R. M., Oar, E. L., Johnco, C. J., Forbes, M. K., Fardouly, J., Magson, N. R., & Richardson, C. E. (2019). Adolescent development and risk for the onset of social-emotional disorders: A review and conceptual model. *Behaviour Research and Therapy*, 103501.

Riglin, L., Collishaw, S., Thapar, A. K., Dalsgaard, S., Langley, K., Smith, G. D., Stergiakouli, E., Maughan, B., O'Donovan, M. C., & Thapar, A. (2016). Association of genetic risk variants with attention-deficit/hyperactivity disorder trajectories in the general population. *Jama Psychiatry*, *73*(12), 1285–1292.

Riglin, L., Leppert, B., Dardani, C., Thapar, A. K., Rice, F., O'Donovan, M. C., Smith, G. D., Stergiakouli, E., Tilling, K., & Thapar, A. (2021). ADHD and depression: Investigating a causal explanation. *Psychological Medicine*, *51*(11), 1890–1897.

Rocco, I., Corso, B., Bonati, M., & Minicuci, N. (2021). Time of onset and/or diagnosis of ADHD in European children: A systematic review. *BMC Psychiatry*, *21*(1), 575. https://doi.org/10.1186/s12888-021-03547-x

Roy, A., Hartman, C. A., Veenstra, R., & Oldehinkel, A. J. (2015). Peer dislike and victimisation in pathways from ADHD symptoms to depression. *European Child & Adolescent Psychiatry*, *24*(8), 887–895.

Schatz, D. B., & Rostain, A. L. (2006). ADHD with comorbid anxiety: A review of the current literature. *Journal of Attention Disorders*, *10*(2), 141–149.

Shulman, E. P., Smith, A. R., Silva, K., Icenogle, G., Duell, N., Chein, J., & Steinberg, L. (2016). The dual systems model: Review, reappraisal, and reaffirmation. *Developmental Cognitive Neuroscience*, *17*, 103–117.

Sibley, M. H., Rohde, L. A., Swanson, J. M., Hechtman, L. T., Molina, B. S., Mitchell, J. T., Arnold, L. E., Caye, A., Kennedy, T. M., & Roy, A. (2017). Late-onset ADHD reconsidered with comprehensive repeated assessments between ages 10 and 25. *American Journal of Psychiatry*, *175*(2), 140–149.

So, F. K., Chavira, D., & Lee, S. S. (2022). ADHD and ODD Dimensions: Time Varying Prediction of Internalizing Problems from Childhood to Adolescence. *Journal of Attention Disorders*, *26*(6), 932–941. https://doi.org/10.1177/10870547211050947

Solmi, M., Radua, J., Olivola, M., Croce, E., Soardo, L., Salazar de Pablo, G., Il Shin, J., Kirkbride, J. B., Jones, P., & Kim, J. H. (2022). Age at onset of mental disorders worldwide: Large-scale meta-analysis of 192 epidemiological studies. *Molecular Psychiatry*, *27*(1), 281–295.

Speyer, L. G., Zhu, X., Yang, Y., Ribeaud, D., & Eisner, M. (2025). On the Importance of Considering Concurrent Effects in Random-Intercept Cross-Lagged Panel Modelling: Example Analysis of

Bullying and Internalising Problems. *Multivariate Behavioral Research*, *60*(2), 328–344. https://doi.org/10.1080/00273171.2024.2428222

Steinberg, L., Icenogle, G., Shulman, E. P., Breiner, K., Chein, J., Bacchini, D., Chang, L., Chaudhary, N., Giunta, L. D., & Dodge, K. A. (2018). Around the world, adolescence is a time of heightened sensation seeking and immature self-regulation. *Developmental Science*, *21*(2), e12532.

University of Essex, Institute for Social and Economic Research. (2024). Understanding Society: Waves 1-14, 2009-2023 and Harmonised BHPS: Waves 1-18, 1991-2009. [data collection]. 19th Edition. *UK Data Service*, *19th Edition.* https://doi.org/DOI: http://doi.org/10.5255/UKDA-SN-6614-20

Volz-Sidiropoulou, E., Boecker, M., & Gauggel, S. (2016). The Positive Illusory Bias in Children and Adolescents With ADHD: Further Evidence. *Journal of Attention Disorders*, *20*(2), 178–186. https://doi.org/10.1177/1087054713489849

Wüstner, A., Otto, C., Schlack, R., Hölling, H., Klasen, F., & Ravens-Sieberer, U. (2019). Risk and protective factors for the development of ADHD symptoms in children and adolescents: Results of the longitudinal BELLA study. *PloS One*, *14*(3), e0214412.

Yang, Y., Obsuth, I., Zhu, X., Ribeaud, D., Eisner, M., & Murray, A. (2025). Occupational future time perspective and mental health problems across adolescence: Random-intercept cross-lagged panel analysis and alternative variations. *Journal of Adolescence*, *97*(2), 526–539. https://doi.org/10.1002/jad.12438

Zhang, Y., Liao, W., Rao, Y., Gao, W., & Yang, R. (2025). Effects of ADHD and ADHD medications on depression and anxiety in children and adolescents: A systematic review and meta-analysis. *Journal of Psychiatric Research*, *181*, 623–639.

Tables

Table 1: Standardised RI-CLPM parameters in Understanding Society datasets

Parameter	Estimate	SE	*p*		Estimate	SE	*p*
	Cohort 1 (baseline age 10)				**Cohort 2 (baseline age 11)**		
Within-person regressions							
H/I 12 → H/I 14	0.296	0.044	<.001*	**H/I 13 → H/I 15**	0.258	0.052	<.001*
Int 12 → H/I 14	0.095	0.038	.013*	**Int 13 → H/I 15**	0.114	0.043	.008*
H/I 12 → Int 14	-0.012	0.034	.729	**H/I 13 → Int 15**	-0.023	0.031	.456
Int 12 → Int 14	0.329	0.047	<.001*	**Int 13 → Int 15**	0.502	0.041	<.001*
H/I 10 → H/I 12	0.182	0.054	.001*	**H/I 11 → H/I 13**	0.207	0.053	<.001*
Int 10 → H/I 12	0.067	0.046	.152	**Int 11 → H/I 13**	0.171	0.048	<.001*
H/I 10 → Int 12	-0.005	0.046	.914	**H/I 11 → H/I 13**	-0.016	0.043	.698
Int 10 → Int 12	0.238	0.059	<.001*	**Int 11 → Int 13**	0.356	0.062	<.001*
Within-person (residual) covariances							
H/I 14 with Int 14	0.299	0.022	<.001*	**H/I 15 with Int 15**	0.336	0.024	<.001*
H/I 12 with Int 12	0.307	0.031	<.001*	**H/I 13 with Int 13**	0.279	0.031	<.001*
H/I 10 with Int 10	0.375	0.038	<.001*	**H/I 11 with Int 11**	0.37	0.044	<.001*

Between-person (random intercept) covariances							
H/I with Int	0.324	0.085	<.001	**H/I with Int**	0.325	0.111	0.004*

Note. * = significant at *p*<.05. H/I = hyperactivity/impulsivity symptoms; Int = internalising problems

Table 2: Standardised RI-CLPM with contemporaneous reciprocal effects parameters in Understanding Society datasets

Parameter	Estimate	SE	*p*	Parameter	Estimate	SE	*p*
Within-person regressions							
H/I 12 → H/I 14	0.298	0.043	<.001	**H/I 13→ H/I 15**	0.252	0.052	<.001*
Int 12 → H/I 14	0.055	0.068	0.417	**Int 13 → H/I 15**	0.137	0.04	.001*
Int 14 → H/I 14	0.12	0.143	0.399	**Int 15 → H/I 15**	-	-	-
H/I 12 → Int 14	-0.067	0.044	0.127	**H/I 13 → Int 15**	-0.091	0.027	.001*
Int 12 → Int 14	0.312	0.046	<.001	**Int 13 → Int 15**	0.466	0.039	<.001*
H/I 14 → Int 14	0.186	0.134	0.167	**H/I 15 → Int 15**	0.296	0.023	<.001*
H/I 10 → H/I 12	0.184	0.053	0.001	**H/I 11 → H/I 13**	0.201	0.054	<.001*
Int 10 → H/I 12	0.041	0.06	0.503	**Int 11 → H/I 13**	0.191	0.047	<.001*
Int 12 → H/I 12	0.11	0.13	0.398	**Int 13 → H/I 13**		-	-
H/I 10 → Int 12	-0.042	0.041	0.306	**H/I 11 → Int 13**	-0.058	0.037	.119
Int 10 → Int 12	0.224	0.058	<.001	**Int 11 → Int 13**	0.289	0.059	<.001*
H/I 12 → Int 12	0.204	0.147	0.165	**H/I 13 → Int 13**	0.309	0.025	<.001*
Within-person covariances							
H/I 10 with Int 10	0.375	0.038	<.001	**H/I 11 with Int 11**	0.389	0.042	<.001*

Between-person (random intercept) covariances							
H/I with Int	0.323	0.084	<.001	**H/I with Int**	0.267	0.115	.02*

Note. * = significant at *p*<.05. H/I = hyperactivity/impulsivity symptoms; Int = internalising problems

Table 3: RI-CLPM parameters in Millennium Cohort Study Dataset

Parameter	Estimate	SE	*p*
Within-person regressions			
H/I 14 → H/I 17	0.184	0.041	<.001
Int 14 → H/I 17	0.100	0.035	.005
H/I 14 → Int 17	0.002	0.028	.951
Int 14 → H/I 17	0.309	0.034	<.001
H/I 11 → H/I 14	0.333	0.03	<.001
Int 11 → H/I 14	0.001	0.03	.963
H/I 11 → Int 14	0.087	0.035	.014
Int 11 → H/I 14	0.200	0.041	<.001
Within-person covariances			
H/I 14 with Int 14	0.284	0.025	<.001
H/I 11 with Int 11	0.215	0.03	<.001
Between-person (random intercept) covariances			
H/I with Int	0.249	0.027	<.001

Note. * = significant at *p*<.05. H/I = hyperactivity/impulsivity symptoms; Int = internalising problems

Table 4: RI-CLPM with reciprocal contemporaneous effects parameters in Millennium Cohort Study Dataset

Parameter	Estimate	SE	*p*
Within-person regressions			
H/I 14 → H/I 17	0.184	0.041	<.001
Int 14 → H/I 17	0.022	0.029	.444
Int 17 → H/I 17	0.282	0.022	<.001
H/I 14 → Int 17	0.002	0.028	.946
Int 14 → Int 17	0.311	0.034	<.001
H/I 11 → H/I 14	0.313	0.031	<.001
Int 11 → H/I 14	-0.036	0.027	.175
Int 14 → H/I 14	0.224	0.02	<.001
H/I 11 → Int 14	0.098	0.031	.002
Int 11 → Int 14	0.201	0.042	<.001
Within-person covariances			
H/I 11 with Int 11	0.254	0.027	<.001
Between-person covariances			
H/I with Int	0.466	0.028	<.001

Note. * = significant at $p<.05$. H/I = hyperactivity/impulsivity symptoms; Int = internalising problems

Figures

Figure 1: Example RI-CLPM

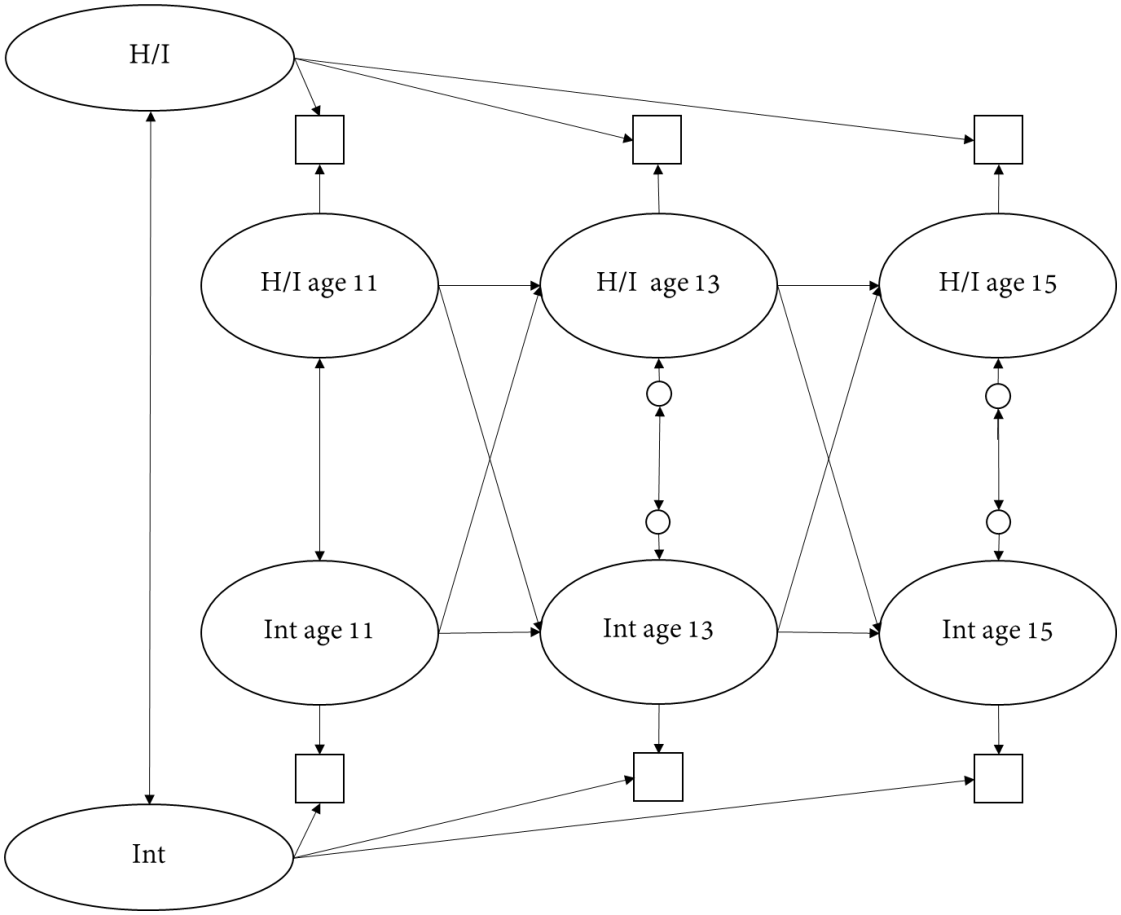
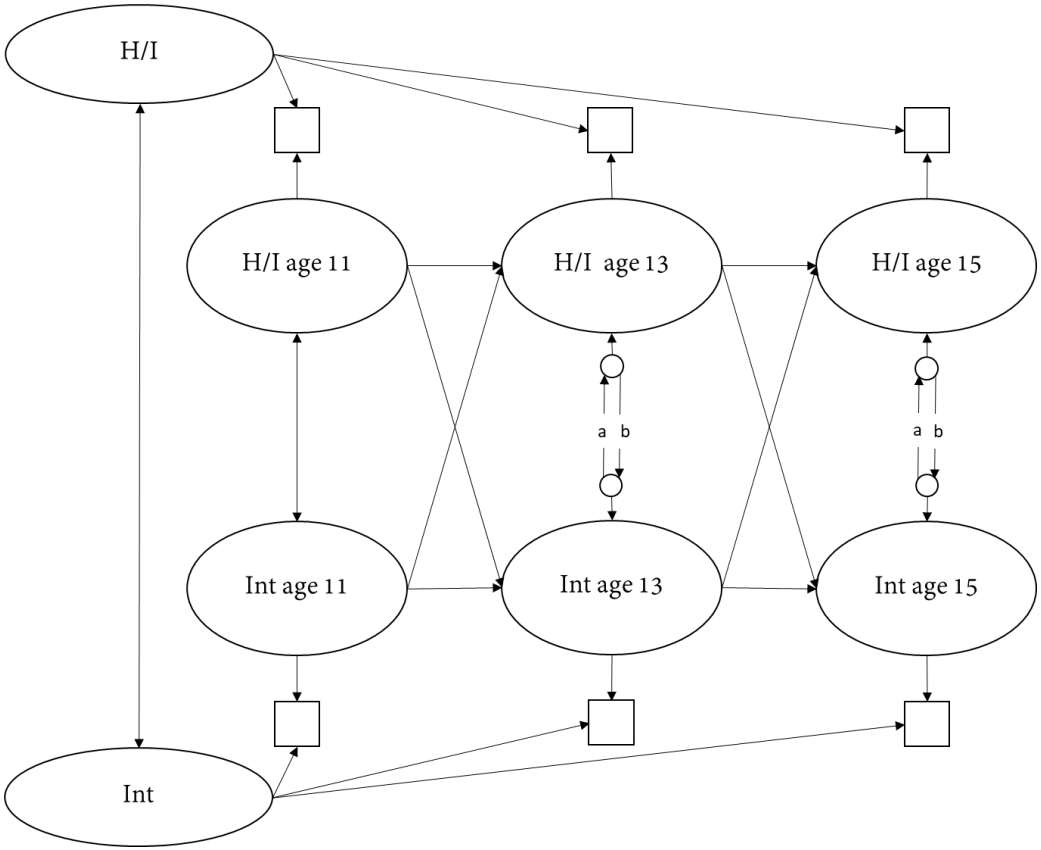
Figure 2: Example Reciprocal model (RI-CLPM with invariant reciprocal contemporaneous effects)