# The psychopathology *p* factor: Will it revolutionise the science and practice of child and adolescent psychiatry?

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Summary

The psychopathology *p* factor has emerged from a series of strong empirical studies, largely in the adult psychiatry literature. Here some of the recent findings relating to the *p* factor in children and adolescents are considered and the implications for child and adolescent psychiatry are discussed. Is it essential to covary for '*p*' when we study specific domains of psychopathology? Do neurodevelopmental conditions make up part of the psychopathology *p* factor? How do we treat the '*p* factor' in clinics? This editorial considers some of the contributions from this issue of *Journal of Child Psychology and Psychiatry* together with the wider literature that speak to these issues.

Editorial

The psychopathology *p* factor refers to a general latent dimension that is derived from a wide range of items measuring adult psychiatric symptoms. It has been proposed that this single latent factor can encapsulate individuals' proclivity to develop all forms of psychopathology included within the broad internalising, externalising and thought disorder dimensions (Caspi et al., 2014).

While some readers may not find those opening sentences a surprise, for others these statements will feel contrary to theories, clinical practices and research findings regarding symptom specificity, heterogeneity and subtype clustering within psychopathology. My guess is that there are literally thousands of research papers reporting findings that might feel in some way in conflict with the psychopathology *p* factor model.

The data supporting the psychopathology *p* factor model are strong, including from population-based cohorts and national registers. This editorial will aim to do two things. First, drawing on some of the papers in this issue, some of the newest considerations relevant to the psychopathology *p* factor model in childhood and adolescence will be discussed. Second, the relevance of the psychopathology *p* factor to child and adolescence psychiatry and thus the readers of this journal will be considered.

## The *p* factor may start early

We see in the Miller et al article of this issue that a general factor underlying psychopathology appears in their data (Miller et al., 2019). They find that factor loadings of items are more robust on what they term a general 'dysregulation profile' factor than on the Child Behavior Checklist subscale factors. This may seem to be more or less repeating the opening paragraph of this article. A key difference, however, is that the participants in Miller et al's study were 36 month old children rather than adults. As the authors mention, there have been only a small number of studies of the factor structure of the 'dysregulation profile' in young children, some of which are in specially selected samples such as clinically referred samples. As such, Miller et al's study together with other recent work in this area e.g.

(McElroy, Belsky, Carragher, Fearon, & Patalay, 2018) is important in showing that even before children have begun formal schooling or had a huge amount of life experience, seemingly specific domains of psychopathology (in Miller et al's case, internalising, externalising and attention problems) fall into a single general factor. Furthermore, Caspi et al (2014) (Caspi et al., 2014) reported that their adult *p* factor derived from the prospectively-assessed Dunedin Multidisciplinary Health and Development Study was negatively correlated with a general measure of brain integrity at age 3 years. As such, for this journal, we have no excuse to dismiss the psychopathology *p* factor model as something specific to research on adults. It's relevant to children, and therefore this journal's readers, too.

**It's not yet clear how neurodevelopmental conditions are involved**

Something to note about the Miller et al study is that the sample was enriched with children who were younger siblings of children with autism. And the eagle-eyed reader – especially those with an interest in neurodevelopment - will have noticed that the dimensions listed in the opening paragraph do not obviously include autism (or a relevant umbrella term such as neurodevelopment). Autism spectrum conditions are not mental illnesses and in diagnostic manuals they are separated from psychiatric disorders. Interestingly, in this issue of the journal we see that Miller et al did not find that genetic liability for autism was a predictor of the dysregulation profile general factor in 36 month olds.

What do we know about autism and the psychopathology *p* factor model from other samples? The Strengths and Difficulties Questionnaire (SDQ) total scale is a commonly used general psychopathology measure (see e.g. (Rimvall et al., 2019)). While it includes a subscale of peer problems, which is one aspect of social difficulties, the SDQ total does not include autistic traits per se. As such, when the *p* factor or general psychopathology is assessed with this measure in children, which is quite often the case, autism or autistic traits are not explicitly included. Some of the most prominent studies on the *p* factor have not included autism thus far, most likely because of their focus on adult psychiatry e.g. (Caspi et al., 2014; Lahey, Krueger, Rathouz, Waldman, & Zald, 2017; Pettersson et al., 2018). It is an interesting and arguably still somewhat open question: how is autism connected to the psychopathology *p* factor model?

Some traditional views and characteristics of autism do not sit naturally with the *p* factor model. The uni-dimensional *p* factor model is associated with a model of psychopathology in which symptoms wax and wane, and individuals cycle through different psychiatric diagnoses over time (because they have a general vulnerability to psychopathology rather than any specific disorder). Most data and current perspectives on autism view it as a condition that does not go away with development (although there are exceptions), does not vary particularly in terms of age of onset, and does not morph into other disorders (though co-occurrence with other psychopathology is high). It is viewed as a form of neurodevelopment rather than a 'pathology'. We also see autism in genetic syndromes, which again are viewed as different to general psychopathology for reasons such as their distinct etiology, their permanence across the lifespan and their profile of physical and cognitive characteristics. Of course traditional views sometimes need to be overturned to enable progress. A discussion of how autism is linked to the *p* factor may help refine thinking about the borders and focus of the *p* factor at different stages of development. The Miller et al study helps to progress our thinking on this front.

**The *p* factor as an essential covariate in studies of specific psychopathology**

Also in this issue, Rimvall et al explore novel questions about how positive psychotic experiences such as hallucinations and delusions are associated with health anxiety and functional somatic symptoms (Rimvall et al., 2019). In their cohort of 11-12 year olds they find significant cross-sectional associations. It is interesting to consider how early traits linked to ruminating about bodily sensations might play a part in the early stage development of symptoms such as hallucinations that are seen later in psychotic disorders. Methodologically, one of the strengths of the study was that the authors checked that the association between psychotic experiences and health anxiety and functional somatic symptoms held after controlling for general psychopathology (here measured using the Strengths and Difficulties Questionnaire). This is a strength because it offers confidence that the associations are specific to psychotic experiences over and above general psychopathology.

We see the utility of the *p* factor for understanding specific aspects of psychopathology demonstrated elsewhere in the issue. Manfro et al contribute to a fascinating (and growing) literature on youth-onset ADHD, that is, the observation that a subtype of ADHD starts in adolescence (Manfro et al., 2019). By exploring general psychopathology scores in childhood and adolescence, Manfro et al's study of trajectories of ADHD in the Brazilian high risk cohort reveals that individuals with youth-onset ADHD are already presenting in childhood with high *p* factor scores and more symptoms from other domains of psychopathology. As such, the authors hypothesise that late-onset ADHD is a combination of susceptibility to general psychopathology coupled with a transition from one domain of psychopathology in childhood to another (ADHD) later on. This finding would not have been possible without taking 'p' into account, and will have almost certainly enriched our understanding of adolescent-onset ADHD.

Have we reached a point where controlling for general psychopathology and or the *p* factor is an essential covariate in studies of *specific* psychopathology? For a long time it has been fairly standard to control for general constructs such as IQ and socioeconomic status in research. Careful consideration, in any particular study, is essential when considering alternative models (Markon, 2019). Certainly the two examples from this issue discussed here demonstrate the capacity for involving the *p* factor or general psychopathology in research on specific psychopathology in childhood in order to strengthen confidence in findings and to develop new hypotheses.

**It's full steam ahead for genetic research on the *p* factor**

In behaviour genetics, structural equation models have been used for decades to explore the structure of psychopathology and to test models of co-occurrence between disorders or their related traits (Lahey et al., 2017). A general genetic factor that influences eight major psychiatric disorders using full and half sibling data from Swedish national registers has been reported (Pettersson et al., 2018). Complementary to these findings from family data, similar conclusions were reached with three other methods that employed measured genotypes (Selzam, Coleman, Caspi, Moffitt, & Plomin, 2018). We see the *p* factor being covaried for in genetic studies on specific psychopathology too. Brikell and colleagues tested the degree to which the ADHD genomewide polygenic score predicts hyperactivity/impulsivity symptoms over and above a general genetic liability towards broad childhood psychopathology (Brikell et al., 2018). Genetic research can speak to some of the mechanisms underlying the *p* factor.

Co-occurrence of symptoms does not necessarily reveal the underlying causal pathway between symptoms. Symptoms can co-occur because of correlated causal influences or because one symptom itself causes another (though these are not mutually exclusive options). In this issue we see how network analysis can inform such issues. Bartels et al employ, amongst other things, a Bayesian approach to model directed acyclic graphs of PTSD symptoms in children and adolescents (Bartels et al., 2019). They demonstrate which symptoms of PTSD are the key drivers of other symptoms within PTSD and thus are able to advise, based on their data, which symptoms would be the optimal targets for treatment.

**Psychopathology may be general, but treatments can still be specific**

Instead of treating specific symptoms, or disorders, should we treat *p*? Causation does not denote treatment, but does factor structure denote treatment? One might imagine a future scenario where patients circulate around a wide range of clinicians trained in specific areas who can together support a patient's individual constellation of *p* factor symptoms. Or should clinical training start to largely avoid specialisation, and rather put the focus on broad expertise across psychopathology? Of course, there could be both specific treatments (and not all variance in psychopathology is explained by '*p*') and general transdiagnostic treatments.

This issue's highly informative practitioner review focuses on post-traumatic stress disorder (PTSD) (Smith, Dalgleish, & Meiser-Stedman, 2019). This review delivers, amongst many other things, a comprehensive overview of the effective treatments for PTSD within psychological interventions, which can include trauma-focussed CBT, cognitive therapy for PTSD and prolonged exposure. Prolonged exposure can involve imaginal exposure of the trauma memory. One wonders to what extent this specific treatment, shown to be effective for trauma, can generalise to all other forms of psychopathology. As pointed out elsewhere, the evidence for the *p* factor model is fairly new and thus caution is needed when considering possible treatment implications (Caspi & Moffitt, 2018). One specific constructive recommendation is for clinicians, where possible, to assess for an array of symptoms of psychopathology beyond the presenting complaint (Lahey et al., 2017).

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