Bifactor model of Psychopathology and the Impact of Routine Intervention on the General Psychopathology Factor within a Child and Adolescent Sample

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Thesis Declaration form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

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Overview

Part 1 – The Conceptual Introduction:

The conceptual introduction reviews the current evidence in order to better understand the extent to which the a general psychopathology factor is supported within a child and adolescent sample. Papers exploring the bifactor model of child and adolescent mental disorder were included in the review. Variations in factors included in the final models and the models' validity and stability over time were also explored. The limitations of the studies were considered in terms of their generalisability to clinical practice in the NHS, therefore setting up the rationale for future research for focus of the empirical paper.

Part 2 – The Empirical Paper

The empirical paper aims to expand on the current evidence base supporting the notion of a general psychopathology factor within a child and adolescent sample. As well as exploring the potential impact of routinely administered intervention on the expression of child and adolescent psychopathology. The study utilised the Child Outcomes Research Consortium dataset to explore the best fitting model of psychopathology and the extent to which intervention predicted model factor scores at follow-up.

Part 3 – The Critical Appraisal

The critical appraisal addresses some of the issues related to the use of secondary data that arose while undertaking the research project. This includes the impact of data quality and completeness on the direction of the research and data analysis. As well as the how secondary data may impact on the extent to which the research may address the limitations of previous research and extend the current evidence base.

Impact Statement

Mental health treatment is largely based on symptoms exhibited rather than root cause. However, it has been consistently shown that disorders often co-occur and diagnoses often share common symptoms. The discovery from this research project adds to the already overwhelming support of the general psychopathology factor. Suggesting that the high levels of co-morbidity may be due to all mental disorder being influenced by a broad set of aetiologic factors. This new way of conceptualising mental disorder has added to our understanding of how psychiatric disturbances tend to unfold across years of development and that disorder specificity increases with age. Which in turn could the influence the approach the UK's National Health Service (NHS) has to research and treatment. Knowledge of a general psychopathology factor highlights the importance of considering an individual's natural tendency towards psychopathology. This brings into question the current structure of NHS organisations and treatment approaches, which are diagnostically driven. Whereas, this research project and prior research suggests that a transdiagnostic approach may be favourable to reduce overall psychopathology. Therefore services may be better arranged around key risk factors that are relevant to multiple diagnostic categories.

Further, findings from this research project highlight the need for further research which include a greater range of child and adolescent psychopathology. For example including more severe psychosis symptomology and life span conditions such as Autistic Spectrum Disorder. As well as including samples from a large and inclusive clinical sample. This will help ensure that the identified model truly reflects child and adolescent psychopathology and is generalisable to a typical NHS service. Additionally, more detailed exploration of the impact of routinely administered interventions is needed to fully understand its potential impact on the structure and expression of psychopathology over time. This will help expand our current knowledge as to the mechanism by which therapeutic intervention impacts on

psychopathology. For example does it just impact on the core symptoms which are captured by the general psychopathology, or whether targeted intervention will impact on the specific factors.

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Part 1: Conceptual Introduction

Bifactor Model of Psychopathology and the General Psychopathology Factor: A Review of the Child and Adolescent Literature

Abstract

This paper will explore the notion that there may be a common set of underlying aetiological factors that may influence all mental disorders. It will first outline the limitations of the current categorisation of mental disorder, including the high rates of comorbidity. It will then explore the shift towards a structural model of psychopathology, which postulates that the observable characteristics of mental disorder could be organised in to higher order structures.

Of particular interest were the identified two- and three-factor models, which were also found to have high correlation between their higher order structures. Suggesting they are influenced by a widely shared set of aetiological factors, which could be understood in the context of a general psychopathology factor. In methodological terms the general psychology factor has been identified using a bifactor model, where each characteristic loads on two factors, one general factor and one specific factor which is related to a subset of items. This way of conceptualising mental disorder could influence the approach the UK's National Health Service (NHS) has to research and treatment.

The review of the current evidence in support of a general psychopathology factor within a child and adolescent sample will include the exploration of the variation in the structure of the bifactor model identified. As well as the validity of the model and the stability of the model over time. Additionally, what these findings mean in terms of support for the general psychopathology factor, development of psychopathology and treatment implications, will be considered. Finally, the limitations of the studies will also be discussed in terms of their generalisability to clinical practice in the NHS, therefore setting up the rationale for future research.

Introduction

The child and adolescent period, which in mental health services is defined as a person between the ages of zero to 18, is a time of marked social, biological and psychological development (Sawyer, Azzopardi, Wickremarathne & Patton, 2018). Worldwide, the prevalence of mental disorders in children and adolescents is approximately 13.4% (Polanczyk, Salum, Sugaya, Caye, & Rohde, 2015). Further, 50% of mental disorders present in adulthood are present already in some form by age 14, with ¾ there by age 25 (Murphy, 2012). Indicating the need to take a developmental perspective when searching for a better understanding of the cause, dimensions and progression of mental disorder. Additionally, in light of the high levels of comorbidity and shared common components within existing diagnoses (Kotov, Krueger, Watson, Achenbach, Althoff, Bagby et al., 2017), it may also be necessary to explore the possibility of a transdiagnostic causal pathway to optimise prevention and treatment strategies.

Diagnostic categories

The most important feature of any system of classifying or structuring mental disorder is that it helps clinicians to identify the presenting difficulty as quickly as possible, while also providing clinically useful information about treatment and management (Brodbeck, Stulz, Itten, Regli, Znoj, & Caspar, 2014). Therefore, mental disorder has traditionally been thought about as distinct disorders, where disorders are clearly distinguished from one another and from normal functioning. This conceptual framework can be useful for research as it is used to guide empirical studies and health-care delivery as it can be used to make prognoses and treatment decisions (Caspi, Houts, Belsky, Goldman-Mellor, Harrington, Israel et al., 2014). Further, evidence suggest that there is high consistency across countries and languages with regards to the classification systems used by mental health

professionals (Reed, Roberts, Keeley, Hooppell, Matsumoto, Sharan et al., 2013). However, the structure of those systems do not appear to be in keeping with the two most widely used diagnostic manuals (ICD-10 and DSM-IV). This finding bring into question the value of these diagnostic manuals and highlights the widespread criticism of the use of diagnostic categories in general.

Firstly, the use of diagnostic categories ignores the substantial evidence that proposes that psychopathology exists on a continuum with normal functioning (Kotov et al., 2017). For example it has been repeatedly shown that externalising liability is best modelled as continuously normal in distribution (Carragher, Krueger, Eaton, Markon, Keyes, Blanco, et al., 2014; Markon & Krueger, 2005). Acknowledgement of these consistent findings can be seen in the move towards a more dimensional representation of mental disorder in the DMS-5, for example the introduction of Autism Spectrum Disorder (ASD) to replace Asperger's Syndrome, Autistic Disorder and pervasive developmental disorder-not otherwise specified (Noordhof, Ormel, Oldehinkel, & Hartman, 2015). Further, traditional diagnoses generally show limited reliability, for example their inability to predict therapeutic response (Kapur, Phillips, & Insel, 2012) and that 40% did not meet the level for acceptable interrater reliability (Regier, Narrow, Clarke, Kraemer, Kuramoto, Kuhl, et al., 2013). It has also been shown that, potentially due to the polythetic nature of diagnosis, many are quite heterogeneous; for example it has been found that in a large sample of psychiatric inpatients there were 170 different ways that major depressive disorder could be diagnosed (Zimmerman, Ellison, Young, Chelminski & Dalrymple, 2015). It maybe for this reason that there have been challenges in identifying one-to-one causal risk mechanisms for specific disorders (Rutter, 2013). Additionally, many patients do not meet the criteria for any disorder, even though they report experiencing distress and/or impairment (Kotov et al., 2017).

However, potentially the most contentious issue it the high rate of comorbidity. A meta-analysis showing substantial co-occurrence of psychiatric disorders, estimating that between 15-75% of depression diagnoses carry a comorbid anxiety diagnosis (Angold, Costello, & Erkanli, 1999). Further, it has been shown that disorders in adolescence and later life often co-occur and specified mental disorders are known to share symptoms and aetiological factors (Lahey, Applegate, Waldman, Loft, Hankin, & Rick, 2004). Overall, research has demonstrated a rough rule of 50%, with half of individuals who meet diagnostic criteria for one disorder also meet diagnostic criteria for a second, then half with two disorder will meet criteria for a third and so on (Newman, Moffitt, Caspi, & Silva, 1998). Therefore comorbidity is a particular challenge when thinking about the value of diagnostic categories, as not only can it complicate clinical decision making, it can also impact the results of clinical studies and affect treatment decisions (Kotov et al., 2017). In light of these criticisms there has been renewed interest in the nosology of mental disorders, particularly the similarities and differences among disorders, the structure of their relationship, and understanding the diversity in presentation in the common disorders (Blanco, Wall, He, Krueger, Olfson, Jin, et al., 2015).

Structural models of mental disorder

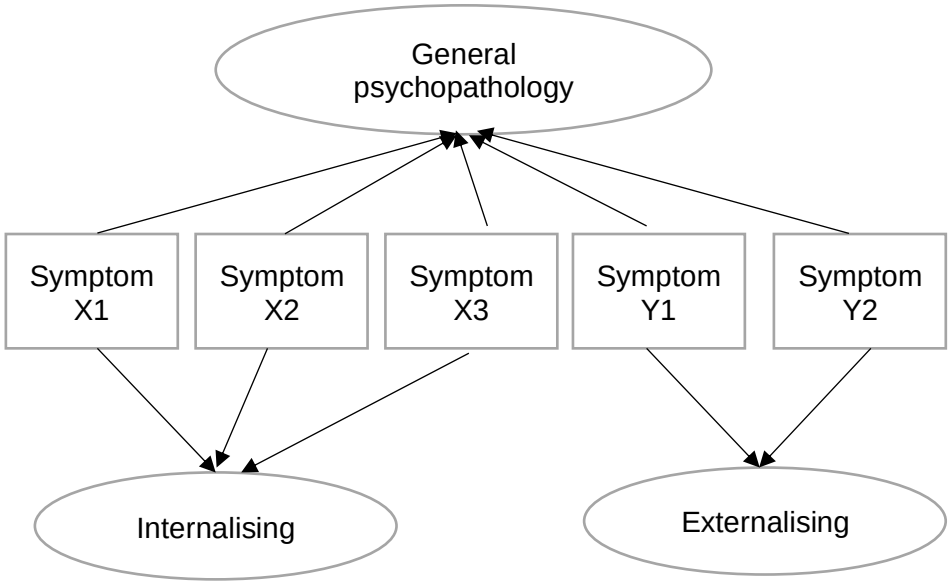
One potential solution to the limitation of the traditional diagnostic categories is quantitative nosology, which measures psychopathology through factors analysis of the symptoms (Kotov et al, 2017). This area of research has been based upon the work of Achenbach (1966) who identified two broad transdiagnostic factors: internalising and externalising, which is now known as the two-factor model, and accounts well for co-occurrence of many forms of mental disorder. The internalising factor represents comorbidity among mental disorders such as major depression, generalised anxiety, panic, agoraphobia and obsessive compulsive disorder.

Whereas the externalising factor represents comorbidity among conduct disorders, anti-social personality and substance use (Kim & Eaton, 2015). Genetic research also provides support for these transdiagnostic factors; for example Kendler, Prescott, Myers, & Neale (2003) found that particular genetic risk factors lead individuals to be more susceptible to either internalising or externalising disorders. Additionally, evidence supports differing age and gender profiles for these two dimensions. Individuals experiencing externalising disorders are more likely to be younger and male, whereas adolescents and females have a higher prevalence of internalising symptoms (Martel, 2013). However, these initial findings have been based upon diagnosis level data of relatively common disorders rather than more homogeneous symptom level data, which may provide more detailed insights into the structure of psychopathology. Therefore, when dimensional factor analytic approaches were applied to symptom level data it was found that the internalising factor can be further divided into fear and distress factors (Krueger, 1999), referred to as the three-factor model. In the three-factor model the distress factor includes mood disorders and generalised anxiety, which can be differentiated from a fear factor, which including phobias and obsessions and compulsions. Further criticism of these structural models is that they are based upon relatively common forms of mental disorder, whereas more severe disorders may represent a distinct form of psychopathology. In light of this further research identified a thought disorder factor, which encompasses psychotic disorders, cluster A personality disorders and bipolar I disorder (Markon, 2010), however, it did not replicate the finding of internalising differentiating into a fear and distress factor (Noordhof et al., 2015).

A review of the literature on structural models of psychopathology (Krueger & Markon, 2006a) found that the 3-factor model provides the best fit of the factor structure of 11 mental disorders in multiple samples. However, there was still shown to be substantial correlation between these specific factors (Krueger & Markon,

2006b). This correlation could be due to the fact that they are all influenced by a broadly shared set of aetiologic factors that are distinct from the ones that cause the specific disorders to correlate together on the higher order factors (Lahey, Applegate, Hakes, Zald, Hairi, & Rathouz, 2012), a hypothesised 'general psychopathology factor'. This can be understood by thinking about cognitive ability. Even though cognitive abilities can be differentiated into separate abilities (verbal, visuospatial, working memory or processing speed), a general factor indicates that individuals who do well on one test are likely to do well on the other tests (Jensen, 1998).

Figure 1: Bifactor model of psychopathology



Exploring the 'general factor' approach to psychopathology involves fitting a bifactor measurement model to psychopathology data (Murray, Eisner, & Ribeaud, 2016). Previous studies which have aimed to better understand the 'general factor' approach to psychopathology have failed to account for the potential independence of the factors (Subica, Allen, Frueh, Elhai, & Fowler, 2016). Bifactor analyses address this constraint as the specific factors (for example internalising and externalising) are not allowed to correlate with each other, as it requires that covariation between specific factors is captured entirely by the general factor (Brown, 2015). Therefore the

basic structure of the bifactor model (see Figure 1) enables each item to load on two factors: one general factor which is associated with all factors and one specific factor which is associated to a subset of items. The relationship between these subset of items is not fully captured by the general factor (Murray et al., 2016).

The bifactor model of psychopathology

The presence of a general psychopathology factor was initially identified by Lahey et al (2012) using a large representative sample of American adults. They identified that a model specifying three correlated specific factors of externalising, fear and distress plus the general psychopathology factor fitted the data best. This model was also replicated in a separate sample of American adults (Greene & Eaton, 2017). The model was also supported by external validity analysis, which showed that the fear, distress and externalising factors were differently associated with multiple external factors; for example fear with a past history of sexual abuse, distress with a family history of depression and externalising with antisocial behaviour. Further, the general factor accounted for an independent variance in future psychopathology, functioning and self-harm (Lahey et al., 2012). A subsequent study also identified the general psychology factor in a longitudinal study of New Zealand adults (Caspi et al., 2014). However, they identified that a model specifying specific factors of internalising, externalising and thought disorder and a general psychopathology factor fitted the data best. This finding was also replicated in a clinical sample of Danish adults who were survivors of childhood sexual abuse (Hyland, Murphy, Chevlin, Carey, Vallières, Murphy et al., 2018). Caspi et al (2014) also found that for some disorders much of the tendency for them to persist is indicative of a general psychopathology factor.

The robust identification of a general factor of psychopathology could influence the way that mental disorders are researched and treated, as it may lead to

a hierarchical conceptualisation of psychopathology in which the aetiology and psychobiological mechanism are conceptualised as both shared and dimension-specific (Lahey, Rathouz, Keenan, Stepp, Loeber, & Hipwell, 2015). It is for this reason that research may have not been able to identify aetiological factors that are linked to one specific psychiatric disorder but not another, in fact all risk factors were primarily associated with the general factor, meaning researchers are unlikely to find singular disorder loyalty in biomarkers (Caspi et al., 2014). Hyland et al (2018) have also considered some of the theoretical and clinical implication of the identification of a general factor in adults, which are important to consider within the context of the NHS. Most importantly if the general factor does exist comorbidity is inevitable, regardless of how precisely defined and delineated disorders are. This only serves to reinforce the above criticism of diagnostic manuals, such as the DSM-IV, which is widely used to inform NHS service structure, treatment recommendations and care provision. Brining to question the validity of these organisational structures. Further, treatments should be equally effective for all disorders within the same dimensions, potentially indicating a transdiagnostic approach is favourable to reduce overall psychopathology (Hyland et al., 2018). However, this is in contrast to how UK treatment guidelines, such as those developed by the National Institute of Clinical Excellence (NICE), are developed and consequent treatment recommendations made. This is despite promising evidence for the use of transdiagnostic approaches (Farchione, Fairholme, Ellard, Boisseau, Thompson-Hollands, Carl, et al., 2012).

However there have also been some criticisms as to the validity of the general factor itself. One alternative hypothesis is that the specific factors load on the general factor because the same symptoms are used to define multiple specific factors. However, when the overlapping symptoms were removed from analysis the bifactor model specifying two specific factors of internalising and externalising and a general psychopathology factor still fitted the data best (Lahey, Zald, Perkins, Villalta-Gil,

Werts, Van Hulle et al., 2018), indicating that this is unlikely. Further it was thought that the general factor may be an artefact of systematic measurement bias (Lahey, Krueger, Rathouz, Waldman & Zald, 2017). However it has been shown that, even after accounting for the variance associated with external and internal factors, the general factor was related to independent measures of impaired school functioning (Lahey et al., 2015), indicating that the general factor is not just a manifestation of systematic measurement biases. Although it appears that these alternative hypotheses are unlikely further evidence would need to be collected in order to determine the robustness of the general factor across various population and across the life course.

General psychopathology factor in children and adolescents

The identification of a general factor of psychopathology is a relatively new area of mental health research. Therefore to better understand the extent to which the current evidence supports the notion of a general psychopathology factor across the life course, a search of the literature was undertaken. The literature review was undertaken within PubMed on the 6th January 2020 and the search terms used were: ((‘singular model’ OR ‘general psychopathology factor’ OR ‘general factor’ OR ‘p-factor’) AND (‘mental disorder’ OR ‘mental health problems’ OR ‘Psychopathology’ OR ‘mental illness’ OR ‘clinical disorder’) AND (‘bifactor’ OR ‘bi-factor’)). This search identified 51 articles (see Appendix 1). Nine were found not to be relevant; 19 used bifactor modelling, but not in the context of looking at a ‘general psychopathology factor’; and three offered a narrative review of the literature. Therefore 19 relevant papers were identified and examination of references identified an additional 10 papers. Given that 50% of mental disorders that emerge in adulthood are present already in some form by the age of 14 (Murphy, 2012), the review was focused on a

child and adolescent sample. Therefore, of these 29 studies, seven were excluded as they looked at the structure of psychopathology within an adult sample.

The 22 included studies were from varied geographical locations including America (Hankin, Davis, Snyder, Young, Glynn & Sandman, 2017; Lahey et al., 2015; McElroy, Belsky, Carragher, Fearon, & Patalay, 2018; Olino, Bufferd, Dougherty, Dyson, Carlson, & Klein, 2018; Snyder, Young, & Hankin, 2017, 2019; Tackett, Lahey, Van Hulle, Waldman, Krueger & Rathouz, 2013) the Netherlands (Bloemen, Oldehinkel, Laceulle, Ormel, Rommelse, & Hartman, 2018; Laceulle, Vollebergh, & Ormel, 2015; Noordhof et al., 2015), Canada (Afzali, Sunderland, Carragher, & Conrod, 2018; Haltigan, Aitken, Skiling, Henderson, Hawke, Barraglia et al., 2018); Britain (Black, Panayiotou, Humphrey, 2019; Brodbeck et al., 2014; Constantinou, Goodyer, Eisler, Butler, Kraam, Scott et al., 2019; Patalay, Fonagy, Deighton, Belsky, Vostanis, & Wolpert, 2015), multiple European sites, including Britain (Castellanos-Ryan, Brière, O'Leary-Barrett, Banaschewski, Bokde, Bromberg et al., 2016; de la Cruz, Vidal-Ribas, Zahreddine, Mathiassen, Brøndbo, Simonoff et al., 2018), Switzerland (Murray et al., 2016), Sweden (Pettersson, Lahey, Larsson, & Lichtenstein, 2018), Australia (Carragher, Teesson, Sunderland, Newton, Krueger, Conrod et al., 2016), and Brazil (Martel, Pan, Hoffmann, Gadelha, do Rosário, Mari et al., 2017). However, only three of the studies (Constantinou et al., 2019; de la Cruz et al., 2018; Haltigan et al., 2018) used data from a clinical sample.

The structure of the bifactor model

Most studies applied the same criteria to assess good model fit (RMSEA > 0.06, CFI and TLI < 0.95, (Hu & Bentler, 1999), however a couple used the acceptable fit criteria (CFI and TLI < 0.90). In keeping with these criteria, all but one of the 22 studies found that a bifactor model of psychopathology fitted the data best, when

compared to a single, two or three factor model. However, the specific factors included in the model varied.

Two factor model.

Nine studies identified that a model specifying two specific factors of externalising and internalising plus the general psychopathology factor was the best fit (Castellanos-Ryan et al., 2016, Hankin et al., 2017; Laceulle et al., 2015; Lahey et al., 2015; Olino et al., 2018; Patalay et al., 2015; Snyder et al., 2017, 2019; Tackette et al., 2013). Only one of these studies used symptom level data (Patalay et al., 2015), this was a British based study which used item level data from the Strengths and Difficulties Questionnaire (SDQ) (Goodman, Meltzer, & Bailey, 1998) and the Me and My School Questionnaire (Deighton, Tymms, Vostanis, Belsky, Fonagy, Brown et al., 2013), both of which have good validity and reliability.

Three factor model.

Three studies found that a model specifying three specific factors of internalising, externalising and thought disorder (Afzali et al., 2017; Carragher et al., 2016); or fear, distress and externalising (Martel et al., 2017) plus a general psychopathology factor fitted the data best. Two of these studies used symptom level data (Afzali et al., 2017; Carragher et al., 2016), both used the SDQ (Goodman et al., 1998) and the Brief Symptom Inventory (BSI) (Derogatis & Melisaratos, 1983); however Carragher et al (2016) added additional items from an abbreviated version of the Rutgers Alcohol Problem Index (White & Labouvie, 1989). Again all scales were well validated for use in an adolescent sample. Only the two studies which identified a specific thought disorder factor actually included an instrument measuring more severe forms of psychopathology, such as psychotic symptoms (Afzali et al., 2017; Carragher et al., 2016), indicating the potential influence of included symptoms on the

variance in the specific factors included in the model. Further, these three studies allowed the specific factors to correlate (Afzali et al., 2017; Carragher et al., 2016; Martel et al., 2017), which is not consistent with a classic bifactor model (Brown, 2015) and may impact on the models validity. The findings from the above 12 studies are broadly in keeping with those from the adult samples.

Alternative models.

The final nine studies identified either a three or four factor bifactor model, which was not in line with the adult findings. The identification of a four factor bifactor model may be partially explained by the fact that four of the studies included ASD and attention deficit hyperactivity disorder (ADHD) symptoms, as across these studies it has been consistently shown that these symptoms form a separate specific factor. Therefore the studies identified that a model specifying four specific factors of internalising, externalising, ADHD and ASD (Bloemen et al., 2018); aggression, ADHD, prosocial and internalising (Murray et al., 2016); internalising, externalising, attention and orientation and ASD (Noordhof et al., 2015); or inattention, conduct, emotionality and impulsivity (Pettersson et al., 2018) plus the general psychopathology factor fitted the data best. These findings bringing into question whether ASD and ADHD symptoms should be included in order to develop a more inclusive model of psychopathology.

Two of the other inconsistent finds may be explained by the measure of psychopathology used, as both studies used symptom level data of the Child Behaviour Checklist (CBCL), from which a factor structure analysis has indicated a separate attentional domain (Achenbach & Rescorla, 2001). It could be for this reason that an additional attention factor was included in the model for both studies (internalising, externalising and attention (McElroy et al., 2018) and internalising, externalising, thought disorder and attention (Haltigan et al., 2018)). This finding was

also replicated in the study by Noordhof et al (2015), who included the attentional subscales of the CBCL. It could therefore be that the use of detailed and highly inclusive symptom level data enables identification of more subtle differences between the subfactors.

One study identified a model specifying three specific factors of hopelessness – suicidal ideation, generalised worry and restlessness – fatigue, in addition to the general psychopathology factor (Brodbeck et al., 2014). With another identifying, that a model specifying three specific factors internalising, externalising and wellbeing and a general psychopathology factor fitted the data best (Black et al., 2019). However, a potential explanation for this finding is that this was the first study to explore the latent structure of mental health in combination with wellbeing. The final study identified a best fitting bifactor model with a general psychopathology factor and four specific factors (anxiety, mood, antisocial and attentional) (Constantinou et al., 2019). The identification of a specific antisocial and attentional factor may be in part accounted for by the population utilised within the analysis. Data was extracted from the START trial, which specifically recruited participants with aggressive, antisocial and violent behaviour (Fonagy, Butler, Cottrell, Scott, Pilling, Eisler et al., 2018), which may account for why these items may have been expressed more strongly.

Non bifactor model.

The only study to not support the bifactor model of psychopathology used data from a clinical sample. The study found that a five-factor structure fitted the symptom level data better than either a two factor structure or a classic bifactor model (de la Cruz et al., 2018). One possible explanation offered by the authors is that the expression of symptoms in low risk populations may be unspecified whereas in a high-risk population the specificity of symptoms for each disorder increases, therefore the two factor model maybe to simplified for a clinical population (de la Cruz et al., 2018).

Although this finding was replicated across two separate clinical samples, the study only utilised one instrument designed to measure of psychopathology. Further, the selection of items was not informed by a recent structural analysis undertaken on the instrument (Goodman, Lamping, & Ploubidis, 2010), which indicated that the reverse scored items should be removed and internalising is represented only by the emotional and peer scale items and externalising assessed using the conduct disorder and hyperactivity scale items. Therefore further research would be needed to see if utilising the specified subset of the questions or augmenting the items with a different instrument may disprove these findings, this is particularly given that the other study to utilise a clinical sample did support a bifactor model (Haltigan et al., 2018).

Validity of the bifactor model

Eight of the studies explored the validity of the models by comparing psychopathology to external factors such as personality traits and temperament, school functioning, depravation and executive functioning.

Personality facets.

Such external validity analysis indicated that the general psychopathology factor demonstrates significant association with a multitude of personality facets (Carragher et al., 2016; Castellanos-Ryan et al., 2016). In contrast the specific factors were associated with specific personality facets, for example high levels of internalising symptoms was associated with hopelessness, negative thinking, anxiety sensitivity and low sensation seeking, whereas high levels of externalising was associated with impulsivity and sensation seeking (Carragher et al., 2016). The general psychopathology factor was also found to be associated with low effortful control (poor attention and self-regulation) and high negative affectivity (greater

propensity to negative mood) (Hankin et al., 2017; Tackett et al., 2013). However, as with the personality facets, the specific factors are associated more strongly with particular traits, for example internalising symptoms are associated with higher negative affect, low positive affect (Hankin et al., 2017) and low daring (Tackett et al., 2013), whereas externalising symptoms are associated with low effortful control (Hankin et al., 2017), low prosociality, high daring and negative emotionality (Tackett et al., 2013). These findings suggest that although certain personality traits and temperament may provide broad based transdiagnostic risk to general psychopathology; particular personality traits and temperaments may be more strongly associated with specific psychopathology syndromes potential indicating a need for trait-focused interventions (Castellanos-Ryan et al., 2016).

School functioning and depravation.

The general psychopathology factor was also found to be related to independent measures of school functioning, such as difficulties in academic performance and in meeting behavioural demands of the classroom (Lahey et al., 2015; Patalay et al., 2015) and social depravation (Patalay et al., 2015). Lahey et al (2015) also found that the internalising factor was positively correlated with teacher rated academic performance and adaptive school functioning. Conversely, Patalay et al (2015) found that when the general psychopathology factor was taken into account the association with educational attainment and social depravation and internalising disappeared. On the other hand, the external factor maintained its positive association with social depravation (Patalay et al., 2015) and negative association with academic performance (Lahey et al., 2015; Patalay et al., 2015). These findings suggest that internalising is associated to education and social problems only to the extent that they are linked with a general vulnerability to psychopathology, whereas the externalising is independently associated to both. Child global executive functioning

was also significantly correlated only with the general psychopathology factor, underscoring the possibility of shared aetiology between mental health and cognition (Martel et al., 2017), which is consistent with the idea that executive functioning deficits are present in several mental disorders. Additionally these external relationship adds weight to the notion that the general factor is likely to be measuring something over and above shared source variance. However, Internalising and externalising problems do not show much impairment in executive functioning beyond that captured by the general factor, except for a strong association between cognitive flexibility and internalising specific factor (Bloemen et al., 2018).

Model stability

Only eight of the 22 papers utilised a longitudinal data set in order to be able to establish both homotypic continuity, the recurrence of the same diagnosis over time, and heterotypic continuity, the occurrence of a different diagnosis over time, within the latent structure of psychopathology.

Across adolescents.

Four of the eight studies examined the stability of the bifactor model from early to mid-adolescence, with all studies indicating that the structure of psychopathology remains relatively stable across this time period (Brodbeck et al., 2014; Castellanos-Ryan et al., 2016; Noordhof et al., 2015; Snyder et al., 2017). Specifically, Snyder et al (2017) found that each factor at age 13 predicted the same factor 18-months later at age 15, for example general psychopathology at 15 was predicted by general psychopathology at 13. They also found that the stability of internalising problems increased with age. Further Castellanos-Ryan et al (2016) showed that there was substantial correlation between factors at age 14 and 16. However there was slight variance in the contribution of psychopathology symptoms overtime, for example drug

and tobacco use became stronger with increased age. One study, which assessed its participants over eight time points from entering school to late adolescence also showed that the strength of the general and specific factors varied very little, this is despite it being a time of social, biological and psychological development (Murray et al., 2016). The findings of all these studies are indicative of homotypic continuity rather than heterotypic continuity. This has important implication for clinical practice, particularly early screening to identify those at risk, as although level of psychopathology may change with age, these studies suggest that individual who experience high levels relative to their peers early on are likely to continue to experience high levels relative to peers (Snyder et al., 2017). However, when this stability is compared to other traits, such as intellectual ability, the correlations across time are far lower (general psychopathology at approximately 0.26, Murray et al (2016) verses intellectual ability at approximately 0.70, Tucker-Drob and Briley (2014)), suggesting that general psychopathology manifests in a comparatively more episodic fashion.

Two of the eight studies did not look at the stability of the structure of psychopathology across adolescents, but did assess mental health outcome at later time points. Findings of these studies supported heterotypic continuity, with both indicating that the general factor of psychopathology predicted adverse mental health outcomes over time (Patalay et al., 2015; Pettersson et al., 2018). This finding was also replicated by Brodbeck et al (2014), who found that the general psychopathology factor at age 14 predicted new a persistent internalising disorders three years later at age 17 and was associated with externalising disorders such as substance use. Further, they found that the specific factors were able to differentiate affective, anxiety and behaviour disorders. For example hopelessness was associated with affective disorders at age 14 and predictive of new and persistent affective disorders at age 17, whereas the generalised anxiety factor was able to differentiate anxiety and

behavioural disorders where high generalised worry at 14 predicted persistent anxiety at 17 and low generalised worry predicted behavioural disorders.

Only one study utilised a clinical sample to examine within person change in bifactor model over an 18 month intervention (Constantinou et al., 2019). The results showed that the mood and attention factors did not change over time; were as there was a reduction in the general psychopathology and antisocial factors and an increase in the anxiety factor. These findings may indicate that psychological therapies have a universal effect, targeting the core functional mechanism in psychopathology (Caspi & Moffitt, 2018). However, as previously mentioned the study utilised a very specific clinical group and intervention which may limit the generalisability of these findings.

From early childhood.

Two of the eight studies looked at data from early childhood; however, due to participant age both studies used parent report rather the child's own psychopathology rating. The most comprehensive of the two studies followed children up from age two to age 14 (McElroy et al., 2018). Again this study found significant homotypic continuity as the variance explained by the general and specific factors remained consistent over time, with the majority of the variance being explained by the general factor. However, there were also significant individual level changes in the way psychopathology was expressed, which all emerged from the general factor. This latter finding is suggestive of heterotypic continuity. For example the general factor predicted all three specific factors and was itself predicted by earlier measurements of internalising and externalising, indicating that specific presentations of psychopathology increases the risk of comorbidity over time and vice versa (McElroy et al., 2018).

The second study assessed participant at age three and followed up at age six, finding that the same bifactor structure fitted the data at both time points, with standard regression coefficients having moderate to large effects (Olino et al., 2018). However, as with the previous study further findings were also suggestive of heterotopic continuity, as the general psychopathology factor at age three was positively associated with externalising age six, suggesting that early common psychopathology differentiates into more specific externalising difficulties in later development. These two studies looking at early childhood indicate that both processes of hetero- and homo-typic continuity maybe occurring in parallel. However one hypothesis is that heterotypic continuity may in fact be due to homotypic continuity in latent psychopathology factors, so due to continuity in common psychopathology (Snyder et al., 2017). For example, an individual may have a propensity to experience certain core symptoms which then lead to secondary issues; however, the phenotypic expression of these issues may change over time depending on particular circumstance (Murray et al., 2016).

Conclusions

Overall, despite the slight variation in model structure, the studies indicate overwhelming support for the bifactor model and a general psychopathology factor within child and adolescent psychopathology. However, potentially concerning is the little remaining variance in psychopathology not explained by the general psychopathology factor (e.g. Martel et al., 2017), which may indicate that a one-dimensional model might be a better fit. Despite this the bifactor model has consistently be found to be a better fit to the data, suggesting the specific factors are meaningful but that potentially different instruments or a more inclusive clinical sample are needed to capture their unique input to the structure of psychopathology.

Further, as previously mentioned, the identified external relationships are suggestive of the fact that the 'general psychopathology factor' is capturing something more than just shared variance. These findings indicated a relationship to particular personality traits, such as poor effortful control and high negative affectivity, as well as difficulties in academic performance and negative classroom behaviour. Caspi and Moffitt (2018) hypothesise that these relationships indicate that psychopathology may represent a diffuse unpleasant affective state, often termed neuroticism or negative emotionality or that the core functional mechanism in psychopathology is poor impulse control over emotions or that deficits in intellectual function characterises psychopathology. However, the hypothesis they favour is that psychopathology captures the disordered form and content of thought that permeates the extreme of practically every disorder (Caspi & Moffitt, 2018). Therefore any individual with a high propensity to psychopathology might, if their disorder grows severe enough, experience unwanted irrational thoughts. Indicating that symptoms are not disorder specific and that a continuous rather than discreate notion of mental disorder may be a more accurate way to understand, research and treat mental disorder.

Findings from the longitudinal studies suggests that the identification of a general psychopathology factor indicates that psychiatric disturbances express themselves across years of development as persistent and comorbid (Caspi et al., 2014). Further, that disorder specificity increases with age, as psychopathology is increasingly expresses in particular ways (Patalay et al., 2015). For example many children exhibit diffuse emotional and behavioural problems, with fewer developing a brief episode of an individual disorder, fewer still developing a persistent internalising and externalising syndrome, and only a very few exhibiting sever conditions such as psychosis (Caspi & Moffitt, 2018). The developmental progression for psychopathology may be suggestive of an early intervention approach to the treatment and management of child and adolescent of mental disorder. Therefore

intervening at the point at which children exhibit defuse emotional and behavioural problems. However, this could lead to the unnecessary pathologising of normal childhood emotional and behavioural expression which could have a negative impact on the young person in terms of sigma of accessing mental health treatment or harm of receiving unneeded treatment. It could therefore be that early intervention could take an approach of watchful waiting rather than intervention, but the cost implications to already stretch NHS service and impact on the young person in terms of constant monitoring need to be considered.

The relationship between the general psychopathology factor and liability to mental disorder and vice versa could indicate further treatment implications. For example, it could be that an initial test of childhood psychopathology could provide a useful tool to estimate general risk of psychopathology over time (Afzali et al., 2018). The general psychopathology risk profile could then be used alongside a separate assessment of specific risk for internalising, externalising and thought disorder in order to provide detailed information about a patients personalised risk profile to guide clinical decision making and inform an intervention tailored to their specific needs (Carragher et al., 2016). This is very much in keeping with the move towards more client centred care within NHS services. For example, if a person scores high on the general psychopathology factor and moderate on a particular specific factor, it could be that they would more likely benefit from a transdiagnostic approach to treatment (Afzali et al., 2018). A move towards a transdiagnostic treatment approach is in keeping with the recommendations made from the adult findings (Hyland et al., 2018). Further, those with greater internalising difficulty may benefit more from treatment that targets negative thinking and anxiety sensitivity, whereas those with externalising difficulties may benefit from treatment that more strongly targets impulsivity and sensation seeking (Carragher et al., 2016). However, as mentioned above, this is in contrast to how therapeutic services and interventions are provided within the UK,

which have tendency to be diagnostically driven rather than based upon current symptomatology.

Limitations

There are a number of study limitations which impact on the generalisability of the above conclusions. Firstly, only a small proportion of the studies used more homogeneous symptom level data, instead of diagnosis level data. This is important as the use of symptom level data helps avoid the issue of artefactual co-morbidity, co-morbidity which arises due to different diagnoses sharing some symptoms (Murrary et al., 2016). The comparability of the studies are further complicated by the variation in the range of child and adolescent disorders included in the studies, for example Afzali et al (2018) did not include ASD and separation anxiety, Carragher et al (2016) did not include obsessive compulsive disorder and ASD and Patalay et al (2015) did not included psychosis. This brings into question whether the identified models truly represent the structure of child and adolescent psychopathology, particularly as those studies which include more complex disorders such as psychosis (Afzali et al., 2018) and lifespan conditions such as ASD (Bloemen et al., 2018) identified slight variations in the structure of the best fitting bifactor model.

Secondly, only two of the 20 studies utilised data from a clinical sample. Although a community sample tend to be more representative of the wider population, due to reduced selective sampling (Alfazil et al., 2018), they may not be representative of the NHS population. Particularly because community samples show a limited range of severity, whereas when included increased severity may impact the factor structure (Carragher et al., 2016) or may disprove the notion of the general factor all together (de la Cruz et al., 2018). It is also important to consider how the stability of the model may vary within a clinical sample (Snyder et al., 2017), which is potentially high-risk and seeking treatment. It is also important to note that to date

none of these studies has explored the impact of mental health intervention on the structure and expression of psychopathology over time.

Thirdly, when examining the longitudinal findings it is important to acknowledge that in all of the studies the general factor was obtained from cross-sectional data, meaning that the general factor expresses comorbidity at a given age, but the underlying meaning may vary developmentally (McElroy et al., 2018). Further, it does not directly inform about how symptoms may ebb and flow over time (Murray et al., 2016). Only one of the longitudinal studies utilised a very specific clinical sample (Constantinou et al., 2019), which is relevant when considering that the degree of stability is likely to differ between populations (Snyder et al., 2017). It could therefore be that both homo- and heterotypic continuity within the structure of psychopathology might vary in high-risk or treatment seeking samples. Further, given the very limited data regarding the impact of therapeutic intervention on the bifactor model it is not possible to make firm conclusions about the potential for change in the structure and presentation of psychopathology over time (Snyder et al., 2017). Lastly, the studies have mainly included American samples, with only four of the 20 studies including data from a British population. In light of the vast differences between American and British health care systems and general populations (Shi & Singh, 2014), it is questionable as to the extent the findings from American populations are generalisable to the British population and therefore an NHS setting.

Future research

Over the past decade there has been renewed interest in the nosology of mental disorders, with much of the research focusing on identifying the previously hypothesised general psychopathology factor. Given the rapidly expanding evidence base, within child and adolescent (Afzali et al., 2017; Black et al., 2019; Bloemen et al., 2018; Brodbeck et al., 2014; Castellanos-Ryan et al., 2016; Carragher et al., 2016;

Constantinou et al., 2019; Haltigan et al., 2018; Hankin et al., 2017; Lahey et al., 2015; Laceulle et al., 2015; Martel et al., 2017; McElroy et al., 2018; Murray et al., 2016; Noordhof et al., 2015; Olino et al., 2018; Patalay et al., 2015; Pettersson et al., 2018; Snyder et al., 2017, 2019; Tackett et al., 2013) and adult research (Caspi et al., 2014; Greene & Eaton, 2017; Hyland et al., 2018; Lahey et al., 2012; Lahey et al., 2017), it appears likely that the general psychopathology factor may help explain the high levels of co-morbidity within mental disorders and provide a framework for a more individualised approach to mental health treatment. In general mental health treatment has been approached differently to physical health treatment, in that it is largely based on symptoms exhibited rather than root cause (Patalay et at., 2015). In contrast the above studies, which support the notion of the general psychopathology factor, highlight the importance of considering an individual's natural tendency for psychopathology. This may involve considering an individual's developmental history, past psychopathology and current symptomology, rather than focusing solely on diagnostic categories and symptoms, to develop a patient's individualised profile and treatment plan (Carragher et al., 2016). Indicating that the identification of the general psychopathology factor promotes the development of interventions that target the shared aspects of disorders, but that can also be tailored to the individual need identified within the bifactor model (Brodbeck et al., 2014). This area of research may also help identify a sub-group of individuals with greater general psychopathology, who are more likely to experience psychopathology, and may be expected to transition through different diagnoses over their lifetime (Patalay et al., 2015).

Despite the clear implications the general psychopathology factor has for mental health treatment, it is also important to consider some of the current study limitations and how they might best be addressed to increase the generalisability of the finding to a UK health care setting. This would most importantly include expanding the evidence using a clinical sample within a British population. As well as building on

the longitudinal evidence base, with particular focus on how NICE recommended mental health intervention may, or may not, impact the structure of psychopathology over time. As research into the general psychopathology factor has focused on child and adolescent rather than adult sample, due it being a time of social, biological and psychological development, the empirical study will utilise routine data collected within Child and Adolescent Mental Health Services (CAMHS) across multiple sites within the UK. Symptom level data, from instruments validated within a child and adolescent population, will be used in an attempt to reduce artefactual co-morbidity. The study will also examine the impact of routine CAMHS intervention on the structure and phenotypic expression of psychopathology in early and later adolescents.

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Part 2: Empirical Paper

Bifactor model of Psychopathology and the Impact of Routine Intervention on the General Psychopathology Factor within a Child and Adolescent Sample

Abstract

Introduction. The general psychology factor has been consistently identified within both adult, and child and adolescent samples. However, predominantly community samples from a non-British population have been utilised, bring into question the generalisability of these finding to the NHS. This study will therefore aim to utilise a British clinical sample to add to the evidence and to explore the impact of interventions on the identified model of psychopathology.

Method. Data was extracted from a pre-existing national CAMHS dataset. Demographic data, intervention type and psychopathology data were included in the analysis. Data was analysed using SPSS and Mplus v8. Exploratory, confirmatory and bifactor analyses were undertaken to identify the best fitting model for the data. Variables were then entered into a hierarchical regression to predict factor scores at last assessment.

Results. At both first and last assessment a bifactor model comprising of four specific factors and a general psychopathology factor fitted the data best. When entered into a linear model the initial assessment factor scores were the best predictor of the final assessment factor score. Being offered an intervention only predicted the general psychopathology factor score.

Conclusion. The findings supported the notion of a general psychopathology factor within child and adolescent samples. Intervention only predicted the general psychopathology factor score at the last assessment, which brings into question what aspect of psychopathology the interventions are targeting. However, further research is needed to be able to make meaningful recommendations from these findings.

Introduction

The structural model of mental disorder

Over recent years there has been renewed interest in the nosology of mental disorder. This is potentially due to the wide spread criticism of the use of diagnostic categories; including their limited reliability and the high rates of co-morbidity (Kotov, Krueger, Watson, Achenbach, Althoff, Bagby et al., 2017). One potential solution is quantitative nosology, which is based upon the work of Achenbach (1966), who identified two broad transdiagnostic factors of mental disorder: internalising and externalising. Where the internalising factor represents comorbidity among disorders such as major depression, generalised anxiety, panic and agoraphobia and the externalising factor conduct disorder, antisocial personality and substance misuse (Kim and Eaton, 2015). Further research into the area identified that when symptom level data was used the internalising factor further divided into a fear (phobias) and distress (mood and anxiety disorders) factor (Krueger, 1999). Additionally, when more severe disorders were included in the analysis a thought disorder factor was identified (Markon, 2010). However, there was still shown to be substantial correlation between these specific factors, potentially due to the fact they are all influenced by a broad set of aetiologic factors, referred to as the 'general psychopathology factor' (Lahey, Applegate, Hakes, Zald, Hairi, & Rathouz, 2012). In methodological terms, the 'general factor' approach to understanding psychopathology involves fitting a bifactor measurement model to psychopathology data (Murray, Eisner, & Ribeaud, 2016).

The general psychopathology factor

The presence of a general psychopathology factor was initially identified by Lahey et al (2012) using a large representative sample of American adults. The same model identifying three correlated specific factors of externalising, fear and distress plus the general psychopathology factor was replicated by Greene & Eaton (2017).

Whereas, Caspi, Houts, Belsky, Goldman-Mellor, Harrington, Israel et al (2014) and Hyland, Murphy, Chevlin, Carey, Vallières, Murphy et al (2018) identified a slightly different model, specifying specific factors of internalising, externalising and thought disorder plus the general psychopathology factor. The findings are relatively similar within the child and adolescent research field, with the majority of studies confirming that a bifactor model of psychopathology fitted the data best. However, as with the adult data the specific factors included in the model vary. The models identified include: two specific factors of internalising and externalising (Castellanos-Ryan, Brière, O'Leary-Barrett, Banaschewski, Bokde, Bromberg et al., 2016; Hankin, Davis, Snyder, Young, Glynn & Sandman, 2017; Laceulle, Vollebergh, & Ormel, 2015; Lahey, Rathouz, Keenan, Stepp, Loeber, & Hipwell, 2015; Olino, Bufferd, Dougherty, Dyson, Carlson, & Klein, 2018; Snyder, Young, & Hankin, 2017, 2019; Tackett, Lahey, Van Hulle, Waldman, Krueger & Rathouz, 2013), and three specific factors of internalising, externalising and thoughts disorder (Afzali, Sunderland, Carragher, & Conrod, 2018; Carragher, Teesson, Sunderland, Newton, Krueger, Conrod et al., 2016) or fear, distress and externalising (Martel, Pan, Hoffmann, Gadelha, do Rosário, Mari et al., 2017) plus a general psychopathology factor. However, these studies suggest that the identified model is heavily influenced by the instruments used to measure psychopathology (Goodman et al., 2010; Patalay et al., 2015). Therefore, unless the measures included assess the full range of mental disorders it is not possible to know if the identified model capture the full breadth of psychopathology.

Longitudinal data indicates that the structure of psychopathology remains relatively stable over time, with substantial correlation between factors across development (Brodbeck, Stulz, Itten, Regli, Znoj, & Caspar, 2014; Castellanos-Ryan et al., 2016; McElroy, Belsky, Carragher, Fearon, & Patalay, 2018; Noordhof, Ormel, Oldehinkel, & Hartman, 2015; Olino et al., 2018; Snyder et al., 2017). These findings are indicative of homotypic continuity and suggest that individuals who experience

high levels of psychopathology relative to their peers early on are likely to continue to experience high levels relative to peers (Snyder et al., 2017). However, there is also evidence of heterotypic continuity, as the general psychopathology factor has been found to be predictive of adverse mental health outcomes over time (Patalay, Fonagy, Deighton, Belsky, Vostanis, & Wolpert, 2015; Pettersson, Lahey, Larsson, & Lichtenstein, 2018). This means that although individuals may have a relatively stable predisposition towards experiencing a certain centre of core symptoms, these core symptoms can cause secondary difficulties and the phenotypic expression of these issues may change over time depending on particular circumstance (Murray et al., 2016). Additionally, there has been shown to be age and gender related patterns; with externalising disorder having earlier onset and being more likely to affect males when compared to internalising disorders (Martel, 2013).

To date only one of the studies has not supported the bifactor model of psychopathology. This study, which used data from a child and adolescent clinical sample, found that a five-factor structure fitted the symptom level data better than either a two factor structure or a classic bifactor model (de la Cruz, Vidal-Ribas, Zahreddine, Mathiassen, Brøndbo, Simonoff et al., 2018). Despite the dearth of evidence in support of the general psychopathology factor there have been some criticisms as to its validity. One hypothesis is that the specific factors load on the general factor because the same symptoms are used to define multiple factors. However, when the overlapping symptoms were removed from analysis the bifactor model still fitted the data best (Lahey, Zald, Perkins, Villalta-Gil, Werts, Van Hulle et al., 2018), indicating that this is unlikely. The bifactor model of psychopathology has also been supported by external validity analysis, which showed that the specific factors were differently associated with multiple external factors; for example the externalising factor has been associated with antisocial behaviour (Lahey et al., 2012), social depravation (Patalay et al., 2015) and certain personality traits

(Carragher et al., 2016; Hankin et al., 2017; Tackett et al., 2013). This indicates that it is also unlikely that the general psychopathology factor is just a manifestation of systematic measurement biases.

Impact of routine clinical intervention

The benefit of confirming the presence of a general psychopathology factor is its potential to change the way mental disorders are understood which may in turn lead to them being more effectively identified and treated. Currently mental health treatment is largely based on symptoms exhibited (Patalay et al., 2015), whereas the notion of a general psychopathology indicates a need to consider an individual's propensity for psychopathology to develop and individualised profile and treatment plan (Carrager et al., 2016). For example, if a person scores high on the general psychopathology factor and moderate on a particular specific factor, it could be that they would more likely benefit from a transdiagnostic approach to treatment (Afzali et al., 2018), which is in keeping with the recommendations made from the adult findings (Hyland et al., 2018). However, these recommendations are in contrast to how current UK treatment guidelines, such as those developed by the National Institute of Clinical Excellence (NICE), are developed and consequent treatment recommendations made.

A review of the literature indicates that there is limited evidence to suggest that any type of intervention, whether transdiagnostic or targeted, has a meaningful impact on the general psychopathology factor or the models specific factors over time. To date only one study has looked at the potential impact of clinical intervention (Constantinou, Goodyer, Eisler, Butler, Kraam, Scott et al., 2019), which showed that a targeted antisocial behaviour intervention was associated with a decrease in the specific antisocial factor at follow-up. However, this study utilised a very specific population and assessed the impact of a very targeted intervention, therefor the

generalisability of these findings are limited. In light of the limited evidence, it is still unclear what the long-term impact of therapeutic intervention on the general psychopathology factor or the overall structural model of psychopathology is. Further, it is not known whether such clinical recommendation, as mentioned above, will have a meaningful impact on the expression of mental health over time.

Rational for further research

Overall, despite the slight variation in model structure, the research indicates overwhelming support for the bifactor model of psychopathology and therefore a general psychopathology factor within the data for children and adolescents (Afzali et al., 2018; Black, Panayiotou, & Humphrey, 2019; Bloemen et al., 2018; Brodbeck et al., 2014; Carragher et al., 2016; Castellanos-Ryan et al., 2016; Constantinou et al., 2019; Haltigan et al., 2018; Hankin et al., 2017; Laceulle et al., 2015; Lahey et al., 2015; Martel et al., 2017; McElroy et al., 2018; Murray et al., 2016; Noordhof et al., 2015; Olino et al., 2018; Patalay et al., 2015; Pettersson et al., 2018; Snyder et al., 2017, 2019; Tackett et al., 2013) and adults (Caspi et al., 2014; Greene & Eaton, 2017; Hyland et al., 2018; Lahey et al., 2012; Lahey et al., 2017). However, it is also important to consider some of the current study limitations and how they might best be addressed to increase the generalisability of the findings to a UK health care setting.

Of particular concern is the lack of studies which utilised clinical samples. Of the four studies that included a clinical sample, only one used longitudinal data (Constantinou et al., 2019), two utilised a multisite naturalistic clinical sample (de la Cruz et al., 2018; Hyland et al., 2018) and three supported the bifactor model of psychopathology (Constantinou et al., 2019; Haltigan et al., 2018; Hyland et al., 2018). The final study supported a five factor structure; one possible explanation is that specificity of symptoms for each disorder increased in a high-risk clinical

population, therefore the two factor model may be too simplified (de la Cruz et al., 2018). In light of these conflicting findings it is clear that further research using clinical data is required to clarify the best fitting model. However, this needs to be achieved in the context of also addressing the current limitations, which include the limited inclusion of longitudinal data, the lack of naturalistic data and the limited exploration of the impact of intervention on the bifactor model. Further, the models have mainly been tested on American samples, with only four of the 20 studies including data from a British population (Brodbeck et al., 2014; Castellanos-Ryan et al., 2016; de la Cruz et al., 2018; Patalay et al., 2015). It is therefore questionable as to the extent the findings from American populations are generalisable to the British population and therefore a UK health care setting.

The identification of the bifactor model of psychopathology is not just about better understanding mental disorders, it is also about recommending more effective interventions to hopefully reduce the expression of psychopathology. To know whether recommendations, such as making changes to the treatment guidelines, are of value; it is also important to understand how current routine intervention impact on the model. However, to date only one study has explored the impact of therapeutic intervention on the factor scores within the bifactor model over time (Constantinou et al., 2019) and the generalisability of these findings are limited due to the clinical sample utilised. This high-lights a gap in the research, meaning it is not possible to form a clear understanding of the impact of current routine intervention on psychopathology and so further research is required before it is possible to know whether there is value in amending treatment guidelines.

Study aims and hypotheses

Aims.

In light of these limitations this study will focus on expanding the evidence using clinical samples from within a British population. Given that 50% of mental disorders to emerge in adulthood are present already in some form by the age of 14 (Murphy, 2012), the study will use routine data collected within Child and Adolescent Mental Health Services (CAMHS) across multiple sites within the UK. Symptom level data, from instruments validated within a child and adolescent population, will be used in an attempt to reduce artefactual co-morbidity. The study will therefore build more generally on the longitudinal evidence base. As well as exploring how routinely administered mental health intervention may, or may not, impact the expression of psychopathology over time. Including assessing the impact of intervention over and above the impact of other factors such as pre-existing levels of psychopathology and certain demographic factors such as age and gender.

Hypothesis.

The primary hypothesis is that a bifactor model with three specific factors of fear, distress and externalising and a general psychopathology factor will fit the data best.

The secondary hypothesis is that being offered a routine clinical intervention will significantly predict post intervention factor scores over and above initial levels of psychopathology and demographic factors including age, gender and ethnicity.

Method

Participants

Dataset.

Data was extracted from the Child Outcomes Research Consortium (CORC) dataset. CORC is a membership organisation that collects and uses evidence to improve children and young people's mental health and wellbeing. It holds a substantial dataset of demographic and outcome information from approximately 133,000 episodes of care, from children who have been supported by CAMHS (www.corc.uk.net). The data will therefore include children and young people who accessed tier 2 and 3 CAMHS and voluntary organisations and completed routinely collected mental health and wellbeing outcome measures. Data was collected at assessment and throughout therapy; in order to maximise paired data, the 'first' and 'last' completed measure will be used. All participants consented for their data to be used for secondary data analysis.

Sample.

The original dataset was comprised of 133,113 episodes of care (mean age: 12.10 (SD: 3.87) years, female: 52.6%, White or White British: 60.9%, mean first assessment Strengths and Difficulties Questionnaire (SDQ) score: 18.90 (SD: 6.23), mean first assessment Revised Child Anxiety and Depression Scale (RCADS) score: 58.61 (SD: 28.06)). Only those for which the participant had completed the self-reported version of the selected psychopathology outcome measure were initially extracted. For the SDQ the self-report measure was validated for those age 11 up to 18 and for the RCADS the age range was age eight to 18. Therefore, only episodes of care for participants age 11 to 18 were included in the sample (N=90905).

Initial analysis, was then conducted on data for which participants had completed both an SDQ and RCADS at first assessment (sample 1: N=30137, mean

age: 14.27 (SD:1.73) years, female: 66.1%, White or White British: 64.5%, mean first assessment SDQ score: 19.01 (SD: 6.12), mean first assessment RCADS score: 60.58 (SD: 28.12)). With all further analysis only including episodes of care where participant had paired first and last assessment data for both the SDQ and RCADS (sample 2: N=2947, mean age: 14.43 (SD: 1.69) years, female: 71.6%, White or White British: 71.0%, mean first assessment SDQ score: 18.62 (SD: 5.95), mean first assessment RCADS score: 62.99 (SD: 26.84)). Examination of the basic demographics and psychopathology data indicates that both of the samples included in the analysis were older, had a higher proportion of females and White or White British participants, and had greater mental distress than those from the original dataset.

Ethics

As anonymised secondary data is being used ethical approval was not required. However, individuals included within the dataset provided consent for their data to be used for the purpose of secondary analysis. Permission has been sort and granted by the CORC board for use of the CORC dataset for the purpose of this research project. Due to the confidential nature of the data, analysis was only be conducted on a secure network and results will not be shared or made publicly available without the consent of the CORC board.

Measures

Demographics.

Demographic data included age in years at the pre intervention assessment, gender and ethnicity. Ethnic categories included where those from the 2001 census: White or White British (British, Irish, Any other White background); Mixed (White and Black Caribbean, White and Black African, White and Asian, Any other mixed

background); Asian or Asian British (Indian, Pakistani, Bangladeshi, Any other Asian background); Black or Black British (Caribbean, African, Any other Black background); Other ethnic groups (Chinese, Any other ethnic group); and not stated.

Psychopathology.

The CORC dataset included a battery of routinely administered measures, those that were used to assess child and adolescent psychopathology included the SDQ (Goodman, Meltzer, & Bailey, 1998) and the RCADS (Chorpita, Moffitt, & Gray, 2005). Item level data which represented either internalising or externalising dimensions were included in the analysis. Multiple measures were selected to increase validity, as this way the factor analysis does not just represent the structure of the measure rather the construct of psychopathology the selected items represent (Patalay et al., 2015). In order to assess change in child and adolescent psychopathology over time, data was collected at the first and last appointment.

Strength and Difficulties Questionnaire.

The SDQ is a 25 item questionnaire asking about the child's positive and negative attributes. It is a widely used self-report measure of child mental health which has been shown to have good construct validity and reliability (Goodman et al., 1998). The internal consistency has been shown to be generally satisfactory (mean Cronbach α 0.73) and the interrater correlations were highly significant (Goodman, 2001).

The items are divided between 5 subsections: conduct problems, hyperactivity, emotional support, peer problems and pro-social behaviour. It is scored on a 3-point scale from 0 (not true), 1 (somewhat true) to 2 (certainly true), 5 items are reverse scored. A score from 0-10 is generated for each of the subscales and the scores from the hyperactivity, emotional support, peer problems and conduct

problems can be summed to generate a total difficulties score (maximum score=40), where a higher score indicated greater difficulties. The items included in the analysis were informed by a recent structural analysis undertaken on the measure (Goodman, Lamping, & Ploubidis, 2010). Therefore, the reverse scored items were removed and internalising dimension was represented only by the emotional and peer scale items and externalising dimension represented by the conduct disorder and hyperactivity scale items.

Revised Child Anxiety and Depression Scale.

The RCADS is a 47 item self-report measure that assesses the main features of five anxiety disorders: separation anxiety, social phobia, generalised anxiety, obsessive-compulsive disorder and panic, as well as major depressive disorder (Chorpita et al., 2005). The scale has been shown to have good reliability when administered on a clinical sample, with internal consistency between 0.78 and 0.88 (Chorpita et al., 2005). Further, retest coefficients at one week were good, ranging from 0.63 to 0.66 (Chorpita, Yim, Moffitt, Umemoto & Francis, 2000).

The RCADS is scored on a 4-point scale from 0 (never), 1 (sometime), 2 (often) to 3 (always), where a high score indicates greater symptom severity. The sum of all scale scores can be calculated to generate a total difficulties score (maximum score=141), where high score indicate greater difficulties. Given that the identified model is heavily influenced by the symptom items used to measure psychopathology (Goodman et al., 2010; Patalay et al., 2015), only the 25 items assessing panic disorder, generalised anxiety and major depression were included in the analysis. The other three subsets were removed in a hope that the final model was not overly influenced by the RCADS items, of which there were already a greater number.

Intervention.

Intervention type was also included in the longitudinal analysis to assess the impact routinely administered intervention had on levels of child and adolescent psychopathology over time. However, due to insufficient sample sizes it was not possible to include individual types of intervention in the analysis, therefore intervention type was categorised by those offered any intervention and those offered no intervention. Of the 1416 participant who had any intervention, 62.3% (n=882) had one type of intervention, 26.0% (n=369) had two types of interventions and 11.7% (n=165) had three or more types of interventions. Further, each participant received an average of 6.0 (SD: 10.3) face-to-face sessions. However, due to data quality it was not possible to include this data in the regression analysis.

Data analysis

Data was recorded in SPSS and analysed using SPSS and Mplus v8.

Statistical Power.

With statistical power set at 80% and risk of making a type 1 error at 20%, given there are 4 latent variable in the model and 40 observed variables, a sample size of approximately 800 would be necessary to detect an effect size of 0.1.

Basic descriptive.

Basic descriptive included mean age at first assessment and mean level of psychopathology at first and last assessment. T-tests were used to test whether there was a significant change in the mean level of psychopathology from first to last assessment. Percentages were used to calculate the gender and ethnic breakdown of the population, as well as the proportion offered any type of intervention.

Model fit.

In order to maximise sample size, initial exploration of best model fit was undertaken on all episodes of care where participant had completed SDQ and RCADS first assessment data (sample 1: N=30137). Firstly, to identify how the included psychopathology items best loaded, an exploratory factor analysis (EFA) was conducted on a random 50% (n=15236) of sample 1. Due to the assumption of shared variance, any item included in the EFA with a final loading of less than 0.3 was removed from further analysis (Field, 2013). Therefore, the EFA was rerun until all items had a loading of more than 0.3. A confirmatory factor analysis (CFA) was then conducted, using the remaining items, on the other 50% (n=14901) of sample 1 to confirm the loading fitted the data well.

Finally, all episodes of care included in sample 1 were included in further structural analysis in order to identify the best fitting structural model. The structural models examined included: a univariate model, where all items load on one factor; a basic four factor model; a second order model, where items were initially load on a one factor then the four factors; and a orthogonal and non-orthogonal four factor bifactor model. For such models a good fit is usually considered when the root mean-square error of approximation (RMSEA) is less than 0.06, Comparative Fit Index (CFI) and Tucker Lewis Index (TLI) is greater than 0.95 (Hu & Bentler, 1999).

Longitudinal analysis.

To explore the stability of the model over time and in the context of routinely administered interventions, further analysis was conducted on only those with paired first and last assessment data (sample 2: N=2947). Firstly, structural model analysis was conducted to confirm the acceptability of the model fit. The factor scores of the best fitting model were then extracted and t-tests and an ANOVA was used to assess difference in mean factor scores over time (Hinton, 2014). Hierarchical multiple

regression analysis was then undertaken to explore whether being offered any intervention significantly predicts last assessment factors scores over and above other independent variables (Gelman & Hill, 2006).

Variables were initially input into a simple regression analysis to identify which, if any, individually significantly predict the factor scores at last assessment. Variable included in the analysis were: the factor scores at first assessment, age, gender (male vs not male), ethnicity (White British vs not White British) and intervention (any type of intervention vs no intervention). Only those variables which individually predicted the factor scores at last assessment were then included in the hierarchical regression. Given that evidence suggests that first assessment factors scores (Snyder et al., 2017) and certain demographic factors (Afzali et al., 2018; Patalay et al., 2015) significantly predict future psychopathology, these factors would be entered in into the hierarchical model first. A second model will then be run to identify the impact of routinely administered intervention on future psychopathology over and above that already explained by the first assessment factor scores and the demographic variables.

Results

Model fit

Sample 1: Demographics and psychopathology.

Of the 30137 participants who had competed data for both the RCADS and SDQ at first assessment (sample 1) 66.1% (n=19924) were female, 64.5% (n=19451) were of a White or White British ethnicity and they had a mean age of 14.27 years (SD: 1.73, range: 11-18). The mean total score on the RCADS (60.58, SD: 28.12) indicates that the population was within the clinical range for anxiety (T=74, cut-off=70) and the borderline range for anxiety and depression (T=66, cut-off=65). The mean SDQ score (19.01, SD: 6.12) puts the sample in the ‘high’ category.

Table 1: Demographic and psychopathology data for sample 1 (N=30137), participants with first assessment outcome data

	(N=30137)	
	n	**%**
Gender		
Female	19924	66.1
Male	10186	33.8
Other	9	0.0
Missing	18	0.1
Ethnicity		
White or White British	19451	64.5
Mixed	1016	3.4
Asian or Asian British	706	2.3
Black or Black British	755	2.5
Other Ethnic Group	448	1.5
Not stated/known	4852	16.1
Missing	2909	9.7
	Mean	**SD**
Age	14.27	1.74
RCADS		
Social Phobia	14.71	7.11
Panic Disorder	9.67	6.72
Separation Anxiety	6.32	4.54
Generalised Anxiety	8.82	4.53
Obsessive-compulsive	6.42	4.21
Major Depression	14.52	6.94
Total	60.58	28.12
SDQ		
Prosocial	7.05	2.09
Hyperactive	5.65	2.39
Emotional	6.29	2.55
Conduct Disorder	3.37	2.10
Peer	3.71	2.21
Total	19.01	6.12

Exploratory factor analysis (EFA).

An initial EFA was conducted on a random 50% (n=15236) of the data from sample 1. The EFA included the 40 items selected from the SDQ and RCADS, as specified in the method section above. None of the models (1-4 factors) met the criteria for good or even acceptable fit (RMSEA>0.06, CFI & TLI>0.90, see Table 2), however, the best fitting model was the four factor model (RMSEA=0.06, CFI=0.90, TLI=0.93). Examination of the item loadings indicated that the SDQ item, 'I get on better with adults than people my own age' (factor loading = 0.19), had a loading of

<0.3, which is considered to be unacceptable (Field, 2013). Therefore, the analysis was re-run excluding that and another two other SDQ items: 'Other children or young people pick on me or bully me' (factor loading = 0.29) and the SDQ item 'I am nervous in new situations' (factor loading = 0.29). The final best sitting model was a four factor model which included 37 psychopathology items (RMSEA=0.06, CFI=0.95, TLI=0.93).

Table 2: Exploratory factor analysis of a random 50% (n=15236) of the data from sample 1

		RMSEA (90% CI)	CFI	TLI
1 factor model	**40 items**	0.11 (0.11-0.11)	0.79	0.78
2 factor model	**40 items**	0.09 (0.09-0.09)	0.86	0.84
3 factor model	**40 item**	0.08 (0.08-0.08)	0.90	0.89
4 factor model	**40 items**	0.06(0.06-0.06)	0.94	0.93
	37 items	0.06(0.06-0.06)	0.95	0.93

Confirmatory factor analysis (CFA).

A CFA was conducted using the 37 item, four factor model identified from the EFA on the other 50% (n=14901) of the data from sample 1. Due to the loading of RCADS question 47 (I feel restless…) it was not possible to run the analysis and so it was re-run excluding that item from the model.

The best fitting model therefore included 36 items and was comprised of four factors (RMSEA=0.07, CFI=0.94, TLI=0.93). The first factor, labelled 'fear – cognitions', included 7 items examining participants' experience of fear related cognitions. The second factor, labelled 'fear – bodily sensations', included 10 items examining participants' experience of fear related bodily sensations. The third factor, labelled 'distress', included 12 factors exploring the level of distress participants experience. The fourth and final factor, labelled 'externalising', included 7 items

exploring the level of externalising symptoms participants experienced (see Appendix 2, Table 1).

Structural model.

All data from sample 1 was then utilised for further analysis and included the 36 items which made up the final CFA. A modified (non-orthogonal) bifactor model comprising of four factors (as identified in the CFA) and a general psychopathology factor was found to fit the data best (RMSEA=0.06, CFI=0.95, TLI=0.94, see Table 3).

Table 3: Structural models explored utilising all sample 1 (N=30137) data

	RMSEA (90% CI)	CFI	TLI
Univariate	0.12 (0.12-0.12)	0.80	0.78
4 factor model	0.07 (0.07-0.07)	0.94	0.93
Second order	0.07 (0.07-0.07)	0.94	0.93
Bifactor (orthogonal)	0.06 (0.06-0.06)	0.95	0.94
Bifactor (modified)	0.06 (0.06-0.06)	0.95	0.94

Longitudinal analysis

Sample 2: Demographics and psychopathology.

Of the 2945 participants who had paired RCADS and SDQ first and last assessment data (sample 2), 71.6% (2110) were female, 71.0% (2091) were White or White British and their mean age was 14.43 years (SD: 1.69, see Table 4). At the first assessment, the mean total score on the RCADS (62.99, SD: 26.84) indicates that the population is within the clinical range for anxiety (T=75, cut-off=70) and the borderline range for anxiety and depression (T=67, cut-off=65). Whereas at last assessment the mean total score on the RCADS (44.38, SD: 27.16) indicates the population is within the normal range for both anxiety (T=61) and anxiety and

depression (T=54). There was a significant difference between the mean first and last assessment total RCADS score (t(2399)=35.22, P<0.001). The mean pre SDQ score (18.62, SD: 5.95) puts the sample in the 'high' category and the mean post SDQ score (15.37, SD: 6.55) puts the sample in the 'slightly raised' category. There was also a significant difference between the mean first and last assessment total SDQ scores (t(2722)=29.51, p<0.001).

Table 4: Demographic and psychopathology data for sample 2 (N=2947), those with paired first and last assessment outcome data

	(N=2947)			
	n		**%**	
Gender				
Female	2110		71.6	
Male	834		28.3	
Other	1		0.0	
Missing	2		0.1	
Ethnicity				
White	2091		71.0	
Mixed	110		3.7	
Asian	72		2.4	
Black	83		2.8	
Other	43		1.5	
Not stated/known	317		10.8	
Missing	231		7.8	
Any intervention	1416		48.0	
	Mean		**SD**	
Age	14.43		1.69	
	First assessment		**Last assessment**	
	Mean	**SD**	**Mean**	**SD**
RCADS				
Social Phobia	15.38	6.82	11.82	6.84
Panic Disorder	10.18	6.61	6.89	5.96
Separation Anxiety	6.62	4.43	4.53	4.10
Generalised Anxiety	9.32	4.32	6.53	4.24
Obsessive-compulsive	6.72	4.16	4.42	3.89
Major Depression	14.81	6.81	10.62	7.02
Total	62.99	26.84	44.38	27.16
SDQ				
Prosocial	7.25	2.03	7.53	2.07
Hyperactive	5.54	2.34	4.77	2.42
Emotional	6.57	2.41	4.96	2.73
Conduct Disorder	2.98	2.01	2.48	1.92
Peer	3.54	2.19	3.15	2.09
Total	18.62	5.95	15.37	6.55

Type of intervention.

Of the 2947 participants 48% (n=1416) were offered any type of intervention (see Table 5); with the most frequently offered being Cognitive Behavioural Therapy (26.9%, n=792), other therapy (16.7%, n=491) and multi-modal therapy (6.8%, n=201).

Structural model.

The best fitting structural model for the sample 2 data for both the first (RMSEA=0.06, CFI=0.95, TFI=0.94) and last (RMSEA=0.06, CFI=0.96, TFI=0.96) assessment was also a four factor (as identified in the initial CFA) modified bifactor model (see Table 5).

Table 5: Structural models explored utilising sample 2 (N=2947) data

	First assessment			**Last assessment**		
	RMSEA (90% CI)	**CFI**	**TLI**	**RMSEA (90% CI)**	**CFI**	**TLI**
Univariate	0.12 (0.12-0.12)	0.78	0.76	0.11 (0.11-0.11)	0.86	0.85
4 factor model	0.07 (0.07-0.07)	0.93	0.92	0.07 (0.06-0.07)	0.95	0.95
Second order	0.07 (0.07-0.07)	0.93	0.92	0.07 (0.07-0.07)	0.95	0.95
Bifactor (orthogonal)	0.07 (0.07-0.07)	0.93	0.92	0.06 (0.06-0.07)	0.96	0.95
Bifactor (modified)	0.06 (0.06-0.06)	0.95	0.94	0.06 (0.06-0.06)	0.96	0.96

Factor scores.

Change in factor scores.

At first assessment the mean factors scores indicated that the sample was weakly positively associated with the general psychopathology factor and externalising factor; whereas it was weakly negatively associated with the fear and distress factors. At last assessment the association remained the same for all factors except the externalising factor where the association became weakly negative. There

was no significant change in any of the factor scores from first assessment to last assessment (See Appendix 3, table 2). When entered into an ANOVA, the mean factor score for those offered and those not offered an intervention was only significantly different for the last assessment general psychopathology factor scores (no intervention mean=-0.01, any intervention mean=0.05; $F(1,2945)=4.47, p<0.05$).

Regression analysis.

All variables (the first assessment factor scores, age, gender (male vs not male), ethnicity (White British vs not White British) and intervention (any type of intervention vs no intervention)) were individually entered into a regression model to assess which significantly predicted each of the last assessment factor scores (see Appendix 4, Table 3). Factor scores at first assessment individually significantly predicted each of the factor scores at last assessment. The higher the general psychopathology factors score at first assessment the lower the fear – cognition, fear – bodily sensation, distress and externalising factors score at last assessment, but higher the general psychopathology factor score at last assessment, and vice versa.

Neither gender nor ethnicity predicted any of the factors scores at last assessment. Age significantly predicted all factor scores at last assessment bar the general psychopathology factor score. Older children scoring higher on the fear – cognition ($F(1,2945)=45.58$, $p<0.001$, $R^2=0.02$), fear – bodily sensation $F(1,2945)=9.38$, $p<0.001$, $R^2=0.003$) and distress factors ($F(1,2945)=24.87$, $p<0.001$, $R^2=0.01$) at last assessment. Whereas younger children scored higher on the externalising factor ($F(1,2945)=10.87$, $p<0.001$, $R^2=0.004$) at last assessment. Being offered any type of intervention only predicted the general psychopathology factor score at last assessment, with those having been offered therapy having a lower general psychopathology score ($F(1,2945)=4.48$, $p<0.05$,$R^2=0.002$) at last assessment.

Hierarchical multiple regression.

Only the variance in the general psychopathology factor score at last assessment was assessed utilising the hierarchical multiple regression analysis, as this was the only dependant variable being offered an intervention significantly predicted. The first model included those independent variable that had individually predicted general psychopathology factor score at last assessment (fear – cognitions factor, fear – bodily sensations factor, distress factor, externalising factor), not including intervention (see table 6). This model showed that the first assessment factor scores significantly predicted the general psychopathology factor score at last assessment and accounted for 19.3% of the variance in the scores across the sample.

Table 6: Multiple regression model exploring the impact of first assessment factor scores and intervention on the last assessment general psychopathology factor score

	Model 1	**Model 2**
Constant	0.03 (0.01)	0.06 (0.02)
First assessment factor scores		
General psychopathology factor	0.54 (0.03)	0.54 (0.03)
Fear – cognition factor	0.10 (0.03)	0.10 (0.03)
Fear – bodily sensation factor	-0.02 (0.04)	-0.02 (0.04)
Distress factor	0.06 (0.05)	0.06 (0.05)
Externalising factor	0.07 (0.03)	0.07 (0.03)
Any intervention		-0.07 (0.03)
F	141.49*	119.14*
R^2	0.19	0.19
Change R^2		0.002**

*significant at the <0.001 level; **significant at the <0.05 level

The second model included the independent variables in model one in addition to intervention to explore the independent effect of intervention on the last assessment general psychopathology factor score. This model showed that being offered any intervention significantly predicted the general psychopathology factor score at last assessment and independently accounted for 0.2% of the variance over and above that explained by the other independent variables (see Table 6).

Discussion

Principal findings

The primary aim of the study was to expand on the evidence for a general psychopathology factor in a British child and adolescent clinical sample. The main outcome was that a modified bifactor model with four specific factors and a general psychopathology factor fitted the data marginally better at both the first (RMSEA=0.06, CFI=0.95, TLI=0.94) and last (RMSEA=0.06, CFI=0.97, TFI=0.96) assessment point than the other model examined. The first of the four factors was labelled ‘fear – cognitions’ and included items such as ‘I worry a lot’ and ‘I worry something bad will happen to me’. The second factor was labelled ‘fear – bodily sensation’ and included items such as ‘when I have a problem, my heart beats really fast’ and ‘I suddenly feel as if I can’t breathe when there is no reason for this’. The third factor was labelled ‘distress’ and included items such as ‘I feel sad or empty’ and ‘I am tired a lot’. The fourth and final factor was labelled ‘externalising’ and included items such as ‘I am restless, I can’t stay still for long’ and ‘I fight a lot. I can make other do what I want’. These findings indicate that the primary hypothesis must be rejected as the model specified four specific factors rather than the three that were initially hypothesised.

The secondary aim of the study was to explore whether routinely administered therapeutic intervention impacted the expression of psychopathology over time, over and above the effect of demographic factors such as age, gender and ethnicity. Firstly, change in factor scores from first to last assessment was examined, which showed that none of the factor scores significantly changed over time, indicating homotypic continuity. When participant characteristics and first assessment factor scores were entered into a regression analysis, the biggest individual predictor of factor score at last assessment was the score on that factor at first assessment.

Neither gender nor ethnicity predicted any of the factor scores at last assessment. Older participant had higher last assessment fear and distress factor scores and lower externalising scores, but age accounted for very little variance in the factor score. Being offered an intervention only predicted general psychopathology factor score at last assessment, with those being offered an intervention having lower general psychopathology scores, but again accounted for very little of the variance in the factor scores. When all significant individual predictors of last assessment general psychopathology factor score were entered into a multiple regression, being offered an intervention was found to be a small, but significant predictor over and above the variance accounted for by the initial assessment factor scores. This indicates that the secondary hypothesis must also be rejected as therapy only significantly predicted the general psychopathology factor, not all factors as initially hypothesised.

Strengths and limitations

This is only the fourth study to examine, using bifactor modelling, the notion of a general psychopathology factor within clinical data from a child and adolescent sample. Further it is the only the second study to have explored the potential impact of clinical intervention on the factor scores over time, and therefore the potential impact of clinical intervention on the expression of different aspects of mental health over time. Particularly as this is the first study to do this in the context of a multisite naturalistic clinical cohort.

The study utilises a large pre-existing, routinely collected, national data set which has enabled a sufficient sample size to adequately power the complex structural modelling to explore the best fitting structure of psychopathology and explore the longitudinal impact of clinical intervention. The size and the large number of national sites contributing to the data collection also maximised the possibility of generalisability of the finding to the UK population who utilise secondary and tertiary

CAMHS. However, even though the original dataset included 133,113 episodes of care, only samples of 30,137 (22.6%) and 2,947 (2.2%) were included in the final analysis. Further, these subsamples were found to be older, to have a higher proportion of females and White or White British participants, and had greater mental distress than those from the original dataset. Indicating that these samples are not representative of the original dataset, limiting the extent to which these findings can be generalised to CAMHS settings.

Using pre-existing data also comes with a number of other limitations. Firstly, it is difficult to rigorously monitor data quality, in terms of the completeness of questionnaires and how consistently clinicians were ensuring data was collected, particularly at follow-up, which greatly impacted the number of participants with paired first and last assessment data. The reasons for poorer response rates at follow-up could lead to selection bias, which certain participants being more likely to attend last appointments, fill out and return the questionnaire and consent to secondary data analysis, which impacts on the generalisability of the findings. Further, it is not possible to know whether all sites are interpreting and categorising data in exactly the same way. A particular example of this the recording of intervention type, as due to the potentially integrative nature of therapy (Gilbert & Orlans, 2010), interventions may not fall neatly into one the prespecified categories. Meaning that intervention type may have been influenced by the opinions of the individual therapists. Additionally, it is not possible to be sure what 'no intervention' refers to as it is unclear why an individual would be under the care of a mental health team and be offered no intervention. However, all sites were provided with information on how to correctly administer and score the questionnaires and it is hopeful that is may have limited across site variations.

Most significantly the analysis is constrained by the specific questionnaires used and the data quality within the pre-existing dataset. Not only does this make

comparisons with other studies more difficult as none have used the exact combination of questionnaire items and none cover the full breadth of psychopathology. Meaning that, comparisons need to be made within the context of acknowledging different and potentially limited psychopathology data was being collected within vastly different service settings. It was also not possible to include potentially important variables, such as number of intervention sessions or type of intervention, in the regression analysis. If included, these variable may have changed the relationship that 'any intervention' had on the last general psychopathology factors score. Therefore, interpretation of the results needs to acknowledge that there may be other variables that account for the variation in psychopathology score that have not been able to be considered in this study.

The study also only included self-report measures of psychopathology, which due to social desirability bias, participants may have been more likely to under report symptoms to avoid stigma of meatal health and may report more improvement at last assessment to 'please' services. Such biases may account for the sample only being within the mild to moderate range despite most secondary and tertiary services tending to support those with higher need. The findings may therefore have limited generalisability to the more severe end of the mental health spectrum.

Comparison with the literature

Structural model of child and adolescent psychopathology.

A modified bifactor model with four specific factors (fear – cognition, fear – bodily sensation, distress and externalising) and a general psychopathology factor fitted the data better than the other models examined. This finding adds to the growing evidence in support of a general psychopathology factor within child and adolescent psychopathology (Afzali et al., 2018; Bloemen et al., 2018; Brodbeck et al., 2014; Castellanos-Ryan et al., 2016; Carragher et al., 2016; Haltigan et al., 2018; Hankin et

al., 2017; Lahey et al., 2015; Laceulle et al., 2015; Martel et al., 2017; McElroy et al., 2018; Murray et al., 2016; Noordhof et al., 2015; Olino et al., 2018; Patalay et al., 2015; Pettersson et al., 2018; Snyder et al., 2017, 2019; Tackett et al., 2013). However, given that the fit of the bi-factor model was only marginally better than the 4-factor or second order model, the strength of this finding needs to be considered in the context of the study limitations. There was also a slight variation in the typically identified four factor structure, in that the fear factor was separated in to fear – cognitions and fear – bodily sensations and no thought disorder factor was identified.

One potential explanation for factor structure identified is that this study used symptom level rather than diagnostic level data. The symptom level data may provide greater detail of how psychopathology is expressed, which could in turn account for the more detailed four factor model identified. When examining previous studies those using simplified diagnostic data were more likely to identify the simplified two factor (internalising and externalising) model (Castellanos-Ryan et al., 2016; Hankin et al., 2017; Laceulle et al., 2015; Lahey et al., 2015; Olino et al., 2018; Snyder et al., 2017a, 2017b; Tackett et al., 2013). Whereas the more symptom level data that is included in the model the more factors that are identified. For example, Afzali et al (2018) included mood, psychotic, externalising symptom and identified a 3 factor model and Bloemen et al (2018) included mood, externalising and attentional symptom and identified a four factor model. Such findings bring into question the validity of studying the structure of psychopathology using only diagnostic level data or limited symptom level data which does not cover the full spectrum of psychopathology symptomology.

A further explanation may be the specific questionnaire used and which items were selected to be included. This study was the first to include items from the RCADS in conjunction with the SDQ. Previous studies including those conducted by Pataley et al (2015) and Afzali et al (2018) used questionnaires such as Me and My school (Deighton, Tymms, Vostanis, Belsky, Fonagy, Brown et al., 2012) and the Brief

Symptom Inventory (Derogatis & Melisaratos, 1983) respectively. Me and My School is a 16 item scale, of which only 10 capture emotional difficulties and although the BSI is a 53 item scale only 12 items capturing emotional difficulties were included in the study. When compared to this study, which includes 24 items from the RCADS, an emotional difficulties scale (Chorpita et al., 2005), it is unsurprising that the final model identified was formed of predominately emotional related factors. Further the study included a higher proportion of fear related items, which may also explain the identification of two distinct fear factors. It was not possible to include any items examining thought disorder symptoms as these were not routinely collected as part of the national data set. It is therefore unsurprising that no thought disorder factor was elicited within the bifactor model, as shown in previous studies that did not include psychosis symptomology (Martel et al., 2017). The limited psychopathology data, such as the lack of psychosis and lifespan condition items (i.e. ASD), included within the model further highlights whether such models give an incomplete picture of the structure and relationships of child and adolescent psychopathology.

Lastly it is possible that the use of clinical rather than community sample may in part account for the model variation. As there are only three other studies using a clinical sample (Constantinou et al., 2019; de la Cruz et al., 2018; Haltigan et al., 2018), it is hard to know if this is the case. Particularly as different questionnaires and symptomology were studied. Haltigan et al (2018) included mood, behavioural, psychosis and attentional symptom level data and identified that a four-factor bifactor model including internalising, externalising, thought disorder and attention plus the general psychopathology factor fitted the data best. Constantinou et al (2019) included item data from the SDQ and the Mood and Feeling questionnaire (Wood, Kroll, Moore & Harrington, 1995), and identified a bifactor model with four specific factors (anxiety, mood, antisocial and attentional). However, as previously mentioned they included participants with very specified mental disorders, which may impact on

the generalisability of these finding. Whereas de la Cruz et al (2018) only used one questionnaire capturing mood and behavioural symptoms found that a five-factor structure fitted the data best. However, as only one questionnaire was included it is possible that the factor analysis just represents the structure of the measure rather the construct of psychopathology the selected items represent (Patalay et al., 2015). Further, the selection of items was not informed by a recent structural analysis undertaken on the instrument (Goodman et al., 2010). However, what is noticeable from these two studies is that the model structure identified appears to be mainly influenced by the symptom items included. This could also be argued in the case of this study, where the identified model appears to have been strongly influenced by structure of the RCADS, from which most items were selected. For example, the fear – cognition factor is comprised of only items from the generalised anxiety subscale of the RCADS; the fear – bodily sensation factor from the panic disorder subscale and the distress factor from the major depression subscale. Not only does this highlight the need to include more than one scale in the structural analysis, but also brings forward the question as to whether the analysis should be too heavily loaded from one particular scale, as the final model may therefore be too heavily influenced by the construct being measured by that questionnaire.

Last assessment factor scores.

There was no change in factor scores from first to last assessment indicating that the model was stable over time, which is in keeping with previous studies that demonstrated homotypic continuity in the bifactor models (Brodbeck et al., 2014; Castellanos-Ryan et al., 2016; Noordhof et al., 2015; Snyder et al., 2017b). In contrast there was a significant change in psychopathology as measured by the RCADS and SDQ. A potential explanation for this discrepancy is that, although the factors within the bifactor model are heavily influenced by the RCADS subscales, they are

measuring distinctly different constructs. As within a bifactor model the specific factors represent the relationship the included items have to each other over and above their relationship captured by the general factor (Murray et al., 2016). It is therefore possible that the change in psychopathology as measured by the questionnaires is captured within the general factor rather than the specific factors. As although the overall the general factor did not change over time, there was a significant difference in the last assessment general psychopathology factor score, but not the specific factors, for those offered and thoes not offered an intevention.

The strongest predictor of post assessment factor scores was the factor score at initial assessment, which is consistent with prior studies which demonstrated homotypic continuity. Specifically, one study found that each factor at age 13 predicted the same factor 18-months late at age 15 (Snyder et al., 2017). With another showing substantial correlation between factors at age 14 and 16 (Castellanos-Ryan et al., 2016). Age predicted the last assessment factor score for the specific factors but not the general psychopathology factor. More specifically the study found that older participants experience less externalising and more fear and distress (internalising) symptoms, which is supported by previous literature (Martel, 2013). Which adds weight to the idea that disorder specificity increases with age, due to a gradual increase in expression of particular psychopathology over time (Patalay et al., 2015). Further, the literature has consistently demonstrated that the general psychopathology factor remains stable from across late childhood and early adolescence (Castellanos-Ryan et al., 2016; Murray et al., 2016).

Neither gender nor ethnicity were found to significantly predict any of the last assessment factor scores. The lack of association between gender and the general psychopathology factor is consistent with prior studies, indicating that gender is not a major component of the aetiology and development of the general liability to psychopathology (Afzali et al., 2018). However, in terms of the specific factors this

finding was unexpected given that previous studies have consistently shown that male gender is associated with higher levels of externalising disorders and female gender with internalising disorders (Afzali et al., 2018; Carragher et al., 2016, Patalay et al., 2015). The fact that the current study utilises a clinical sample may in part account for this unexpected finding. As although de la Cruz et al (2018), one of the few studies to also utilise a clinical sample, did not support a bifactor model they also found that there was no fluctuation in their model across genders. Suggesting that at a certain level of severity the gender difference in internalising and externalising disorders may become less prevalent. None of the previous studies have explored the association of ethnicity with the general psychopathology or specific factors therefore it is not possible to be sure of how this finding compares to others. However, it has been consistently shown that those of black and ethnic minorities are more likely to be diagnosed with psychosis (Kirkbride, Barker, Cowden, Stamps, Yang, Jones et al., 2008) and previous studies have shown that Black males reported engaging in highest levels of aggression (McLaughlin, Hilt & Holen-Hoeksema, 2007). Given these findings it may have been expected that there would be an association between ethnicity and the factors scores, however the simplified category of White and not white could have expunged this association.

Being offered an intervention predicted the last assessment factor score for the general psychopathology factor, but none of the specific factors. More specifically, those who were offered an intervention had lower general psychopathology factors scores at the last assessment. Further being offered an intervention predicted a small, but significant, amount of variance in the general psychopathology factor score at last assessment over and above that predicted by the first assessment factor scores. One potential explanation for this finding is that just comparing intervention versus not intervention was too simplified to capture any change within the specific factors. For example, if there had been sufficient power to include parenting intervention, which

are offered to target the expression of externalising disorders such as ADHD (Heath, Curtis, Fan & McPherson, 2015), in the regression analysis, that specific intervention may have predicted the externalising factor score. This is in line with the findings from Constantinou et al (2019), who showed that a targeted antisocial behaviour intervention was associated with a decrease in the specific antisocial factor at follow-up. However, it could also be that, as mentioned previously, the routinely administered intervention only targets an individual's general vulnerability to psychopathology. Specifically, targeting the disordered form and content of thought that has been hypothesised to spread throughout practically every disorder (Caspi & Motit, 2018). Rather than the specific symptomology that forms the specific disorders captured by the specific factors. When interpreting this data it is also important to note that other, potentially significant variable were not able to be considered in the model, so it is not possible to ascertain the true extent to which intervention predicted the final general psychopathology factor score.

Clinical implications and future direction

To date most studies have continued to support the notion of a general psychopathology factor, indicating that comorbidity within mental disorders is inevitable. Such support highlights the drawback of using diagnostic driven service structure and treatment recommendation which is common within the NHS. Services may therefore be better arranged around a risk factors that are relevant to multiple diagnostic categories (Castellanos-Ryan et al., 2016). Further, the notion of a general psychopathology factor that is common to most or all items may indicate the need to consider the use of transdiagnostic interventions to target overall psychopathology. Particularly given that in the current study intervention only impacted the general psychopathology factor, meaning that to be most effective intervention should focus on targeting the core symptoms that are thought to cause secondary difficulties as

expressed by the specific factors (Murray et al., 2016). However, current NHS treatment guidelines are diagnosis specific and tend to support the implementation of diagnostically driven interventions, such as CBT for social anxiety (NICE, 2013). This is despite promising evidence for the use of transdiagnostic approaches (Farchione, Fairholme, Ellard, Boisseau, Thompson-Hollands, Carl et al., 2012).

The homotypic continuity of the bifactor model indicates that those who experience high levels of psychopathology early on are likely to continue to experience high levels of psychopathology (Snyder et al., 2017). Indicating the potential benefit of early screening to identify those at risk, particularly as disorder specificity has been found to increase with age. For example, many individuals may exhibit a broad spectrum of mild problems, with a few experiencing more and more extreme expressions of psychopathology over their development (Caspi & Moffitt, 2018). However, early screening, watchful waiting, followed by early intervention could potentially halt such developmental progression. In addition, the finding that factor scores at first assessment account for a significant proportion of the variance in factor scores at last assessment, may indicate that initial tests of psychopathology could aid clinicians in determining those at greater risk over time (Afzali et al., 2018). This risk profile could then be used, alongside a separate assessment of specific risk for internalising, externalising and thought disorder in order to provide detailed information about a patient's overall risk profile to inform an intervention tailored to their specific needs (Carragher et al., 2016).

The above findings have been, for the most part, demonstrated across multiple studies and potential clinical recommendations are in keeping with the ethos of the NHS to provide least restrictive and client centred care (Mead & Bower, 2000). However, before any clinical recommendations can be implemented the generalisability of the study findings need to be fully considered. A number of studies, including this one, have only collected data on a limited spectrum of psychopathology

exhibited within a child and adolescent sample. This is particularly important given the suggestibility of the final model to the measures and symptoms items included. Further, only a handful of studies have included data from a clinical sample. Such limitations need to be addressed to ensure that any model of psychopathology truly reflects the full range of symptomology exhibited across the developmental stages and levels of severity. As once these limitations are addressed it may be found that the bifactor model is no longer the best fitting model to reflect the psychopathology structure, particularly given that the bifactor model fit was only marginally better than the other models examined. Further, recommendations based upon the impact of intervention on factor scores need to be made even more cautiously as the study was only able to explore the impact of intervention versus no intervention on factor scores. It could therefore be that if future research was to include domain specific treatment, such as cognitive behavioural therapy for depression, this may be found to account for the variance on the distress factor. However, sufficient treatment numbers within each domain would be needed to ensure sufficient power to explore the potential impact on each of the specific factors.

Conclusions

This study adds to the growing evidence base for the bifactor model of psychopathology and therefore a general psychopathology factor within child and adolescent samples. Adding weight to the idea that the specific disorders (fear, distress, externalising) share a set of aetiologic factors that are distinct from the ones that cause the specific disorders themselves (Lahey et al., 2012). However, the structure of the model varies across studies depending on factors such as sample type, questionnaires and symptomology assessed. Future research should therefore focus on data which covers a range of mental health severity, the full spectrum of psychopathology symptoms within a clinical and non-clinical sample to ensure any

identified model capture the full psychopathology spectrum. As with previous literature there appears to be strong homotypic continuity within the bifactor models, with intervention having no impact on factors scores apart from general psychopathology factor. These finding bring into question what aspect of psychopathology that the interventions are and should be targeting. However, findings need to be interpreted cautiously as this is the only the second study looking at the impact of intervention on the bifactor model and further research is required.

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Part 3: Critical Appraisal

Introduction

The critical appraisal will address in greater detail some of the issues that arose while undertaking the research project. The challenges surrounding this project were predominantly related to the use of secondary data and the limitations this comes with. Firstly, it will start by exploring the impact of data quality and completeness on the direction of the research and research questions. Particularly as the lack of sufficient paired data significantly impacted which data was included in the final analysis. Secondly, it will look at how the restrictions as to which questionnaire data was included in the analysis has impacted the extent to which the limitations of previous studies may have been addressed within the empirical study. Finally, it will address the limit to which this research project was able to add to and extend the current evidence base, particularly in relation to the impact of routinely administered intervention on the stability and phenotypic expression of psychopathology.

The direction of the research

Interest in the research project was initially sparked by the prospect of better understanding the impact of child and adolescent resilience on therapy outcomes. Having worked across a number of services which provide intervention for children and adolescence, it was clear that there was a subset of individuals, who despite exposure to significant adversity are able to maintain positive adjustment. In the literature these children are referred to as resilient (Masten, 2011). Therefore, the initial proposed project was to utilise a large pre-existing, national, child and young people dataset to develop and validate a measure of resilience and explore the extent to which qualities of resilience in children and young people can explain the variance in therapy outcome.

Initial scoping of the data indicated that measures which capture the main domains of resilience were included. Such measures were the Strength and

Difficulties Questionnaire (SDQ), a 25 item scale focusing on children's positive and negative attributes (Goodman, Meltzer, & Bailey, 1998) and the SCORE 15, a 15 item scale looking at family functioning derived from the original SOCRE 40 (Stratton, Bland, Janes, & Lask, 2010). However, there was significant site variation in the frequency to which certain measures were completed within services. Therefore, when these measures were combined there was not a sufficient number of participants with paired data for both scale. Meaning that, due to insufficient power, it would not have been possible to conduct the analysis required to develop a resilience questionnaire. In fact, despite the initial data set including just over 133,000 participants, only 58 had paired data for the SDQ and SCORE 15. This brought to the forefront two of the major challenges of utilising secondary data. The first is that as a researcher you cannot influence what data is included in the dataset and therefore are limited to how you might go about answering your research question. Second is the potential for the completeness of the data to be of poor quality and to be great variation in the type of data collected across sites. This is particularly the case for this dataset which includes routinely collected data (Benchimol, Smeeth, Guttmann, Harron, Moher, Petersen et al., 2015), as within health setting it is possible for clinicians to forget or not have time to administer or check the completeness of the questionnaires. Therefore, unfortunately, the direction of the project was to a major extent dictated by which questionnaires had sufficient data to enable the analysis to be conducted.

More detailed exploration of the data identified that there were only two measures which had sufficient completed and paired data. These measures were the SDQ and Revised Child Anxiety and Depression Scale (RCADS), a 47 item self-report measure that assesses the main features of five anxiety disorders: separation anxiety, social phobia, generalised anxiety, obsessive-compulsive disorder and panic, as well as major depressive disorder (Chorpita, Moffitt, & Gray, 2005). Given the limited

information collected by these scales, which mainly included symptomatology of common mental disorders, the research questions that could be addressed by the dataset was restricted. Additionally, although the data did not supporting the original research question; it was important that the topic addressed was still meaningful within a clinical context. The final proposed research project therefore focused on exploring the extent to which a bifactor model, with both specific factors and a general psychopathology factor, fitted the data better than a categorical model of mental disorder in children and adolescents. This is particularly pertinent in light of the challenges related to categorising and treating mental health difficulties due to the high levels of co-morbidity (Kotov, Krueger, Watson, Achenbach, Althoff, Bagby et al., 2017). Therefore, it is possible that looking at psychopathology from a different perspective and not the diagnostically driven categories currently used, may aid in us better understanding and therefore treating mental distress.

Overall, though detailed scoping of the data, it was possible to overcome the challenge of ensuring sufficient data to answer a meaningful research question. However, it is questionable as to the impact such initial exploration had on the a prior nature of the study hypothesis, as it was not possible to confirm power without conducing some very basic initial analyses. However, there were still a number of limitation with the data, which were related to the extent to which the study finding may have been generalisable to a wide reaching British health care service. This was particularly in the context of the limited scope, in terms of mental health severity and range of symptomology, of the questionnaires included in the analysis.

The generalisability of the findings

There were a number of study limitations identified with in the current evidence base examining the existence of a general psychopathology factor and use of a bifactor model within child and adolescent data. These included the use of diagnostic

rather than symptom level data, despite the risk of artefactual co-morbidity. As well as the variation in the mental disorders included, for example some studies included more complex or lifespan conditions such as psychosis (Afzali, Sunderland, Carragher, & Conrod, 2018; Carragher, Teesson, Sunderland, Newton, Krueger, Conrod et al., 2016) and Autistic Spectrum Disorders (ASD) (Bloemen, Oldehinkel, Laceulle, Ormel, Rommelse, & Hartman, 2018; Noordhof, Ormel, Oldehinkel, & Hartman, 2015). Lastly, there was little consistency in the measures used within studies to collect the psychopathology data and many of the previous studies only included American samples (Hankin, Davis, Snyder, Young, Glynn & Sandman, 2017; Lahey, Rathouz, Keenan, Stepp, Loeber, & Hipwell, 2015; McElroy, Belsky, Carragher, Fearon, & Patalay, 2018; Olino, Bufferd, Dougherty, Dyson, Carlson, & Klein, 2018; Snyder, Young, & Hankin, 2017, 2019; Tackett, Lahey, Van Hulle, Waldman, Krueger & Rathouz, 2013). All of these limitations impact on the generalisability of the study findings to a British health care setting.

It was possible to address some of these limitations within the study, for example the sample was a British clinical sample and the psychopathology data collected was symptom rather than diagnostic level data. However, due to the limited choice of questionnaire data to extract from the dataset, the items included within the analysis meant that any model of psychopathology was not able to reflect the full range of symptomology exhibited across the developmental stages. As mentioned previously only the RCADS and SDQ was included. The RCADS only captures symptoms related to common anxiety disorders, such as generalised anxiety and panic disorder, and depression (Chorpita et al., 2005). Further, there are a disproportionate number of anxiety related items as compared to depression symptoms. The SDQ also measures a number of emotional difficulties; however, some items did capture symptoms related to hyperactivity and conduct disorder (Goodman, Lamping, & Ploubidis, 2010), but these were disproportionally lower. It

would therefore only be possible for the psychopathology model to capture internalising and externalising disorders. Further, due to the higher weighting of items capturing emotional difficulties, the model would be more strongly influenced by the internalising disorders, particularly fear disorders. It was therefore not possible to explore whether the externalising factor also further divide with the inclusion of more fine grained symptomology data, as has been previously found with internalising disorders. Previous research has suggested that the externalising factors may divide into two factors, one capturing the problems of attention, impulsivity, and hyperactivity and the other capturing conduct problems and aggressive behaviour (Frick & Kimonis, 2005) as shown in the study by Pettersson, Lahey, Larsson, and Lichtenstein (2018). Another limitation of being able to only include the RCADS and SDQ is that the externalising disorder items only captured conduct and hyperactivity symptoms. Meaning that adolescent issues such as alcohol and drug use were not included as it has been in other prior studies (Carragher et al., 2016).

No items assessing symptoms of psychosis or ASD were included in the measures selected. Therefore, it was not possible to examine the impact of these more complex mental health problems or lifespan conditions on the bifactor model. For example, to confirm the presence of a thought disorder factor, which had been identified in two of the three prior studies conducted on a clinical sample (Haltigan, Aitken, Skiling, Henderson, Hawke, Barraglia et al., 2018; Hyland, Murphy, Chevlin, Carey, Vallières, Murphy et al., 2018). Nor would it be possible to further explore whether developmental conditions such as ASD load onto their own specific factor, as has been shown in a number of prior studies (Bloemen et al., 2018; Murray, Eisner, & Ribeaud, 2016; Noordhof et al., 2015; Pettersson et al., 2018). Restricting the analysis to such a limited spectrum of severity is likely to have limited the generalisability of the model of psychopathology to a more mild to moderate presentation. A further challenge is that there is little consistency in the questionnaire

data included in the bifactor analysis across the studies. This particularly important given that the raw questionnaire appears to have a substantial impact on specific factors extracted. For example in the empirical paper the fear – cognition factor is mainly comprised of items from the generalised anxiety subscale of the RCADS; the fear - bodily sensation factor from the panic disorder subscale and the distress factor from the major depression subscale. To enable more direct comparison it would therefore have been beneficial to try and use measures that have been included in previous studies. Whereas the SDQ has been used in a number of previous studies (Afzali et al., 2017; Carragher et al., 2016; Patalay, Fonagy, Deighton, Belsky, Vostanis, & Wolpert, 2015), this was not the case for the RCADS. Making it harder to attribute what variables led to the extraction of such vastly different specific factors, was it the use of a clinical sample or the particular questionnaire items that are included in the analysis.

Overall, being able to include only two questionnaires meant that the range of symptomology and disorder severity captured by the psychopathology model was limited. Which bring to question the extent to which the final bifactor model truly represents child and adolescent psychopathology and therefore how far the study finding can be generalised to the British health care system. Another major criticism of the research project was the concern over data completeness. Which in turn potentially impacted in the extent to which the study was able to not just add to but also extend the current evidence base in a way that would be clinically meaningful.

Extending the current evidence base

Previous studies have made a number of clinical recommendations based upon their finding. These include, using individual's scores on the specific and general factors, of the bifactor model, to develop an individualised psychopathology profile and treatment plan (Carrager et al., 2016). With those scoring higher on the general

psychopathology factor potentially benefiting from a transdiagnostic approach to treatment to target the core symptoms which underly most disorders (Afzali et al., 2018; Hyland et al., 2018). Although these recommendations may lead to more effective client centred care, they have been mostly made from studies which have not included clinical data. In fact, to date, only three studies have included data from a clinical sample (Constantinou, Goodyer, Eisler, Butler, Kraam, Scott et al., 2019; de la Cruz, Vidal-Ribas, Zahreddine, Mathiassen, Brøndbo, Simonoff et al., 2018; Haltigan et al., 2018). Further, only one study has actually assessed the potential impact of such interventions on the stability of the bifactor model and phenotypic expression of psychopathology (Constantinou et al., 2019). In light of this, the research project aimed to extend the current evidence base by starting to explore the extent to which interventions routinely administered within CAMHS influence both the specific factors and general factor at last assessment.

The specific interventions offered to each participant was included with in the national dataset and appear to have sufficient completed data to be included within the analysis. Intervention type was initially collapsed into boarder categories such as, individual therapy, family intervention, parenting intervention, pharmacological advice. However, even when collapsed into these broader categories, no group of interventions had a sufficient number of participants to be included separately in the analysis. Therefore analysis had to be conducted at the broadest level of whether a participant was offer or not offered an intervention. Collapsing the data to these two categories may have had a substantial impact on whether there was enough detail within the data to detect an impact of intervention on child and adolescent psychopathology. This is particularly as the interventions included a broad range that had been developed to treat vastly different mental health symptomology. Therefore, when all intervention were combined in to one category ‘offered any intervention’, this may have artificially created a category which, which at a sample level, is assessing

the impact of a transdiagnostic intervention. As mentioned previously, studies have hypothesised that those with a higher general psychopathology score might benefit from a transdiagnostic approach to treatment (Afzali et al., 2018; Hyland et al., 2018). Therefore, the use of the more inclusive categories may account for why the empirical research found the intervention only significantly predicted the last assessment factor score for the general psychopathology factor. Therefore, it would have been more beneficial to have used the more specific categories or even the individual intervention type within the analysis. If possible, such analysis may have found that certain intervention types may have impacted one but not all of the specific factors. Taking Multi Systemic Therapy (MST) as an example, which is an intervention developed to support young people who have significant offending and behaviour problems (Van der Stouwe, Asscher, Stams, Deković, & van der Laan, 2014). If entered individually into the analysis, MST may have been found to significantly predict the last assessment factor scores of the externalising factor which is comprised of items which are used to assess conduct disorders, as previously found (Constantinou et al., 2019). However, in the absence of such analysis, it is not possible to be certain as to whether this explains why intervention type only impacted on the general psychopathology factor. It could in fact be the case that intervention only impacts on the core symptoms which underly disorders and the specific factors remain stable over time.

The potential for site variation in how the intervention data was recorded created further complexity when interpreting the findings from the empirical paper. For example, how sites or for that matter, individual therapists may have categories the intervention offered may be quite different. This is because within routine care intervention type can be integrative, pulling on aspects of different interventions to meet individual need (Gilbert & Orlans, 2010), meaning the intervention may not fall neatly within one of the prespecified categories. Further, within the data set, of those offered an intervention 37.7% were offered more than one intervention. Therefore,

even if it was possible to look at individual interventions, it would not have been possible to be certain that it was that particular intervention that had the impact. Of particular concern was the group that had been offered no intervention. As all participants were under the care of CAMHS; it is not clear why an individual would be under the care of mental health service and not be offered any intervention. Particularly, as the data captured an individual's episode of care not just a snap-shot in time, which might have meant that those individuals were on a 'waiting list' for care. In light of this, it is not possible to know what 'no intervention' actually looked like. Could it be that intervention was not accurately recorded for those individuals or did they drop out before therapy was offered. Further, it is likely that 'no intervention' may look very differed in each site and was no way to control for such variation.

Overall, there are substantial limitations regarding the quality of the data capturing the intervention type. Particularly with regards to the sample size at the individual intervention level and the extent to which data was comparable between sites. Such limitation will have considerably impacted on the extent to which this research project was able to extent the current evidence bas. Therefore, although it was possible to start to explore the impact of routinely administered intervention on the stability and phenotypic expression of psychopathology the finding need to be interpreted with considerable caution. Meaning that clinical recommendation based upon such finds, should only be made following further research.

Conclusions

The research project has highlighted some of the major strengths and limitations of utilising secondary data, particularly that collected at a national level. Despite, the challenges that have been overcome throughout the projects. From the need for paired data limiting the choice of questionnaire data that could be included in the analysis to the potential variation in data quality and control across sites. It is

clear that given the limited time and man power allocated to the project it is only through the use of a secondary dataset that such a project would have been possible. The use of the CORC dataset not only enable the project to be sufficiently powered for the analysis to be conducted. The inclusion of data from service across Britain increases the generalisability of the finds to those utilising secondary and tertiary CAMHS. Therefore, any limitations needed to be weighed up against the potential benefits of utilising a particular dataset. Overall, the project was able to achieve its study aim of examining, using bifactor modelling, the notion of a general psychopathology factor within clinical data from a child and adolescent sample. Further it is the only the second study to have explored the potential impact of clinical intervention on the factor scores over time, highlighting the need for further research in this area.

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Appendix

Appendix 1: Refences for the 51 articles extracted from PubMed on the 6th January 2020

Included: Bifactor modelling in child and adolescent sample

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Appendix 2, Table 1: Model results for the 36 item confirmatory factor analysis 4 factor model

	Factor 1 Fear - Cognitions		Factor 2 Fear – Body sensations		Factor 3 Distress		Factor 4 Externalising	
	Estimate	**S.E**	**Estimate**	**S.E**	**Estimate**	**S.E**	**Estimate**	**S.E**
RCADS 1 - I worry about things	1.000	0.000						
RCADS 13 - I worry that something awful will happen to someone in my family	0.692	0.008						
RCADS 22 - I worry that bad things will happen to me	1.081	0.006						
RCADS 27 - I worry that something bad will happen to me	1.070	0.006						
RCADS 35 - I worry about what is going to happen	0.975	0.006						
SDQ Worries - I worry a lot	0.979	0.007						
SDQ Fears - I have many fears, I am easily scared	0.729	0.009						
RCADS 3 - When I have a problem, I get a funny feeling in my stomach			1.000	0.000				
RCADS 14 - I suddenly feel as if I can't breathe when there is no reason for this			1.161	0.010				
RCADS 24 - When I have a problem, my heart beats really fast			1.120	0.010				
RCADS 26 - I suddenly start to tremble or shake when there is no reason for this			1.173	0.011				
RCADS 28 - When I have a problem, I feel shaky			1.172	0.010				
RCADS 34 - All of a sudden I feel really scared for no reason at all			1.268	0.011				
RCADS 36 - I suddenly become dizzy or faint when there is no reason for this			1.041	0.011				
RCADS 39 - My heart suddenly starts to beat too quickly for no reason			1.216	0.011				
RCADS 41 - I worry that I will suddenly get a scared feeling when there is nothing to be afraid of			1.217	0.010				
SDQ Aches - I get a lot of headaches, stomach-aches or sickness			0.812	0.012				
RCADS 2 - I feel sad or empty					1.000	0.000		
RCADS 6 - Nothing is much fun anymore					0.827	0.006		
RACDS 11 - I have trouble sleeping					0.702	0.008		

RACDS 15 - I have problems with my appetite		0.654	0.008		
RCADS 19 - I have no energy for things		0.893	0.006		
RCADS 21 - I am tired a lot		0.827	0.007		
RACDS 25 - I cannot think clearly		0.921	0.006		
RCADS 29 - I feel worthless		0.993	0.005		
RCADS 37 - I think about death		0.832	0.006		
RCADS 40 - I feel like I don't want to move		0.892	0.006		
SDQ Solitary- I am usually on my own. I generally play alone or keep to myself		0.488	0.010		
SDQ Unhappy - I am often unhappy, down-hearted or tearful		0.950	0.006		
SDQ Restless - I am restless, I cannot stay still for long				0.742	0.008
SDQ Tempers - I get very angry and often lose my temper				0.628	0.010
SDQ Fidgety - I am constantly fidgeting or squirming				0.797	0.008
SDQ Fights - I fight a lot. I can make other people do what I want				0.450	0.012
SDQ Distractible - I am easily distracted, I find it difficult to concentrate				0.802	0.010
SDQ Lies or cheats - I am often accused of lying or cheating				0.396	0.012
SDQ Steals - I take thing that are not mine from home, school or elsewhere				0.336	0.015

Appendix 3, Table 2: first and last assessment mean factor scores and change

	First assessment		Last assessment			
	Mean	**SD**	**Mean**	**SD**	**T (DF)**	**p**
General Psychopathology	0.00301	0.890774	0.02273	0.845381	-1.153 (2946)	0.249
Fear - cognitions	-0.04771	0.725082	-0.01844	0.696344	-1.933 (2946)	0.053
Fear – bodily sensation	-0.00145	0.824076	-0.00214	0.770066	0.043 (2946)	0.965
Distress	-0.00777	0.853502	-0.01716	0.788453	0.582 (2946)	0.560
Externalising	0.00102	0.858241	-0.01191	0.804850	0.809 (2946)	0.419

Appendix 4, Table 3: Individual predictors of all last assessment factor scores

			First assessment								
			GF	**F1**	**F2**	**F3**	**F4**	**Gender**	**Ethnicity**	**Age**	**Therapy**
Last assessment GF (General factor)	Constant	B	0.021	0.014	0.022	0.020	0.023	0.023	0.015	-0.175	0.054
		(SE)	(0.014)	(0.015)	(0.015)	(0.015)	(0.015)	(0.016)	(0.016)	(0.134)	(0.022)
	Predictor	B	0.407	-0.177	-0.293	-0.327	-0.321	0.000	<0.000	0.014	-0.066
		(SE)	(0.016)	(0.021)	(0.018)	(0.017)	(0.017)	(0.000)	(0.000)	(0.009)	(0.031)
	F		665.695*	69.856*	260.743*	361.183*	349.610*	0.177	2.658	2.219	4.479**
	R^2		0.184	0.023	0.081	0.109	0.106	0.000	0.001	0.001	0.002
Last assessment F1 (Fear – cognition)	Constant	B	-0.018	-0.003	-0.018	-0.017	-0.019	-0.019	-0.023	-0.751	0.002
		(SE)	(0.013)	(0.012)	(0.012)	(0.012)	(0.013)	(0.013)	(0.013)	(0.109)	(0.018)
	Predictor	B	-0.089	0.318	0.214	0.200	0.081	<0.000	<0.000	0.051	-0.042
		(SE)	(0.014)	(0.017)	(0.015)	(0.015)	(0.015)	(0.000)	(0.000)	(0.008)	(0.026)
	F		38.484*	363.785*	202.749*	187.408*	29.929*	0.015	1.579	45.579*	2.630
	R^2		0.013	0.110	0.064	0.060	0.010	0.000	0.001	0.015	0.001
Last assessment F2 (Fear – bodily sensation)	Constant	B	-0.002	0.010	-0.002	0.000	-0.002	-0.003	-0.002	-0.372	0.008
		(SE)	(0.014)	(0.014)	(0.013)	(0.013)	(0.014)	(0.014)	(0.015)	(0.121)	(0.020)
	Predictor	B	-0.188	0.257	0.384	0.291	0.228	0.000	<0.000	0.026	-0.021
		(SE)	(0.016)	(0.019)	(0.016)	(0.016)	(0.016)	(0.001)	(0.000)	(0.008)	(0.028)
	F		145.577*	183.302*	598.532*	342.918*	202.586*	0.585	0.002	9.383*	0.528
	R^2		0.047	0.059	0.169	0.104	0.064	0.000	0.000	0.003	0.000
Last assessment F3 (Distress)	Constant	B	-0.016	-0.004	-0.017	-0.014	-0.017	-0.017	-0.015	-0.632	-0.004
		(SE)	(0.014)	(0.014)	(0.013)	(0.013)	(0.014)	(0.015)	(0.015)	(0.124)	(0.020)
	Predictor	B	-0.268	0.285	0.361	0.401	0.302	0.000	<0.000	0.043	-0.027
		(SE)	(0.016)	(0.019)	(0.016)	(0.015)	(0.016)	(0.001)	(0.000)	(0.009)	(0.029)
	F		297.758*	216.611*	488.578*	682.846*	356.541*	0.184	0.218	24.872*	0.869
	R^2		0.092	0.068	0.142	0.188	0.108	0.000	0.000	0.008	0.000
Last assessment F4 (Externalising)	Constant	B	-0.011	-0.007	-0.011	-0.010	-0.012	-0.012	-0.006	0.404	-0.004
		(SE)	(0.014)	(0.015)	(0.014)	(0.014)	(0.013)	(0.015)	(0.015)	(0.127)	(0.021)
	Predictor	B	-0.271	0.109	0.292	0.309	0.428	0.000	<0.000	-0.029	-0.017
		(SE)	(0.016)	(0.020	(0.017)	(0.016)	(0.015)	(0.001)	(0.000)	(0.009)	(0.030)
	F		290.573*	28.816*	287.998*	354.859*	776.136*	0.069	1.677	10.866*	0.315
	R^2		0.090	0.010	0.089	0.107	0.209	0.000	0.001	0.004	0.000

*significant at the <0.001 level; **significant at the <0.05 level