Modelling Psychopathology: Towards a Transdiagnostic Understanding of Psychopathology

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Thesis declaration form

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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

The scope of the current body of work addresses the matter of the paradigm of the empirically derived structure(s) of psychopathology in adults. The discussion addresses how the comorbidity or the co-occurrence of symptoms influences how the nosology of mental ill health is organized and tabulated. The first part of the thesis is a systematic literature review of empirically defined models of psychopathology that have been derived using latent modeling techniques. The narrative of over 40 years' worth of research is discussed in terms of the nosological conceptualization of how patterns of discrete mental health symptoms occur and co-occur. Specifically, efforts were made to look into the over-arching 'multifactorial models' of psychopathology.

In the second part of thesis a concept known as the general factor of psychopathology denoted as *p*, that represents a statistical summary of comorbid patterns of psychological ill health is taken further and explored in a mixed sample of patient and control participants. The hope is that this work will be taken forward in support of the current zeitgeist in the fields of psychiatry and clinical psychology which favour transdiagnsotic concepts in nosology and guide research efforts into the aetiology of mental ill health and applications thereof in the clinic.

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Part 1: Literature Review

To date, what has latent modelling revealed about the general structure of psychopathology in adults and the processes that underlie it.

Abstract

Aims: The aim is to investigate the narrative of published empirical efforts made to investigate the general structure of psychopathology; specifically, multivariate models of common mental health symptoms. Furthermore, the aim is also to extract the proposed theoretical functional underpinnings of proposed structure(s) of psychopathology.

Method: A systematic search using two databases and was conducted. Empirical papers were selected using exclusion criteria. Abstracts were screened for studies which described latent modelling techniques for common mental health symptoms, within a multivariate model of psychopathology for adult populations only. Empirical studies using cluster analysis and latent class modeling focused on typologies or the grouping of participants were excluded.

Results: In total 20 papers were selected for inclusion. Considerations were made for the vast and often incomparable classes of analytic methods.

Conclusions: The wide variety of empirical investigations over the past 40 years indicate that a multifactorial model of psychopathology converges towards an Internalising- Externalising (INT-EXT) division among common Mental Health Symptoms. However there are further efforts for the successful reconceptualization of a general factor of psychopathology denoted as *p*, which subsumes but does not contradict the INT-EXT as a clinically useful construct.

To date, what has latent modelling revealed about the general structure of psychopathology in adults and the processes that underlie it?

It has been five years since the publication of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) of the American Psychiatric Association (APA, 2013). Since the publication of the first edition in 1952, the advent of every revision highlighted subtle variations in the professional conceptualisation of the nature of mental health disorders. Brought together, these variations tell a narrative of the paradigmatic shifts in the scientific understanding and treatment of mental ill health. Beginning with the elimination of grand theories and introduction of the descriptive, and theoretically neutral classification system that occurred between DSM-II to DSM-III in 1980, to the removal of many diagnostic inconsistencies that lead to DSM-III-R in 1987; each publication represents a refinement to the grievances of its predecessor ("American Psychiatric Association", 2016). In doing so, it directs our understanding of the processes that underlie mental illness in key ways.

Among the many specific changes in the latest edition are the combining and splitting of specific mental health disorders, such as relocating Bipolar Disorder away from 'Mood Disorders' and under 'Psychotic Disorders'. As well as the additions and removals, renaming of disorders is the recognition of a broader clustering among diagnostic entities, to name but a few of the recent changes. Clusters of diagnostic entities are represented as "meta-structures" or the organizational framework through which discrete

diagnostic entities are thought to share pathophysiology, genetic loadings, health correlates, and other findings from neuroscience and clinical presentation. This not only entails a re-tabulation of the mental health nosology but incur new systems of formulating and researching the scientific study of psychopathology (Reiger, 2013). This systematic literature review aims to review empirical research efforts that have sought to describe and organise the meta-structure of common mental health disorders and/or symptoms, and relay the implications of their findings as they pertain to daily clinical practice and research.

Understanding Comorbidity

According to Hyman (2010) comorbidity poses a problem for both researchers and clinicians alike. In line with the criticisms of the categorical approach to psychopathology, Lillenfeld et al. (1994) argue that the use of the term comorbidity is not helpful in psychopathology research because it erroneously substantiates constructs of mental health disorders as having discrete and established aetiologies. From a clinical perspective, concerns have also been raised about comorbidity, (e.g. Kaplan et al. (2001)), who remarked that few children in a clinic represent prototypical cases of specific disorders. For instance, describing a child as having “comorbidity” may not be helpful in case formulation and in parent communication. Instead it may be better to communicate about general mechanisms that may unite putatively distinct disorders.

Historically, the problem was particularly prominent in the development of the DSM –III (APA, 1980). Boyd et al., (1984) questioned the hierarchical exclusionary rules of the then classification system, which they found affected

no less than 60% of disorders. Using empirical methods in the large-scale Multi-site Epidemiological Catchment Area project, Boyd et al., (1984) found among similar diagnostic entities, the presence of one disorder greatly increased the probability of having a differential disorder. As exclusionary rules were relaxed or removed with further publications of the DSM, an explosion of research across clinical and epidemiological samples occurred. However, as documented by Clark et al. (1995), the presence of comorbidity may be due mainly to the descriptive, categorical system of classification that is also atheorteical in that it offers no aetiological underpinnings. Other researchers go as far as stating that "[Clinical] comorbidity may be nothing more than an artefact of an imperfect diagnostic system" (Belzer & Scheier, 2004, p. 297), "largely the product of a nosological system that classifies mental disorders categorically, presupposing discrete diagnostic entities" (Krueger & Markon, 2006b .

What does Comorbidity Mean?

In much of the conceptual literature reviewed on the matter, there is a narrative that lends the term comorbidity a negative connotation. This is based on the logical assumption that the presence of two or more distinct constructs in a clinical setting represents measurement error in clinical assessment. This is can be framed by the assertion by Vella et al. (2000) who suggest that "comorbidity should be defined as two or more diseases, with distinct aetiopathogenesis (or, if the aetiology is unknown, with distinct pathophysiology of organ or system), that are present in the same individual in a defined period of time" (p. 25).

From a transdiagnostic perspective the ‘problem’ of comorbidity may be a fallacy, especially if we are to consider the psychpathology in terms of the Common Factor Model. Comorbidity comprises the unit for factor analytic research at the level of psychometrics that may guide further investigation to explore the narrative or aetiopathoenesis driving psychopathology. Using latent modelling to delineate transdiagnostic processes, Rodriquez- Seijuas et al. (2015) state that this paves a path for treatment options. For instance, in the case of Cognitive Behavioural Therapy (CBT), focusing on reframing distressing cognition has far reaching implications in ameliorating low mood as well as anxiety (Rodriquez-Seijuas et al., 2015). Research on comorbidity offers the potential to better illustrate aetiology in terms of bio-psychological processes. This allows for multi-systemic foci for intervention and prevention of psychopathology, by encouraging researchers and clinicians to formulate comorbid conditions in functional terms.

Towards a Transdiagnostic Approach

Rodriquez- Seijas et al. (2015) address and summarise the weaknesses of the traditional classification systems, used in previous versions of DSM but not DSM-IV, whereby an individual’s clinical presentation requires meeting a certain number criteria threshold for a particular disorder. Each criterion tallies equally towards a diagnosis, and the final diagnosis is dichotomous - present or absent. Although the DSM-5 still lists disorders, there is a realisation that the symptoms that occur in one disorder may occur in another disorder. There had been an intended shift toward “dimensional assessments” which allow clinicians to consider the severity of symptoms and

account for specific symptoms that cut across multiple diagnoses (Clay, 2011). Clinically, these symptoms are formulated along transdiagnostic psychological processes that underlie variations in clinical presentations (Brown & Barlow, 2009). Brown & Barlow (2009) go on to state that, among the advantages of the categorical diagnostic system, it favours high rates of diagnostic reliability and professional communication. However, one cost of this communication is the loss in construct validity.

Another problem in the categorical system is discontinuity: a dichotomous communication of psychopathology. Discontinuity potentially ignores critical information about individuals who do not meet the full criteria for a diagnosis. Clinically, the implication of this system is that it disadvantages individuals who appear with subclinical presentations from receiving recognition for their difficulties and could prevent them from accessing necessary services.

In one solution to this problem, Rodriquez- Seijuas et al. (2015) advocate for the shift towards a tansdiagnostic approach of understanding psychopathology. They expand on previous research that conceptualise the wide array of clinical diagnoses empirically shown to be reduced to latent factors or Internalising (INT) and Externalising (EXT). Through the use of Psychometric Factor Analytic Techniques, variables not directly measured, but accounted for may represent the relationship between directly measured variables. In the well-replicated two factor INT-EXT model, INT represents and is defined as the relationship between most mood and anxiety disorders. In contrast, EXT describes the relationship among antisocial personality disorder, behavior problems, impulsivity, and ‘acting out’ (Krueger, 1998).

Krueger & Makron (2006a) argue that INT-EXT may represent degrees of severity of normal personality. This conceptualization allows for a breakdown of discrete definitions of psychopathology by allowing continua to these characteristics and in doing so circumvents the discontinuity and heterogeneity problem of the categorical system. Rodriquez- Seijuas et al. (2015) propose that mental health disorders may be best represented and investigated as variants of similar underlying factors. They make reference to Thurston's Common Factor Model (1947) which posits many related observed phenomena (or in the case of mental health, symptoms) are potentially manifestations of a reduced number of latent dimensions and/or processes, which they believe accounts for patterns of comorbidity between certain disorders.

Research Domain Criteria

The implications of understanding inter similarities and co-occurrences of diagnostic entities, as they are clinically recognised by modelling latent variables, is that it spurs further research to identify shared bio-psycho-social patterns or transdiagnostic processes. The National [US] Institute of Mental Health's (NIMH) Research Domain Criteria (RDoC) initiative was set up to establish the study of psychopathology by investigating clinically relevant processes within the bio-psycho-social framework. RDoC is an attempt to foster the zeitgeist of dimensional conceptions of mental health problems around clinically relevant process constructs that are recognised as having neurobiological as well as psychological referents (Kozak & Cuthbert, 2006). RDoC provides a provisional matrix that includes rows of multiple specific psychological constructs:

- *Negative Valence systems* such as response to acute threat or potential harm as in the case of anxiety;
- *Positive Valence Systems* such as reward valuation;
- *Cognitive systems* that include, but are not limited to, attention, cognitive control, memory and language;
- *Systems for Social processes* such as affiliation and attachment;
- *Arousal Regulatory Systems*, defined as systems responsible for homeostatic regulation and response to various contexts;
- *Arousal* that consists of the continua of sensitivity of organisms to internal and external stimuli.

Modelling Comorbidity

In a review on the literature on comorbidity and the debate on the structure and nosology of psychopathology, Krueger & Markon (2006b) present a synthesis of the hypothetical models that could logically explain pathogenesis of comorbidity. Mainly based on papers by Klein & Riso (1993) and Neal & Kendler (1995), known as the KRNK bivariate models (See Figure 1 for a reproduction) these models provide a theoretical guide as to how investigations have described the manner in which disorders relate to one another and could explain the underlying processes:

The associated liabilities model. (Kreuger & Markon, 2006b) postulates that, between any two correlated disorders or symptoms, there is hypothetical latent liability factor (*r*), described as the propensity to develop the manifest disorders. The occurrence between the two hypothetical disorders may occur by chance or coincidence in which case the liability factor is uncorrelated (*r=0)*. At the other extreme, if the manifest symptoms present

alternative forms of the same disorder, the liability factors are perfectly correlated (*r=1)*.

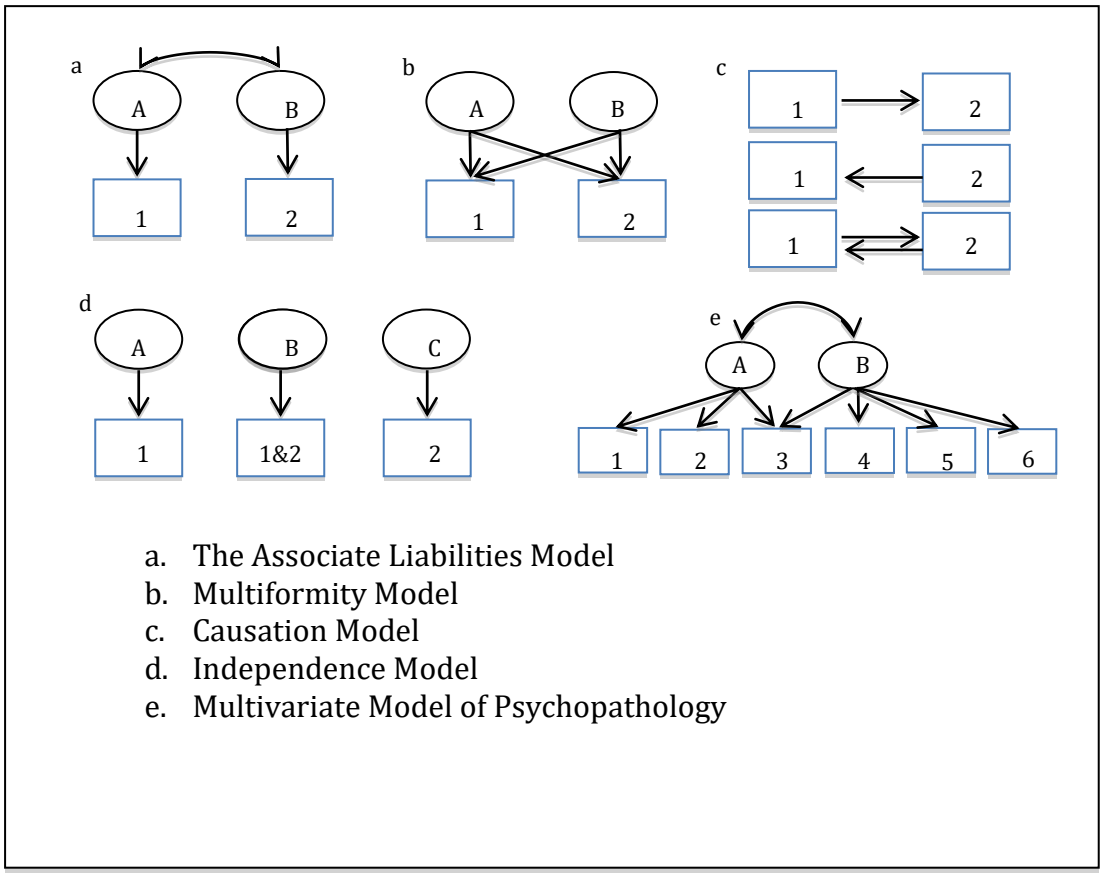
Multiformity Models .maintain that there are several liabilities to each of the manifest variables that are independent and uncorrelated, but that both liability factors can influence either manifest variables.

Causation model. This model does not consider latent liability factors influencing manifest variables. Rather, it posits that comorbidity arises through the reciprocal interplay between two disorders or manifest symptoms directly influencing one another.

The independence model. Iin this model**,** a disorder reflects an independent condition separate from the other disorders. This allows parameters to postulate that several theoretically uncorrelated liability factors may be at play in influencing respective manifest disorders or symptoms, while another liability factor may underlie the combined presence of the manifest conditions. In this theoretical model, comorbidity of disorders does not represent the combined presence of two distinct disorders, rather a third distinct disorder.

Multivatiate models. In addition to the bivariate models, Krueger & Markon (2006b) cite the multivariate models of psychopathology (e.g. Krueger, 1999) accounting for more than two disorders, which can be thought of as extensions to bivariate models. They use the term, ‘liability-spectrum’ to refer to the underlying constructs that may be driving various symptoms of psychopathology that, in actuarial terms, represent a continuum of risk to the manifestation of symptoms.

Figure 1: Reproduction of the comorbidity models from Kreuger & Markon (2006b)



a. The Associate Liabilities Model
b. Multiformity Model
c. Causation Model
d. Independence Model
e. Multivariate Model of Psychopathology

Methods

In this review, I look at publications that have made attempts to chart multivariate models of psychpathology using latent modelling. I aim to review publications describing phenotypic symptoms represented as manifest variables related to one another. My objective is to investigate the questions:

(1) What has Latent Modelling revealed about the meta-structure of psychopathology?

(2) What are the functional processes that underlie the structure of psychopathology?

(3) Where does this body of literature lead in terms of how transdiagnostic processes can help our reconceptualization of psychopathology?

Initial Database Search

A systematic review of the literature was run using ‘SCOPUS’ and ‘PsycInfo’. The following search terms using Boolyen operators were used: (Psychopathology OR mental health OR internalising OR externalising OR Psychiatry OR comorbidity) AND (structure OR latent OR factor OR nosology). An initial review of the findings yielded 25,793 results on Scopus, and 12,721 articles. Only articles from peer-reviewed journals were included. In total 38,514 publications were listed.

Although the literature was replete with various investigations examining comorbidity between specific disorders and narrower latent constructs, for the purpose of this initial review a decision was made to screen only for Multivariate Models of Psychopathology at the highest order. Figure 2 illustrates a flow chart for the review process.

Inclusion Criteria. Articles were included in this systematic review if:

(1) They attempted to make use of latent modeling techniques across a wide array of symptoms and/or disorders. Specifically, multivariate models of psychopathology as demonstrated by Kreuger & Markon (2006b).

(2) Were a peer-reviewed publication.

(3) Were written in English.

Exclusion criteria. A review of the massive body of research that handled models for certain disorders, but not a general array of disorders. Papers were excluded if:

(1) The paper was conceptual in nature and did not report empirical findings.

(2) Publications exploring the psychoanalytic use of the term 'Latent Factor' were excluded.

(3) Empirical studies using latent modeling techniques for specific constructs, e.g. "Internalising Disorders", "Externalising Disorders" etc. were also excluded.

(4) Empirical studies using latent modeling techniques for the psychometric validation of particular most often psychometric instruments were also excluded.

(5) Further exclusion criteria removed empirical studies that used Cluster Analysis and Latent Class Modeling, that are focused on typologies or the grouping of participants.

(6) Conceptual publications that were related but non-empirical, and studies of specific construct e.g. Structure of Personality Disorders.

(7) An initial review of the outcome produced a series of publications that looked at narrow domains such as ‘Internalising’ disorders. A decision was made to reject these investigations in the interest of focus.

(8) Only studies with adult participants were considered. Exceptions were made for studies that included adolescents, where the age range surpassed 18, e.g. Yoder et al. (2008) whose age ranged between 18-25.

A hand search of the reference section of all articles was also conducted to determine if there were other articles that met the inclusion criteria.

Results

In total, the database results yielded a sizeable 38,514 peer-reviewed journal articles. Following the exclusion and inclusion criteria 18 peer-reviewed journal articles remained after all criteria were applied. It became apparent that there was a temporal distinction between studies using only Exploratory Factor Analysis and studies using Confirmatory Factory Analysis and variations thereof, in that all studies prior to 1998 used EFA (All EFA papers are listed in Table 1). I present them separately here.

Exploratory Factor Analysis.

Across the papers reviewed, chronologically, all initial investigations used EFA. In total, four studies were found to have made exclusive use of Exploratory Factor Analysis. The EFA of the articles found go as far back as 1970s used exploratory techniques such as Principal Component Analysis (PCA) and Exploratory Factor Analysis (EFA) with various aims. For instance, Lanyon et al. (1974) and Vet & Ware (1983) used PCA on various existing scales, with the aim of devising comprehensive clinical scales that could

delineate psychopathology from “normality”. Many of the interpretations of the factor solutions appeared uniquely in respective investigations as they were not subsequently replicated in any of the proceeding reviews, (e.g. Lanyon et al. (1974)’s Protestant Work Ethic).

Additionally, Rhoades et al. (1991) sought to build on the evidence base for the validity of the Diagnostic Inventory of Personality and Symptoms (DIPS; Vincent, 1985), based on the DSM-III, by exploring the common factors among its fourteen subscales across Axis I and Axis II. A four-factor solution was found and interpreted as neurotic, psychotic, characterologic [mmature] and somatic.

One study using exploratory techniques, Gotlib et al. (1984) examined relationships among several self-report measures of both nonclinical and subclinical groups. This study may have been the first to herald the notion of a single-factor solution for psychopathology, a construct later explored by Lahey at al. (2012). Gotlib et al., (1984) employed a battery of ‘gold standard’ measures of the day, such as the first edition of the Beck Depression Inventory (BDI-I, Beck et al,1961) and State Trait Anxiety Inventory (STAI, Spielberger, 1970). Although the factor solution was interpreted as having two factors, where the primary factor accounting for more than 50% of the variance was labelled ‘General Psychological Distress’.

Although no further claims were made to the general structure of psychopathology, the study echoed the high comorbidity between symptoms of depression and anxiety. A measure of assertiveness, the Rathus Assertiveness Schedule (Rathus, 1973) was used and found to load heavily on the primary factor with other measures of depression and anxiety. Gotlib

(1984) postulated that participants may have been “caught in the pathology … of maladaptive functioning” (p. 26) and alludes to the model of learned helplessness to account for the findings.

Figure 2. Flow Chart of Systematic Review Process

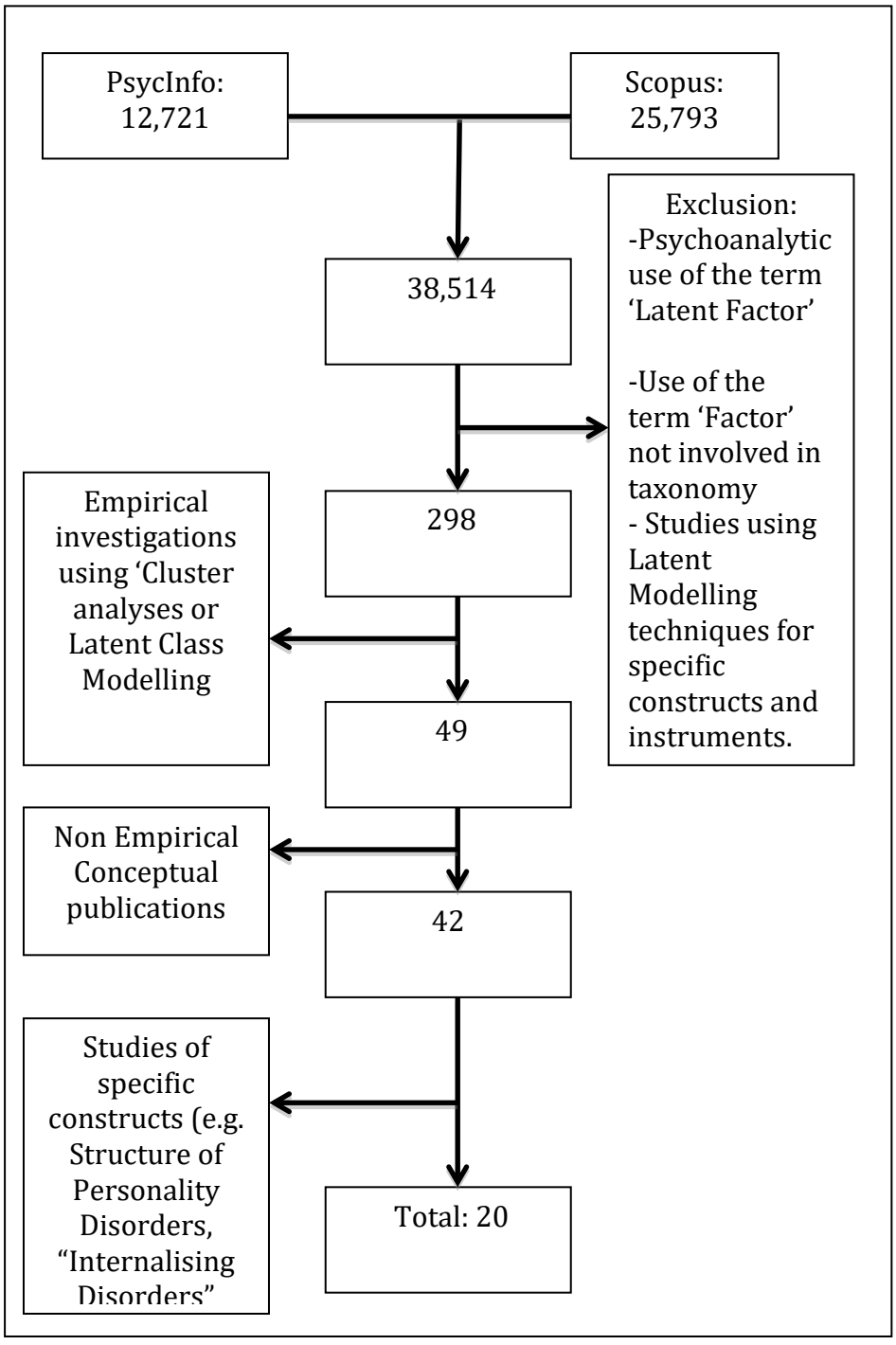


Table 1. Results of Papers using Exploratory Factor Analysis

	Title	Sample Characteristics	Time Points	Manifest variables	Method	Results/ Factor Interpretation
1	Lanyon et al.(1974)	Non-Clinical, N= 400	Cross-sectional	**Psychological Screening Inventory** (PSI;Lanyon, 1970)	PCA	**Five factors** (“Alienation, Extraversion”, “Acting out” ,”Protestant work ethic”&” General maladjustment”)
2	Vet & Ware (1983)	N=5,089 , National Health Insurance (NHI) Study (Multicentre, non-clinical)	Cross-Sectional	**Mental Health Inventory** (MHI)	PCA	**Hierarchical:** “Psychological Distress” -> “Anxiety, “Depression”, “Loss of behavioural/emotional Control” &”Psychological wellbeing”-> “general positive affect”, “emotional ties”
2	Gotlib (1984)	N=443 (Undergraduate)	Cross-sectional	**Beck Depression Inventory I (BDI-I;** Beck et al., 1961), **D-30 (of the Minnesota Multiphasic Personality Inventory** I; Depmsey, 1964**), Dysfunctional Attitudes** Scale (DAS; Weismann & Beck, 1978), **State** T**rait Anxiety Inventory** (STAI ; Spielberger, 1970) , **Multiple Affect Adjective Checklist** (MAACL(Depression, Anxiety , Hostility); Zuckerman &Lubin,1965), **The Symptom Checklist** (SCL-(Somatization ,obsessive Compulsive ,Interpersonal Sensitivity, Depression ,Anxiety ,Hostility, ,Phobic anxiety ,Paranoia, Psychoticism); **Rathus Assertiveness Schedule (**Rathus, 1973)	PCA Varimax	**One Factor (**50% of variance located, labelled “General psychological distress”, “dysphoria”, “General Malaise”.
4	Rhoades et al. (1991)	Clinical sample, N = 170	Cross Sectional	14 clinical scale scores of the **Diagnostic Inventory of Personality and Symptoms** (DIPS; Vincent,1985)	PCA-Oblique	**Four factor solution** : ‘Neurotic’, ‘Psychotic’,’Charecteraological [Immature]’, ‘Somatic’

Confirmatory Factor Analysis

All 16 studies using CFA were conducted over eight large datasets (See Table 2). Initially this was viewed as strength of this analysis, as it allowed for consistency and meaningful comparison between models constructed using data from the exact same participants.

Inspired by the works of Achenbach &, Edelbrock (1978), in children and adolescents, Krueger et al. (1998) was the first study to suggest the INT-EXT structure of psychopathology in adults. It was also the first publication to acknowledge the 'problem with comorbidity' and suggested a more parsimonious structure of psychopathology than the taxonomy of the DSM-III. It was also the first in the review that I have found to use hypothesis-driven Confirmatory Factor Analysis as opposed to other exploratory methods. It uses a epidemiological dataset - the Dunedin Multidisciplinary Health and Development Study - which samples longitudinal data of two birth cohorts at age 18 and 21 years in the city of Dunedin, New-Zealand.

The researchers highlight the two distinct problems with using EFA, put forth by Watson et al (1994). First, there are no infallible guidelines to help determine the appropriate number of factors to extract. Second, there are no guidelines to aid in determining the correct orientation of the factors in multidimensional space. This seminal investigation set precedence to the use of CFA to model the structure of psychopathology and has been cited by most of the publications highlighted in this systematic review. In fact, most of the studies reviewed selected as their final model a variation of the INT-EXT

construct on the basis of this study. It was therefore useful to code for comparisons against it (See Table 2).

Studies based on a single dataset were collated so that models tested could be meaningfully compared. Many of the studies followed different protocols for the comparison between models, selecting various model specification and techniques to model specification indices. Additionally, there was a myriad of various manifest variables and different scales measuring those variables to consider. It was for this reason that a quality assessment checklist or guide was not possible to procure against which studies could be scrutinised or selected on the basis of methodological soundness. The studies were tabulated according to the datasets used (highlighted in bold; Table 2)

Table 2 . Results of Papers using Confirmatory Factor Analysis and Exploratory Structural Equation Modelling

Title	Time Point	Models Tested				Chosen Model
		Other	Bifactor	1 factor	INT-EXT	
Dunedin Data set						
1- Kreuger et al. (1998)	Two Wave: 18: *n*=930 21: *n*=937			X	X	INT-EXT stable across both waves
2- Caspi et_al. (2013)	Longitudinal *n*= 1,037	X	X	X	X	Bifactor Model: General *p* factor; narrow Factors: INT-EXT (No THD)
National Comorbidity Study (NCS) of Mental Disorders						
3- Krueger (1999)	Cross-sectional 15-54: *n* = 8098		X	X	X	3 Factor: INT-(Anxious-Misery, Fear) and 'EXT'
Netherlands Mental Health Survey and Incidence Study (NEMISIS)						
4- Vollebergh et al. (2001)	Longitudinal: Wave 1: *n* = 7076 Wave 2: *n* = 5618			X	X	Three Factor: INT-(Anxious-Misery, Fear) and 'EXT' -Stability assessed across two time points

Table 2 Continued. Results of Papers using Confirmatory Factor Analysis and Exploratory Structural Equation Modelling

Title	Time Point	Models Tested				Chosen Model
		other	Bifactor	Single Factor	INT-EXT	
Collaborative Psychiatric Epidemiological Surveys (CPES), an integration of three nationally representative multi-stage area probability samples: the National Comorbidity Survey Replication (n=5692), the National Survey of American Life (*n*=6082)						
5- Forbush & Watson (2013)	Cross-sectional *n* =16,233		X		x	20 Models compared: Initial EFA completed to determine 7 factor solution -> followed by Hierarchical CFA , 'INT-EXT' where 'Distress', 'Fear', 'Eating Pathology', 'Dysphoria', and 'Bipolar' are subsumed under 'INT'
National Epidemiological Study of Alcohol and Related Conditions (NESARC)						
6- Lahey *et* al. (2012)	Cross-sectional *n* =23,557	x	x	x	x	Bifactor Model: General *p* factor; narrow Factors: Fear, Distress, EXT
7-Eaton et al. (2013)	Cross-Sectional *n*= 43,093)	*Tested Lahey et al.'s (2012) model to indicate invariance among ethnic groups				
8- Kim&Eaton	Longitudinal **ESEM * Bass Ackwards**	X				Bifactor model: General *p* factor; narrow Factors: Fear, Distress, EXT

Table 2 Continued. Results of Papers using Confirmatory Factor Analysis and Exploratory Structural Equation Modelling

Title	Time- Point	Models Tested			Chosen Model	
		other	Bifactor	1- factor	INT-EXT	
9- Hoertel et al. (2015)	Longitudinal *n=34,653*	x				Bifactor Model: General *p* factor; narrow Factors: INT1- INT II-EXT
10- Keyes et al. (2013)	Cross-sectional *N* = 34 653	X (EFA), Additional 4-factor solution tested with THD	X	X	X	Three factor: INT- (Anxious-Misery, Fear, THD) and 'EXT
The Early Developmental Stages of Psychopathology Study						
11- Wittchen et al. (2009)	Cross Sectional N= 3021					Rejects 3 factor structure.
12- Beeso-Baum et al. (2009)	Cross Sectional N= 3021	X		x	x	Three factor: Anx-Misery, Fear and EXT Omits the Higher order INT Factor from Kreuger (1999).

Table 2 Continued. Results of Papers using Confirmatory Factor Analysis and Exploratory Structural Equation Modelling						
Title		Models Tested				Chosen Model
		other	Bifactor	One factor	INT-EXT	
Virginia Twin Registry,Non-clinical						
13- Khan et al. (2005)	Cross Sectional , *n*= 7,588	X				1 factor interpreted as “Neuroticism”
Midwest Longitudinal Study of Homeless Adolescents (MLSHA).						
14- Yoder et al. (2008**)**	Cross-Sectional *n*= 428	x	x		x	Three factor Solution: INT-EXT-I
British Office for National Statistics Survey of Psychiatric Morbidity						
15- Markon (2010)	Cross Sectional *n*= 8,405	**X**				Four Factor solution : “INT”, “EXT,” “THD”and “Pathological Introversion”
Misc. Database						
16- Wright &Simms (2015)	Cross Sectional *n* = 628	**X ESEM**				Five Factor model: internalizing, disinhibition, psychoticism, antagonism, and detachment.

Two and Three-Factor Models (INT-EXT). Krueger et al. (1998) used the Diagnostic Interview Schedule (DIS; Version III-R; Robins et al., 1989) to estimate the presence of ten common diagnoses: Major depressive episode, dysthymia, generalized anxiety disorder, agoraphobia, social phobia, simple phobia, obsessive–compulsive disorder; marijuana and alcohol dependence in addition to conduct disorder–antisocial personality disorder in a large epidemiological adult sample in Dunedin, New Zealand. They considered the number of factors that underlie these ten disorders and the possibility of a one-factor solution such that they likened to the general intelligence factor, followed by the possibility that the structure of psychopathology in adulthood maybe congruent with the INT-EXT construct conceived for children by Achenabach & Edelbrok (1978.)

A final two-factor INT-EXT model was considered based on the section divisions in the DSM-IV (American Psychiatric Association, 1994) comprising of substance-related disorders, mood disorders, anxiety disorders and antisocial behaviour. Guided by model fit indices (e.g. Chi-square goodness-of-fit statistic (GFI) and the Bayesian Information Criteria (BIC)) they concluded that the two-factor INT-EXT offered the best and most parsimonious account of the correlations found between the measures of disorders.

Kreuger et al. (1998) considered the internalising factor in terms of the writings of Karen Horney (1945), who hypothesised about the existence of a basic anxiety in all individuals that is modulated in various ways. In their paper (Kreuger et al., 1998) they refer to the conceptual works of Haag et al. (1991) who formulated all internalising disorders as comprising of “withdrawal from

the external world", which in Major Depression is mediated by self-referential thought patterns or dwelling on negative thoughts about the self, whereas this may take the form of social anxiety or agrophobobia.

Subsequently, Krueger (1999) sought to replicate the earlier findings in the Dunedin study on the National [Comorbidity Study (NCS) of Mental Disorders that fielded the United States. Using a substantially larger adult sample (*n*= 8,098) with a greater age range (15- 54 years), lifetime prevalence of the ten disorders (major depression, dysthymia, agoraphobia, social phobia, simple phobia, panic disorder, alcohol and drug dependence, and antisocial personality disorder) measured using the Composite International Diagnostic Interview (CIDI; Wittchen, 1994). Three competing models were evaluated including, a one –factor model representing general model of psychopathology, the INT-EXT model, and a four[1]-factor hierarchal variant of the INT-EXT model where next to substance abuse and antisocial disorders that comprised the EXT Factor, The INT factor is split into 'Anxious-Misery' consisting of major depression, dysthymia, and generalized anxiety; along with 'which comprised of the remaining anxiety disorders : social anxiety, simple phobia, agoraphobia and panic Disorders .

The final model four-factor model (a variant of the two factor INT-EXT) organised anxiety disorders, mood disorders, substance abuse disorder and antisocial disorder separately to reflect common clinical parlance of how mental disorders are conceptualised. Although this model was found to be the best model as indicated using goodness-of-fit-indices for the general population, it failed among a small subset of treatment seeking individuals (*n*=

[1] Although Krueger (1999) refers to this model as the '3-factor model' to describe, I have chosen to describe it as a four factor model in this review as it consists of four latent factors, to avoid confusion with models in further studies.

251), as the distinction between the sub factors ‘Anxious-Misery’ and ‘Fear’ could not be retrieved in this subsample.

The three factor model was replicated across further databases such as Netherlands Mental Health and Incidence Study (NEMESIS; Vollebergh et al. 2001). In Nemesis the exact manifest variables also measured using CIDI were used to test the Four Factor model (‘Fear’, ’Anxious-Misery’ under Internalising and Externalising) were tested using the same protocol as Krueger (1999). Data was collected at two intervals and results supported the structural stability of the three factor model during a one-year period was substantial, and the differential stability of the latent dimensions. This represents the most congruent replication attempt across the studies reviewed. However, they did not comment on the theoretical underpinning of the finding or any matter pertaining to shared aeitiology driving the factors. Similarly efforts by Beeso-Baum et al. (2009), using a similar model comparison protocol as Kreuger et al. (1999), found similar results in the Early Developmental Stages of Psychopathology Study (EDSP) sample. However, they also found good indices of fit for the variation of the model where the higher order INT factor was omitted.

Other studies found similar, albeit varying results using CIDI in different samples such as Yoder et al. (2008), who suggested that suicidality forms a distinct latent variable independent from the INT-EXT dichotomy. However, their model used a reduced number of manifest variables, relying only on measures for major depression and post-traumatic stress disorder (PTSD) to account for the INT factor, whereas EXT was indicated by disorders; by lifetime diagnoses of conduct disorder, alcohol abuse, and drug abuse. Some

studies demonstrated that the INT-EXT paradigm remained robust against additions of other variables. Forbush & Watson (2013) used a combination of exploratory and confirmatory factor analytic techniques demonstrated that bipolar disorder and eating pathology are subsumed under INT alongside fear and distress, but did not comment further on any aetiological underpinning for the structure, suggesting that it may be genetic it it's origin. However with the intention to test the stability of the model Wittchens et al. (2009) found that the addition of further manifest variables from the Munich-Composite International Diagnostic Interview (DIA-X/M-CIDI) (Wittchen et al., 1998a; Wittchen & Pfister, 1997) that the structure was not stable as various clinically meaningful patterns with good fit substantially go beyond the original three-factor structure. They concluded that psychopathology could not be reduced to a limited number of factors.

Thought Disorders. Markon (2010) raised a pertinent query, namely, where is the position of psychotic disorders was in the multivariate structure of common mental health disorders? He posited that the symptoms used in earlier studies were far too common to model a structure of psychopathology that extends to other disorders, particularly as the Internalising- Externalising construct cannot adequately account for disorder characterised by thought disorder (e.g. psychosis). Additionally, the ability for the earlier models to generalise to accommodate Axis II disorders was questioned. Markon (2010) used a series of exploratory factor analyses followed by cluster analyses and finally specified a CFA on items from the Clinical Interview Schedule (CIS-R; Lewis Pelosi, 1990; Lewis et al. 1992), which assesses symptoms of anxiety and depression along with the Psychosis Screening Questionnaire (PSQ;

Bebbington and Nyani, 1995) and the SCID-II personality disorder screen (First et al., 1997) to gauge psychosis and personality disorders, respectively. The sample size was considerably large (*n*=8,405 individuals) from the 2000 British Psychiatric Morbidity Survey.

Markon established from EFA that the optimum number of latent factors to extract was 20. These 20 factors were interpreted and considered lower order factors. Exploratory analyses were then used on the estimated inter-factor correlation scores to determine the number of superordinate higher order factors. A four factor superordinate structure was found to be the most optimum. Using CFA, an INT- EXT model was found to hold acceptable fit indices; INT did not bifurcate into anxiety-misery and fear, as suggested by Kreuger (1999). The additional two factors were thought disorder and pathological introversion. Thought disorder comprised of the: hallucination delusion, paranoia, eccentricity, schizoid characteristics, inflexibility and disorganized attachment. Pathological introversion comprised of social anxiety, unassertiveness and dependence. The independence of thought disorder in the sample by used Markon (2010) was not supported by further publications in other studies, such as Forubush & Watson (2013), who located bipolar disorder under INT and Keyes et al. (2013) who also located items of thought disorder under INT in National [US] Epidemiologic Study of Alcohol and Related Conditions (NESARC), a data set of n=16,233.

General Single Factor 'P factor': An Alternative Perspective. A later development in the conceptualisation is the advent of the General Single Factor, proposed by Lahey et al. (2012) that was replicated and dubbed the

‘Psychopathology Factor’ (P factor) by Caspi et al. (2013). As with Eaton et al. (2013 and Lahey et al. (2012) used the NESARC. The researchers used Twelve- month DSM-IV diagnoses (major depression, dysthymia, GAD, social phobia, specific phobia, agrophobia, antisocial personality disorder and alcohol, tobacco, marijuana or other drug dependence). They acknowledged the better fit of the three factor model proposed by Krueger (1999) where symptoms are organized into distress, fears, and externalising over the two factor Internalising-Externalising model. However they noted that the three latent factors in the three factor model remained substantially correlated. Kreuger (1999) had, previously referred to the one-factor model as “-fetched” and likely to give an account of no more than the DSM –IV axis V ‘Global Functioning Score’.

Furthermore, Lahey et al. (2012) fitted a third model, known as a bifactor model. In a bifactor solution, manifest variables load on a single latent variable while also loading on internalising and externalising latent factors. Covariances between the internalising and externalising factors were set at ‘0’ (Gibbon and Hedecker, 1992). Based on Bayesian Index Criterion (BIC) and log-likelihoods indices the general bifactor model fit better than the three factor model.

The general factor in the bifactor model also proved more useful as it related more closely to the externalising factor in the three factor model when compared against validators and indicators of wellbeing. Although the emergence of a general factor may indicate a shared aetiological path towards psychopathology, the researchers were cautious in interpreting their

findings and proposed that it may be due to reporting biases among participants.

The next large sample investigation into the bifactor structure was Caspi et al. (2013) who coined the term, P factor. Using the Dunedin sample and structured they used longitudinal data to model the structure of psychopathology by using CFA to compare the One Factor, bifactor, Two and Three factor structures. Unlike Lahey et al. (2012) who depended on continuous measures, ordinal measures for disorders (major depression, dysthymia, agoraphobia, social phobia, simple phobia, panic disorder, and alcohol, marijuana, tobacco and hard dependence) were used, measured at 18, 21, 26, 32 and 38 years old (note the dissimilarity in the choice of variables from Kruger (1999) in the same sample). Examining the association between the factors against family developmental histories, personality factors, further tested the validity of the structures and neurocognitive correlates.

They set out to compare three models: a general factor model onto which all manifest variables load, a specific model relating all manifest variables to three specific latent variables (Internalising, Externalising and Thought Disorder) and a bifactor. The Three-factor model met the necessary criteria on the fitness indices. The bifactor also showed goodness of fit indices, however contrary to previous suggestions by Markon (2010) Thought Disorder factor was subsumed under the general factor, as it could not form a factor independently of the general factor.

Interestingly, Caspi et al. (2013) noted the variations in the standardised factor loadings of the manifest variables on the latent variables

between the bifactor and the INT-EXT model. For some measures (e.g. conduct disorder) the factor loading on the specific externalising variable was reduced after a general factor was fitted, while the loadings for others remained (e.g. marijuana dependence). They interpreted loadings that differentially increased toward the general factor as indicative of a general psychopathology, rather than specific to a specific externalising style. Whereas, marijuana dependence is more reflective of an externalising style. Caspi et al. (2013) asserted that the central premise of the P factor is inspired by the general factor of human intelligence (or g factor). The g factor is a pinnacle of intelligence research, and one of psychology's most reliably replicated constructs.

Additionally, prior to introducing the general factor the inter-factor correlation between internalising and externalising was positive. After the introduction of the general factor it fell to negative. Chronologically, this is the only case among the reviewed studies to state such a finding, thus far. The general factor psychopathology factor (*p*) was described as a liability to mental health disorder. The positive relationship between internalising and externalising disorders is present in as much as it accounts for *p*, however individuals prone to externalising disorders are less liable to internalising disorders and vice versa. All extracted variables in both models were found to be related to low trait agreeableness, low conscientiousness and high neuroticism. Similar to the findings in Lahey et al. (2012) in finding that factor scores in the internalising- externalising factor predicted a history of childhood maltreatment, however in the bifactor model, the *p* factor accounted for much more of the variance, than the specific factors. The pragmatic utility of the

bifactor solution was also demonstrated in accounting for variance in external criteria such as suicide attempts and alcohol abuse. This finding was replicated by Hoertel et al. (2015), who expanded on Lahey et al.'s (2012) bifactor model in NESARC by incorporating second wave data. Hoerel et al. (2015) found that as a latent measure the general *p* factor mediated psychiatric diagnosis and suicidality. Moreover, the risk of suicide was not uniquely associated with any single disorder, but rather it was mediated by the *p* factor, which they interpreted as the latent liability to psychopathology. Furthermore the researchers found that the structure was invariable to sex.

Exploratory Structural Equation Modelling (ESEM). Two studies reviewed made use exploratory structural equation modelling (ESEM), Wright and Simms (2015) and Eaton & Kim (2015). The aim of the former was to reconcile the existing literature of the meta-structure of common mental health disorders, (e.g. the two factor INT- EXT model, Krueger, 1999); three factor model that incorporates an additional thought disorder dimension (Markon, 2010) with a five factor meta structure meta-structure of personality disorders proposed by Krueger et al. (2012). Wright and Simms (2015) combined the ordinal ratings of the personality inventory for the DSM-5 (PID-5; Krueger et al., 2012) where clinical syndromes were assessed using the sixth edition of the Mini International Neuropsychiatric Inventory (MINI; Sheehan & LeCrubier, 2010) and the structured clinical interview for the DSM-IV-TR Personality Disorders (SCID-II; First et al., 2002) on an adult, clinical sample (*N = 628*). The results yielded a five factor solution: negative affectivity (e.g. emotional labiality, separation insecurity disinhibition (e.g., risk taking, impulsivity), antagonism (e.g., narcissistic PD and histrionic PD) and trait antagonism

(e.g., callousness, manipulativeness), pathological introversion (e.g., avoidant PD, schizoid PD) and detachment (e.g., withdrawal, restricted affectivity), and thought disorder (e.g., psychotic symptoms, schizotypal PD) and psychoticism (e.g., unusual beliefs, perceptual dysregulation. No other study reported similar results.

Eaton & Kim (2015) used data from NESARC to perform a series of exploratory structural equation models (ESEM). Analysis was conducted on 12 common mental disorders from a large, two-wave nationally representative sample, using the Bass-Ackwards method to explore the hierarchical structure of transdiagnostic comorbidity factors. Wave 1 factors were then linked with the bifactor model and with mental disorders at wave 2. Results indicated that common mental disorder comorbidity was structured into an interpretable hierarchy. As with Caspi et al. (2013) and Hoertel et al. (2015), predictive validity analyses prospectively predicting subsequent diagnoses indicated that transdiagnostic factors outperformed disorder-specific variance.

Other Constructs. A further postulate of Kreuger et al. (1998) to understand the INT-EXT structure is that they may be best understood in terms of liabilities in dimensions of normal adult personality and individual differences. The liability factors are thought of as individual differences stemming from genetics. Among the empirical publications in the review, Khan et al. (2004) examined the association between major depression, generalised anxiety disorder (GAD), panic disorder, any phobia, alcohol dependence, drug dependence, anti- social personality disorder and conduct disorder personality traits measured as neuroticism and extraversion as part of the Eysenk Personality Questionnaire (Eysenck et al., 1985; Heath et

al.1992), novelty seeking as part of the Tridemential Personality Questionnaire (TPQ, Cloninger et al., 1991; Heath et al., 1994), using a structural equation model to test the hypothesis that variations in dimensions in personality traits influence caseness of psychopathology and account for comorbidity. Khan et al. (2004) found that Neuroticism, defined as emotional instability and vulnerability to stress, accounted for 26% of the comorbidity among the disorders. Extraversion was found to explain very little of the comorbidity between disorders in the sample; whereas novelty seeking explained the largest proportion of comorbidity between externalising disorders (11.9%). Khan et al. (2004) interpreted these as evidence for a shared genetic variance influencing all disorders as conceptualised in 'Neuroticism'. Although the researchers acknowledged that the cross-sectional nature of their study prevents any assertion direction of causality, the associations are best understood as high Neuroticism representing a predisposition or liability towards the manifestation of symptoms, much in line with Caspi et al. (2013)'s partial conceptualisation of the *p* factor.

Discussion

The aim of this review was to examine publications that investigated the general structure of psychopathology in adult populations and to extract any conclusions about the processes underlying any latent structures. The result was a narrative review of latent modelling methodology. Across the literature reviewed there is a marked chronological distinction between the use of EFA and CFA. This distinction may reflect a shift in research practices and culture. However, it is important to note that nearly all the studies that made exclusive use EFA resulted in conclusions around the general structure

of psychopathology bespoke to their own studies, none of which were replicated thereafter.

Kreuger (1998)'s seminal paper presented a break from that research tradition of depending on small, non-representative samples to the use of large databases that are often nationally representative. The seminal work also introduced the INT-EXT paradigm and extended it to psychpathology in adults. Research into the multivariate structure of common mental health processes revealed that, in general it is useful to make the distinction between the constructs of Internalising Disorders that incorporate symptoms of anxiety and low mood and Externalising disorders that consist of drug and alcohol dependence and antisocial personality traits, in as far as they describe comorbid features. However, across the papers reviewed, descriptions of the all researchers were reticent to interpret the liability factors driving the higher-order construct. Furthermore, studies that sought to test for its capacity to incorporate other constructs, most notably, Thought Disorder (e.g. Markon, 2010, Caspi et al.) yielded equivocal conclusions. The results indicate that the INT-EXT construct remained the dominant paradigm until the concept of the general factor of psychopathology, or *p* factor (Lahey et al., 2012) modelled in bifactor form.

Arguably, the advantage of flexibility in latent modelling brings with it many difficulties when comparing two or many sets of works. Primarily, factorial structures are unique unto them and reflect the relationship between the manifest variables inputted. For instance, among the papers reviewed Yoder et al. (2008) concluded that suicidality represents a factor unto itself, distinct from the INT-EXT construct. Although they had used similar measures

as other papers e.g. the CIDI as with Vollbergh et al. (2001), they did not use the full range of manifest variables nor did they follow a particular model comparison protocol that had led other researchers to similar conclusions. The topic of suicidality was also addressed by Hoertel et al. (2015), who treated it as a dependent variable against a bifactor multivariate model of psychopathology.

In response to the original question, to date, what has latent modelling revealed about the general structure of psychopathology in adults and the processes that underlie it? The answer may be that the multivariate model of psychopathology in the broadest scope has demonstrated the validity of the INT-EXT paradigm and potentially the possibility of a superordinate general factor. The *p* factor is an interesting concept, as it accounts for the remaining correlation between INT-EXT. Currently, it poses further more questions than answers. It has been posited as a yet to be identified driving factor driving liability to psychopathology, presumably rooted in temperament and therefore may bear a genetic loading. An alternative, arguably more parsimonious suggestion, may be that it simply represents subjective distress and is therefore little more than a statistical artefact. As yet, In light of the impotence of researchers reviewed to allocate or comment on aetiology, it seems that these models serve at best, as descriptive tools that helps us re-think 'the problem of comorbidity' and lead our focus, when conducted at the broadest level. It may be best to reiterate from Kreuger (1999), that it is possibly too "farfetched" to expect for a global, functional, aetiological driver to account for all aspects adult psychopathology or even two or three.

With regards to RDoC, the findings outline that at the level of psychometric analyses, consensus indicates that the common symptoms of psychopathology are best conceptualised and described along the INT-EXT spectra. The current aggregation of finding directs any future investigations further down the research domain matrix, into the functional processes and systems, to be posited and conceptualised within the respective domains. For instance, a comprehensive research endeavour into the aetiopathogenesis of depression may require, in part, an account of anxiety disorders. Furthermore, in the interest of establishing a descriptively accurate and functionally useful diagnostic system, the characteristics of discrete diagnoses of mental disorders (as they are currently known) must be outlined, in addition to the aetiological paths that link them. This is particularly pertinent when considering liability models that postulate shared liability factors among discrete symptoms. It is important to note that as far as clinicians who make use of diagnostic systems are concerned, it would be premature to abrogate the current classification system. However, it is crucial to maintain that certain comorbidities of discrete mental health disorders are expected to occur within the respective INT-EXT spectra.

Appraisal and Future Directions

An advantage of the use of latent modelling for comorbidity is that it allows exploration into unmeasured concepts and raises further lines of questioning. However, Based on the papers reviewed, there has been very little discussed or suggested in terms of aetiology or pathogenesis particularly with regards to the functional processes of psychopathology as outlined by

RDoC. Overall, assumptions or postulates are made or rather inferred that the liability factors are rooted in individual characterological differences such personality.

The original review of the literature for the general structure of psychopathology yielded a sizeable number of sources and references, many of which are difficult to parse through and organise as various researchers used and followed different research strategies, albeit within the same family of CFA strategies. Difficult decisions had to be made to the exclusion criteria to allow for a meaningful conceptualisation of the discussions and efforts to understand the general structure of psychopathology. Comparative reviews of CFA data were challenging to interpret, as in confirmatory analyses; researchers must have a firm a priori sense, based on evidence and theory: the number of factors that exist in the data, and the indicators or manifest variables required to specify the factors. There is a requirement to pre-specify all aspects of the model; this raises the opportunity to lead into circular arguments. Furthermore decisions to reduce and narrow the scope of the search to include only adults comes at the cost of removing investigations in databases that made use of longitudinal data. As mentioned earlier, longitudinal data is essential to elaborate and test for specific functional theories for the structure of psychopathology in adults that go beyond descriptive accounts for the structure of symptom comorbidity. Future directions will benefit from narrowing the scope of investigations to understanding the relationships between fewer select sets of mental health disorders within the broader factors outlined in this review and making use of

longitudinal data across the life-span, to determine the functional interaction between them.

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Part 2: Empirical Paper

Modelling Psychopathology: The structure of common mental health disorders in a mixed sample of patient and control participants

Abstract

Aims: The aim of this investigation is to explore the latent structure of common mental health symptoms in a mixed control and patient sample, where the patient sample consists of treatment seeking individuals with a known diagnosis of a personality disorder. Specifically, a bifactor solution defining a general factor of psychopathology known as the *p* factor is explored and tested.

Method: Data was collected as part of the Probing Social Exchanges Study. Initial Confirmatory factor analysis was conducted using items of the Brief Symptom Inventory (BSI). Further exploratory analysis was required to allocate item level manifest variables to latent factors. Confirmatory techniques were then used to define the factors were conducted. The factors were inputted into a regression analysis to predict caseness between the control and patient group, determining the utility of the selected model.

Results: A bifactor solution proved to be the optimum fit for the data. It was defined by a general *p* factor with three narrow factors (Internalising-Depressive, Anxious-Somatic and Externalising. Regression analysis indicated that the *p* factor statistically predicted caseness over the narrow factors. The findings were found to be consistent with previously defined transdiagnostic models of psychopathology.

Conclusions: Despite the limitation around the corss-sectional nature of the investigation, the findings provide evidence for the transdiagnostic conceptualization of common mental health presentations in adults.

Modelling Psychopathology: The structure of common mental health disorders in a mixed sample of patient and control participants

Introduction

The DSM-IV has stated that that “there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders” (2, p. xxxi, American Psychiatric Association). Since, further reviews of the seminal health mental health classification system, currently in its fifth edition (DSM-5 ; American Psychiatric Association, 2013), have acknowledged the overlap between mental health disorders. They take into account gradations of severity in psychopathology and the overlap between the disorders by encouraging dimensional aspects of disorders alongside categorical diagnoses (Reiger et al., 2013). The shift in the DSM towards a transdiagnostic, continual understanding of the meta-structure of psychopathology is the culmination of empirical studies. Clinicians have long recognised a similar pattern of positive correlations in the presentation of psychopathology symptoms in clinical populations. This positive manifold in the nosology of mental health is a more commonly recognized as ‘comorbidity’, or the presence of two or more diagnosable disorders occurring in an individual at certain time (Cummings, 2014). Although the DSM-5 continues to list categories of mental health disorders, there is the realisation that the symptoms that occur in one disorder may occur in other disorders. Rodriquez- Seijas et al. (2015) address and summarise the weaknesses of the traditional classification systems whereby an individual’s clinical presentation requires meeting a certain number of criteria to pass the threshold for a particular disorder.

In the current system each criterion tallies equally towards differential diagnoses and the final diagnosis remains dichotomous, present or absent. An advantage of the categorical diagnostic system is that it favours high rates of diagnostic reliability and facilitates professional and research communication. However, this comes at the cost of loss in construct validity. In an effort to resolve the 'problem of comorbidity', Krueger and Markon (2006a) reviewed the conceptual literature on comorbidity that demonstrate that comorbidity among mental health disorders can be accounted for by quantitatively modelling correlations among clinical presentations. They maintain that psychopathology is best-understood using empirically based models that integrate behaviour, genetics, personality and individual differences research, and quantitative psychology (Krueger & Markon, 2006b).

One of the first studies to empirically examine a general structure of psychopathology by mapping correlations between discrete diagnoses was carried out by Krueger (1998). This was done using structural equation models (SEM) to generate latent factors among discretely defined symptoms to account for the comorbidity between indicators of 11 mental health disorders in a large adult sample (*n* =23,557). Krueger's findings indicated that most common diagnostic entities within adult mental health could be reduced and described along two factors 'Externalising' and 'Internalising'.

The Externalising factor consisted of antisocial personality disorder And drug and alcohol dependence in contradistinction to Internalising Disorders that involve a general "withdrawal from the external world… into the negative, self-referential thought patterns into depression…or away from the

world" entirely, as in anxiety (Krueger, 1998). Further investigations refined the internalising spectrum by dividing it into two further factors, 'Fear' and 'Distress', accounted for a better fit of the model. Fear consisted of Social Phobia, Simple Phobia, agrophobia and panic disorder; whereas Distress was a factor that defined major depressive episode, dysthymia and general anxiety disorder (Kreuger, 1999).

Despite achieving a considerably adequate fit, there remained considerable comorbidity between the Internalising and Externalising Factors (inter-factor correlation r = .51). This further beckoned the query for the presence of a higher superordinate single factor. Initially proposed and defined by Leahy et al. (2012) and further elaborated by Caspi et al. (2013), the *p* factor is a latent representation of the shared variance between all common mental health disorders. Within a bifactor structure every measurable manifest item representing discrete measure of symptoms or a diagnosis loads on two factors. The first is a 'general' latent factor that is common to all items, and the other is a specific or narrow factor common to a subset items that are related to one another over the relation accounted for by the general factor. In most cases the general factor is orthogonal to the specific factors and by convention the specific factors are usually set as orthogonal to one another. These specifications allow for the common variance amongst a set of items to be partitioned to what is common to all manifest items and that which is specified to the specific domains.

Using the Dunedin Multidisciplinary Health and Development Study, a longitudinal investigation of health and behaviour of a whole birth cohort,

Caspi et al. (2013) championed a bifactor model as the best representation for the a general structure of psychopathology in an adult population (N = 1,037, 52% Male)., based on accepted indices of model fit . Through a series of Multi-Trait Multi-Method models, the investigators used longitudinal data from 12 time points and constructed the integrated model Using Confirmatory Factor Analytic (CFA) techniques. As such they were able to construct the integrated model that tracked the relationship between 11 disorder/ symptoms: dependences on cannabis, hard drugs, and tobacco as well as conduct disorder; they are routinely identified as 'Externalising'. Major depression, generalised anxiety disorder, phobias, obsessive compulsive disorder, mania as well as positive and negative symptoms of Schizophrenia load on either an 'Internalising' and 'Externalising' latent factor. The presence of a 'Though Disorder' as a separate factor as espoused by other researchers (e.g. Markon et al., 2010) domain factor was also considered, but was later rejected on the basis of illness of fit. In the selected best fitting model, the general factor of psychopathology, *p,* accounted for the variance among all diagnoses to a certain extent. The remaining variance is accounted for by narrower latent variables 'Internalising' and 'Externalising'; where 'Internalising' represents symptoms of depression and anxiety and 'Externalising' liability to antisocial and substance abuse disorders.

The researchers interpreted the superordinate structure to suggest that scoring highly on the general factor is an indication that an individual possesses an increased propensity or liability (Kreuger & Makron, 2006b) to developing any and all forms of psychopathology. Statistically, it proved useful in predicting negative life impairment, worse developmental histories, and

more compromised early- life brain function above and beyond the narrow factors. They also found that adults who had scored high on each of the levels, indicate that maltreatment is associated with greater 'General Psychopathology' but not with any specific manifestation type, indicating a possible aetiological link.

The central construct of the *p* factor is inspired by the general factor of human intelligence, *g*. The *g* factor has dominated the cognitive sciences for little more than a century to the present day. Many studies have replicated the single factor, yet it remains one of the most contentious topics in Psychology and this is partly due to its nature as a latent construct. The architecture of intelligence as it maps on the brain and the neural processes driving intelligence is yet to be discovered or fully articulated. Despite its opaqueness, the *g* factor remains one of the most reliable and useful measures in the cognitive sciences and individual differences. General intelligence is ubiquitously the strongest predictor achievement in academic and professional settings (Kuncel et al., 2001; Kuncel et al., 2004; Deary et al., 2007). It is essentially a summary of the positive correlation among tasks of cognitive ability. This has inspired and guided further research to understanding mental ability (Deary,I.J., 2012).

Both, Lahey et al. (2012) and Capsi et al. (2013) interpret their findings as evidence in support of the hypothesis that prevalent forms of psychopathology have important common and differential features. In the grand scheme of improving Psychiatric nosology for clinicians by providing one that solves the problem of comorbidity, as liability models allow for multiple symptoms to be expressed as manifestations from a single driving

factor. They view their work as the initial step towards further research. This is chiefly achieved by determining if the shared variance is due to shared elements of aetiology and neurobiological mechanisms. They postulate that the existence of common trans-diagnostic features across the various forms of psychopathology may have important implications for understanding the nature and aetiology of psychopathology, and guiding the process thereof. With regards to how disorders commonly associated with a 'Thought Disorder' fit within the general structure, and the models utility within a clinical setting (Caspi et al., 2013) hypothesis is that as a dimension of severity, *p* carries thought disorder symptoms at its 'pinnacle', and any individual who carries a strong 'General Psychopathology' liability would experience psychotic thought processes, if their disorder is severe enough, irrespective of the presenting diagnosis.

What does the *P* Factor Mean?

Scepticism towards the *P* factor is largely based on the premise that it may represent no more than a statistical artefact. The argument goes further against the methodology by detractors from the general use of Latent Models to understand Psychopathology, most notably by proponents of *Network Analysis* (e.g. Borsboom, 2013). They argue that from an epistemological perspective, a Latent Factor presumes common cause for various manifest measurements. Contentions specific to bifactor Models, Bonifay et al. (2017) raise several concerns. The first reifies concerns around presumptions that a *Positive Manifold* is causal. The second is that bifactor models have a tendency to outperform other models because they capture unwanted 'random patterns' and is therefore nothing more than a statistical artefact, or

at best an epiphenomenon. Finally, that the models are often structurally described in isolation, with no links to any meaningful criteria (e.g. genetic or neuropsychological substrates).

However, investigations to explain the statistical summary provided by the *p* factor, as a cogent construct include Murray et al. (2016) who examined two competing explanations. The first is the presence of a common underlying cause influencing the cascade of various symptoms of psychopathology at any given point in time and predicting future occurrences. The second explanation is that the *P* factor is best accounted for by the process of *Dynamic Mutualism* where discrete symptoms at any point raise the possibility of developing further (dys) functioning. A *Dynamic Mutualism* theory of the positive manifold asserts that the correlation is an epiphenomenon, which emerges progressively during development as a consequence of interactions among initially uncorrelated factors. Through the use of longitudinal data they found that *p* factor scores within individuals did not vary considerably over time. This finding departs from what would be expected in a *Dynamic Mutualism Model* and suggests that *p* factor is a stable construct, that represents a measure for liability to develop symptoms of Psychopathology that become more specified with time. Another study to make use of longitudinal data with children, in support of the *p* factor and bifactor models as cogent and useful constructs, found that *p* factor at an earlier time point predicted future functioning in academic attainment, as well mental health (Patalay et al., 2015).

Clinical Samples

Earlier latent models of a general structure for psychopathology were

conducted using large community data sets that have the advantage of high statistical power and generalizable validity. Naturally, investigations on pathology have shifted to the clinical settings, to include more varied and specified manifest variables pertinent to clinicians. Results have indicated that the general factor and in particular, bifactor models are useful constructs (Subica et al., 2015; Brodbeck et al., 2014). One notable investigation in the *p* factor was also conducted in a mixed control and clinical sample, with clinical participants diagnoses of Personality Disorders (PD) (Gibbon, 2017). The study found evidence for a bifactor solution better fitting than a single factor solution and other solutions using only narrow domain constructs. A final bifactor solution was selected where the narrow domain constructs were specified as 'Antisocial' (analogous to Externalising), 'Internalising' and a third factor known as 'Borderline'. The study made use of various measures including the Broad Symptom Inventory (BSI; Derogatis, 1993) and the Personality Disorder Inventory- Borderline Features (PDI-BOR; Moray, 1991), among others. Similar to Caspi et al., (2013) a 'Thought Disorder' factor was rejected. In fact, despite several replications that demonstrate a General Factor, few studies have demonstrated congruencies in the narrow factors or domains modelled. For instance, Caspi et al. (2013) who identified EXT-INT domains in distinction to Lahey et al. (2012), who built on Kreuger's (1999) Three factor model.

Current Study

In the current climate, science and the study of Psychology in particular is in the grips of the 'Replication Crisis', where the lack of systematic

methodological research procedures among researchers and research proposals lead to disparate results and conclusions. Simons et al. (2011) argue that the most costly error is the *false positive*, or the incorrect rejection of the null hypothesis. They coin the term *Research Degrees of Freedom* to denote the extent of decisions and liberties made by researchers to find a 'positive' result. Arguably CFA is a modelling tool that is susceptible to *False Positives*, particularly as the selection of models, input variables, constraints (or lack thereof) and the use model fit indices involve considerable subjective choices and allowances in making such decisions. Despite various studies successfully locating a general structure of psychopathology within the confines of a bifactor model and qualms remain.

Additionally, across the growing literature on the *p* factor there remains very little consistency in the modelling specifications, use of manifest variables and the model fitness indices used to assess and champion models. For instance, Lahey et al. (2012) made use of DSM identified symptoms from The Alcohol Use Disorder and Associated Disabilities Interview Schedule–*DSM-IV* Version (AUDADIS-IV) (Grant et al., 2003)), whereas Caspi et al. used various measures to account for manifest variables. Other concerns may be raised around the structures and models compared. For instance, Lahey et al. (2012) only compared two models and additional structures that were present in some studies were not present in others, as Caspi et al. (2013) were able to refute the sufficiency of a single factor general factor model by directly comparing the model to a bifactor structure, a step absent in Lahey et al. (2012).

The aim of this project is twofold. The first is test the reliability of the

bifactor structure across common indicators of psychopathology in the same database as Gibbons (2017), that consists of a mixed sample of individuals diagnosed with diagnoses of PD and controls and to build on some of the methodological limitations of Gibbons (2017). Gibbons stated, as part of their limitations is the absence of exploratory investigations to lead the specification of manifest measures of symptoms to latent variables. As such, we aim to incorporate data-driven techniques in addition to *apriori* model building and hypothesis testing methodology. The second aim is to test the predictive validity and usefulness of a general factor in determining caseness or rather the diagnostic presence of a personality disorder, over and above specific factors. Determining the extent to which the general factor predicts caseness between PD and non-PD participants as an external criteria.

This investigation departs from that of Gibbon (2017) by a using 'finer level' measures of symptoms represented in item level indicators of the BSI. Additionally, further indicators of model fitness will be used to apply stricter criteria for all models tested. Gibbons (2017) used several measures from a broad battery in a specific dataset that were not present or plausibly analogous to other measures in another data set, as in her 'Borderline' construct. The risk in this approach is that the addition of any manifest variable may constitute an additional latent factor that may be specific to manifest variables inputted into the solution, but not generalizable to most common symptoms. In light of the concerns specified by Bonifay et al. (2017) regarding the bias towards 'over fitting', championing a bifactor solution on the basis of statistical fit indices. This may represent an example of a *False Positive*.

Models Tested

Confirmatory factor analyses was chosen to test the hypotheses previously proposed by Caspi et al. (2013. Analysis was conducted using the Lavaan Package on R (Rosseel, 2012), and individual items of the BSI were used. In the interest of reliability, item analysis level manifest variables representing common symptoms of general psychopathology were fitted to closely follow the procedure used by Caspi (et al., 2013). Specifically, four models nested within each other will be tested against indices of goodness of fit: (1) A General model consisting of a single latent factor specified by all manifest measures (2) A Three Factor EXT-INT- Thought Disorder (THD) factor (3) A bifactor model consisting of the aforementioned three factor Sslution as narrow domains, in addition to a General Factor. (4) A modified bifactor solution championed by Caspi et al. (2013), where a THD was removed, and specified items loaded directly on the General factor (See Figure 1).

Initial Hypotheses

(1) The bifactor structure will emerge and will have a better fit than the three factor model that categorizes common psychological symptoms into ‘ Internalising’, ‘Externalising’ and ‘Thought Disorder.

(2) The general Factor *p* representing a transdiagnostic entity will predict caseness better than the sub-factors.

Recruitment

Data was acquired from an on-going imagery project, ‘Probing Social Exchanges- a Computational Neuroscience approach to the Understanding of

Borderline and Anti-Social Personality Disorder'. Control participants were recruited from a large number of clinical services for personality disorders and probation services in the Greater London area. Participation was open to participants who had recently undergone assessment, were on the waiting list or were in early stages of treatment. The patient population were service users in mental health services in advanced stages of treatment were excluded to avoid confounding effects of treatment. Recruitment has been agreed with the London Probation Service (LPS), specifically for patients with ASPD. Adolescent and adult control participants were matched in IQ, age, gender, and socioeconomic status. For inclusion in the study all participants were required to be fluent in spoken and written English. Exclusion criteria included current or past history of neurological disorders or trauma including, epilepsy, head injury, and loss of consciousness. Control Participants were excluded on the basis of a score greater than '3' on the Standardised Assessment of Personality – Abbreviated Scale (Moran, 2003).

Participants. All participants took part over a two-day period and were asked to complete a battery of psychometric assessment tools. A total of 491 individuals participated in the study. Most participants were female *n* = 321 (64%). Two individuals identified as transgender were included as part of their identified gender, and two participants in the PD group preferred not to disclose their gender. Ages ranged from 18 to 65 years, (*M* = 31.4, SD = 10.7). The total number of control participants (non-PD) was *n* = 168. Ninety nine participants were female (58%). Ages in the control group ranged from 18 to 54 years (*M* = 29.9, *SD* =10.9). Most participants belonged to the PD

group *n* = 323 (67% of the all participants). The majority of PD participants were female *n* = 191 (65 %). Ages in the PD sample ranged between 18 to 65 years (*M* = 32.2, *SD* = 10.5). All participants completed the 53 items of the BSI. The nine sub-scales were computed using the instructions in the BSI Administration, Scoring and Procedures Manual (Third Edition; Derogates, 1993; See Appendix A for Detailed Descriptive statistics).

Measures. The BSI was the central measure in constructing the higher order factor model. It is an instrument that provides pattern reported data to help clinical decision-making at intake, and during the course of treatment in multiple settings. Its strength is in its applicability across the continuum from adult non-patient to adult patients, and from inpatient to outpatient settings as it has established norms for the aforementioned categories. The scale is comprised of 53 items with a five-point Likert rating scale. The nine sub-scales that constitute the BSI are: Somatization, Obsessive Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation and Psychoticism. The BSI offers a wide variety of common mental health disorders .The factorial structure has been studied extensively for the purpose of scale validation. It has been translated and adapted to several languages and client groups through the use of various modelling techniques including Spanish (Pereda et al., 2007), where a Principle Component Analysis (PCA) showed adequate fit for a nine factor solution, Chinese (Wang et al., 2013), in which a single factor, identified as ‘ Psychological Distress’ was fitted using CFA. Other studies had used the scale to fit bifactor solutions (Thomas, 2012; Brodbeck et al., 2014).

Models specifications. An initial Three Factor Model (Model A) was run consisting of the all the items of Somatization (SOM), Obsessive-Compulsive (O-C), Interpersonal Sensitivity (I-S), Depression (DEP), Anxiety (ANX), Phobic Anxiety (PHOB) dimensions as manifest variables loaded on an Internalising (INT) latent factor. Items for the Hostility (HOS) dimension were fitted as indicators that loaded on the Externalising (EXT) Factor. The remaining two dimensions Paranoid (PAR) and Psychoticism (PSY) were fitted to a Thought Disorder Factor (THD). In Model B, A bifactor model was tested. A general factor was introduced to the Three Factor model, extending loadings to all the manifest variables. In the third model (Model C) all manifest variables were set to load on a single general factor (See Figure 1 for diagrams for initial CFA models). In the Fourth Model (Model D), known as the modified bifactor model, the items for PAR and PSY load exclusively on the general factor and the THD is removed. This allows us to test whether symptoms commonly associated 'as thought' disorders may be subsumed by the general factor, or whether they should be identified, in their own right as a separate factor.

Results

Initial Confirmatory Factor Analysis

Following guidelines from Kline (2010) and using cut off values for fit model statistics set by Hu & Bentler, (1999) Chi-squared test, the Root mean square error of approximation (RMSEA), the comparative fit index (CFI), and the standardised root mean square residual (SRMR). Were requested. All three models fell short of acceptable measures of fit indices except on SRMER ranged between .049 to .165; SRMR is greater than 0.08 'acceptable

fit'. Otherwise, χ2 were all-significant (P <. 01). CFI ranged between .821 - .863 indicating 'not a good fit'. RMSEA ranged between .077 to .081; above the required cut off .06 indicating 'not a good fit' (See Table1.)

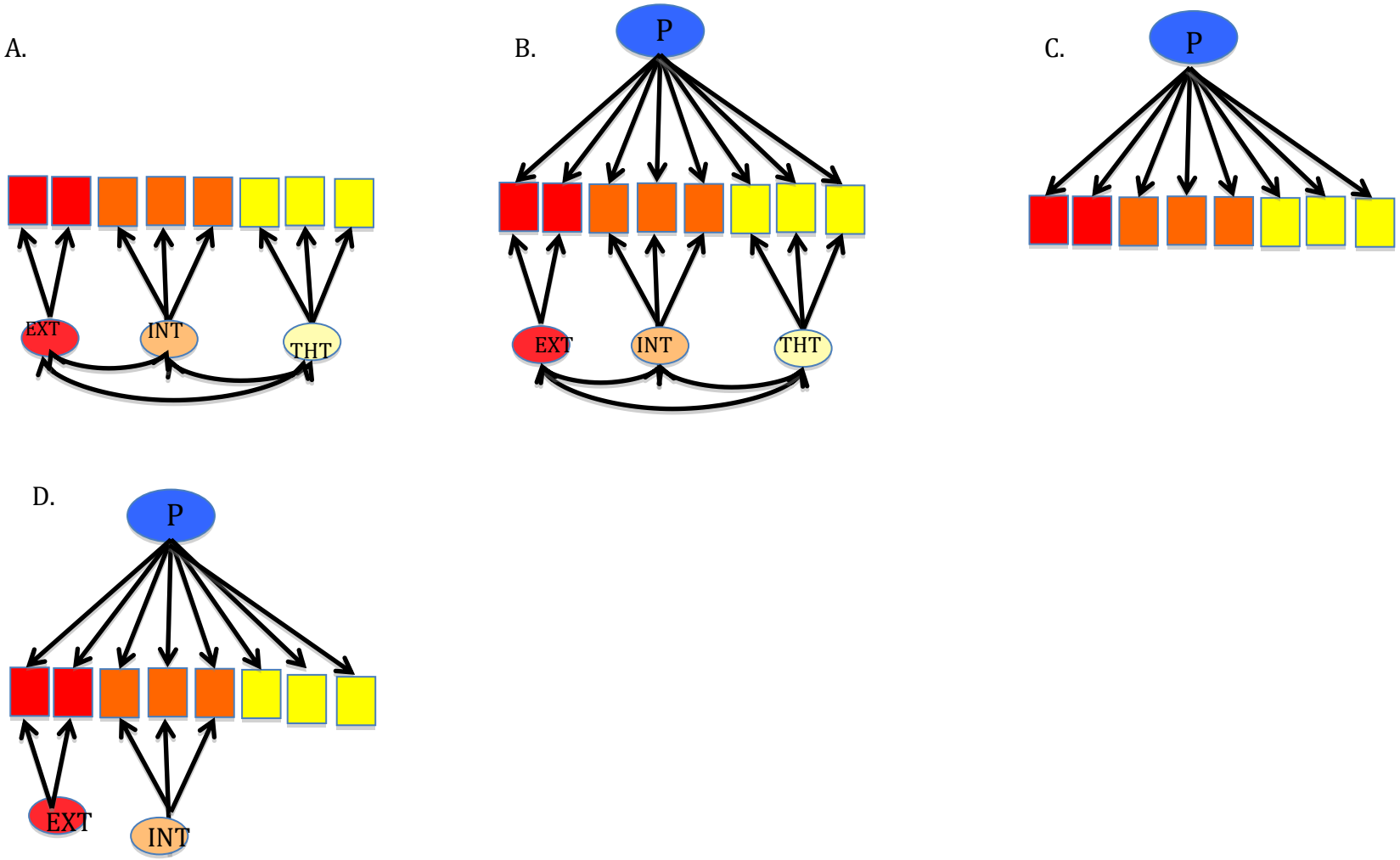
As models A, B and C failed to reach acceptable cut-off criteria for model fit indices, they fail to represent the true structure of the data. As with Caspi et al. (2013) a fourth model D was specified, to test whether the misspecification was the inclusion the third Thought Disorder factor, which they had found was best subsumed by the 'General Factor'. In Model D items pertaining to Thought Disorder were specified directly to the General factor. However, as with the previous models, apart from SRMR = .043, all other fit indices were did not indicate goodness of fit. A potential reason for the models' ill-fitting was considered due to the specification of latent variables and the allocation of the manifest variables on to them. To remedy this problem, a data driven exploratory approach was administered to understand how the latent variables best form within the dataset's correlation matrix.

Table 1. Model Fit Statistics for Initial CFA

Model Fit Statistics	Model A	Model B	Model C	Model D
Chi-Square Statistics (ML)	3846.103	3797.497	5036.113	3976.914
Significance P	<0.01	<0.01	<0.01	<0.01
Degrees of freedom	1031	1079	1127	1087
Comparative Fit Index	.846	.863	.803	0.854
SRMR	0.05*	.043*	.051*	0.043*
RMSEA	0.075	.072	.084	0.074

* indices of 'Good Fit'

Figure 1. Diagram fit for Initial CFA



* In the interest of graphical clarity, the number of manifest variables has been abbreviated.

Exploratory Factor Analysis (EFA)

An Exploratory Factor Analysis (EFA) was used on all 53 items in the entire pooled sample to determine the factor structure. The 'Psych' (Revelle, 2016) was used on R studio to complete the analysis (Rstudio Team, 2015). The Keiser- Meyer- Olkin measure was used to verify the sampling adequacy for the analysis, KMO = .98, 'superb' (Kaiser, 1974). Additionally all KMO values for the individual items were > .94. Bartlett's Test of Sphericity, $\chi 2$ (1378) = 3742, $p<0.001$, showed that correlation between items were sufficiently large for Factor Analysis.

An initial analysis was run to obtain eigenvalues for each component of the data. Using Eigen decomposition of the correlation matrix, results of Horn's Parallel Analysis for component retention after 1590 iterations resulted in six components with Adjusted Eigenvalues that were greater than one: Component 1= 27.8, Component 2= 1.96, Component 3 = 1.83, Component 4: 1.30, Component 5 = 1.15, followed by component 6 = 1.11 . A scree plot was run and proved ambiguous as it hinted at a third factor upon subjective observation. (Figure 2).

Figure 2. scree plot

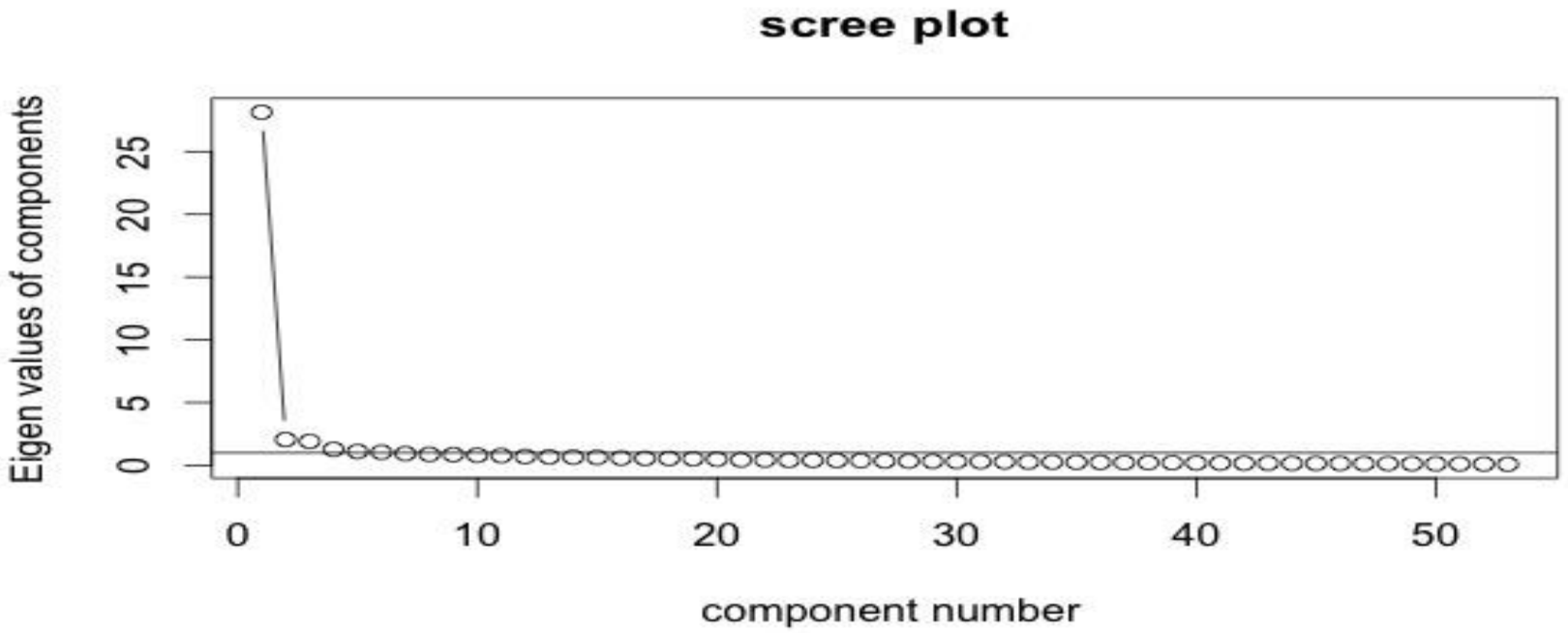


Table 3. Item number, descriptions and standardized item loadings on factors including Communality and Uniqueness scores for the Three Factor EFA Oblimin solution.

Item	Description	Factor 1	Factor 2	Factor 3	H2	U2
35	Feeling hopeless about the future	.96			.76	.25
50	Feeling of worthlessness	.91			.82	.18
17	Feeling blue	.86			.77	.23
16	Feeling lonely	.84			.67	.33
18	Feeling no interest in things	.78			.68	.32
52	Feelings of guilt	.71			.55	.45
14	Feeling lonely even when you are with people	.70			.70	.30
22	Feeling inferior to others	.68			.57	.43
42	Feeling very self-conscious with others	.66			.67	.33
44	Never Feeling close to another Person	.62			.51	.49
36	Trouble Concentrating	.58			.62	.38
9	Thoughts of Ending Life	.57			.55	.45
20	Feelings hurt easily	.54			.67	.33
39	Thoughts of death	.53			.64	.36
15	Can’t get things done	.52			.54	.46
27	Difficulty with decision	.48	.33		.60	.40
21	Feeling people are unfriendly	.45	.31		.61	.39
38	Feeling Tense	.42			.66	.34
34	“I should be punished”	.42			.36	.64
51	“People will take advantage of me”	.34			.56	.44
29	Trouble catching breath		.80		.54	.47
28	Afraid to travel		.74		.58	.42
2	Faintness or dizziness		.71		.43	.57
12	Suddenly scared for no reason		.71		.71	.29
45	Spells of terror or panic		.70		.73	.27
31	Avoid certain places		.69		.63	.37
30	Hot or Cold spells		.66		.45	.55
8	Afraid of open spaces		.65		.52	.48
33	Numbness in body		.65		.38	.62

Table 3 Continued **Item number, descriptions and standardized item loadings on factors including Communality and Uniqueness scores for the Three Factor EFA Oblimin solution.**						
26	Having to Check and double check		.55		.53	.47
23	Nausea		.54		.41	.59
32	Mind gone blank		.54		.55	.45
19	Feeling fearful	.45	.52		.72	.28
37	Feeling weak in parts of body		.51		.47	.53
1	Nervousness	.31	.51		.59	.49
7	Pain in chest		.48		.33	.67
43	Uneasy in crowds	.35	.48		.60	.40
3	Someone in control of your thoughts		.48		.28	.72
24	Feeling of being watched		.47		.59	.41
49	Feeling restless		.43	.33	.53	.47
5	Trouble remembering		.43		.39	.61
11	Poor appetite		.39		.33	.67
13	Temper outbursts that you cannot control			.78	.70	.30
41	Having urges to break or smash things			.77	.62	.38
46	Getting into frequent arguments			.73	.62	.38
40	Having urges to beat injure, or harm someone			.69	.44	.56
6	Feeling easily annoyed or irritated			.51	.54	.46
4	Feel others are to blame for troubles			.44	.37	.63
10	Most people cannot be trusted			.35	.61	.39

Factor Analysis using an Oblimin rotation was requested to allow for the factors to correlate and to avoid misleading solutions (Brown, 2006, P.31). The extraction of three factors yielded a fit based upon off diagonal values of 99% explained 56% of the variance, where the initial factor contributed to 24% of the variance followed by 22% and 10% for the second and third, respectively. A three-factor extraction gave a 100% fit based upon off diagonal values. All communalities were less than '1' for all of the items.

Table 3 shows the item loadings on the first general factor (Factor 1) Followed by loadings on the two other factors (Factor 1 and Factor 2) as well as communalities (H2) and Uniqueness (U2) scores for every item. Factor loadings less than .35 have been excluded. Factor 1 consisted of almost all the items for the Depression dimension, in addition to two items from the Psychoticism dimension: 'Feeling lonely even when you are with people', and the 'Idea that something is wrong with your mind''. Two more items from the Interpersonal Sensitivity dimension also loaded on the general factor: 'Feeling inferior to others' and 'Your feelings being easily hurt'. The second factor (Factor 2) consisted of items belonging to two the Somatization dimension: 'Trouble getting your breath' and 'Faintness or dizziness'; in addition to items from both the Anxiety and Phobic Anxiety dimensions: 'Suddenly Scared for no Reason', 'Spells of terror or panic', 'Feeling afraid to travel on buses, subways, or trains'. The third Factor consisted of items pertaining to the Hostility dimension. Apart from the items of the BSI Hostility dimension loading on an Externalising Factor, the previous allocation of items into 'Internalising' and 'Thought Disorder' dimensions had been done arbitrarily. A more accurate representation of the three-factor solution would be to interpret

them as ‘Internalising-Depressive’, ‘Anxious-somatic’ and Externalising. Inter-factor correlation showed that ‘Internalising-Depressive’ correlated substantially with ‘Anxious-Somatic’, *r* = .79 and ‘Externalising’ at *r* = .63. While ‘Anxious Somatic’ and ‘Externalising’ correlated at *r* = .63 (See Table 4)

Table 4. Inter-Factor Correlation for Three Factor Exploratory Solution with ‘Oblimin Rotation’

	Factor 1 ‘Internalising-Depressive’	Factor 2 ‘Anxious- Somatic’	Factor 3 ‘Externalising’
Factor 1 ‘Internalising-Depressive’	1.0		
Factor 2 ‘Anxious- Somatic’	.79	1.0	
Factor 3 ‘Externalising’	.63	.63	1.0

Model Refitting

Interestingly, the three Factor Solution produced by the EFA showed a similar structure to one found by Krueger (1999) and further examined by Kim & Eaton (2015), where a three Hierarchal Factor Solution was selected consisting of: Externalising and Internalising, where Internalising further splits into ‘Fear’ and ‘Anxious-Misery’. Whereby Major Depressive Disorder (MDD), Dysthymia and Generalized Anxiety Disorder (GAD) define the Anxious Misery’ sub-factor) and the ‘Fear’ factor is defined by Social Phobia, Simple Phobia, Agoraphobia and Panic Disorder. Similarities may extend in that the ‘Fear’ factor may underlie Internalising symptoms that are characteristically identified through somatic or physiological experience, whereas ‘Anxious-Misery’ represent cognitive distress.

Further models were fit to test for a transdiagnostic factor using the results of the EFA. Of all the items used, thus far only items with loadings on

any of the latent factors at or greater than .5 were retained for further CFA, numbering 35 of the original 53 items. Again, three models were tested against each other however, using the narrow latent factors produced by the EFA. Model E represents a Three Factor Solution: Internalising- Depressive, Internalising- Somatic and Externalising, with cross loadings permitted among the latent factors. In Model E, a General Factor is introduced to form a bifactor solution and Model F, represents a single Factor Solution.

The bifactor solution came out as the best fitting solutions. Although was significant, $\chi 2$ (429) = 1402.127, p <0.01; all other indices achieved acceptable cut off points for goodness of fit, CFI = .92and RMSEA= .06 (See Table 5). Apart from SRMR below the accepted cut-off point .06, Models E and F fell short of reaching acceptable limits of fit indices. The general factor extracted received high loadings (defined as > .7) from both the 'Internalising-Depressive' and the 'Anxious-Somatic' factors, these include 'Feelings of worthlessness', 'Feeling Fearful', 'Nervousness', Suddenly scared for no reason, , 'Feeling very self-conscious with others', 'Avoid certain places' and Feelings of guilt. Items specified to the 'Externalising' Factor loaded on the General Factor .32-.59, less strongly than the other narrow factors. Covariate scores between the factors remained high, .63 between 'Anxious- Somatic' and 'Externalising, found identical between 'Externalising' and 'Internalising-Depressive'. Covariates between 'Internalising-Depressive' and 'Anxious-Somatic' also remained high, despite at .79, despite the extraction of the general factor (See Table 7). Generally, items loading from the 'Internalising-Depressive' Factor loaded highest on the General Factor. Further Analysis comparing a bifactor model with the Three Factor model (Model E), to

determine the nature of the relationship between the narrow factors after the extraction of a general factor was not possible, as the Model E contained several *Heywood* cases, identified by standardized Loadings >1, indicating an inappropriate factor solution, not suitable for comparison.

Table 5. Model Fit Statistics for CFA After Refitting

Model Fit Statistics	Model E	Model F	Model G
Chi-Square Statistics (ML)	1746.399	1402.127	2873.016
Significance P	<0.01	<0.01	<0.01
Degrees of freedom	461	429	495
Comparative Fit Index	.89	.92*	.0.81
SRMR	0.04*	.033*	.059*
RMSEA	0.07	0.06*	.09

* represents indices of 'Good Fit'

Table 6. Inter-Factor Correlation Model F

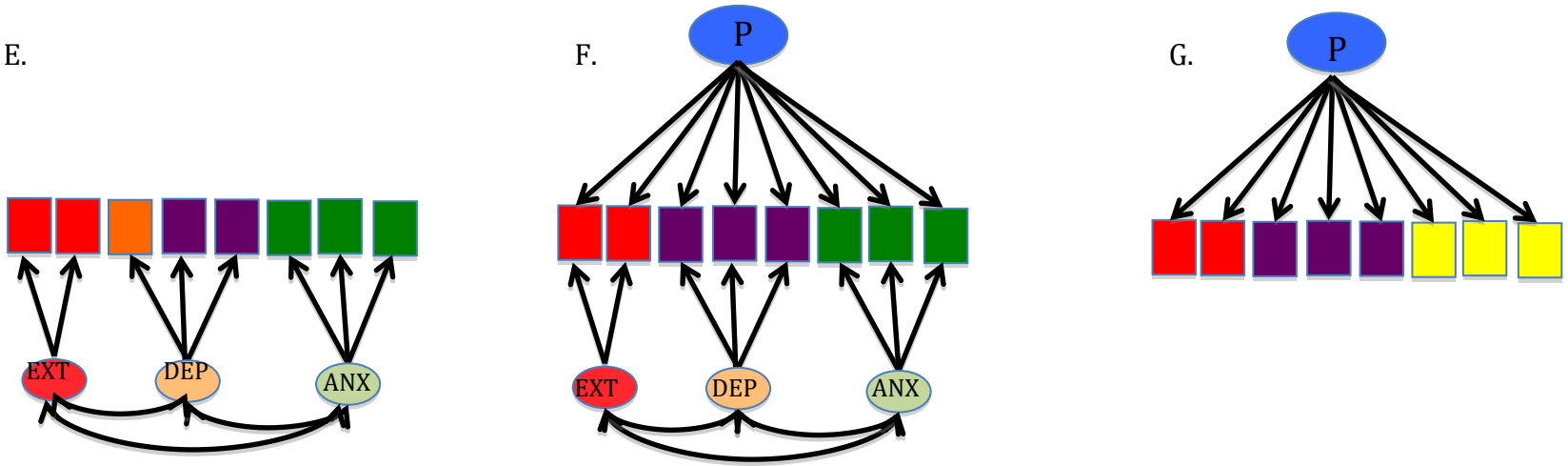
	Factor 1 'Internalising-Depressive'	Factor 2 'Anxious- Somatic'	Factor 3 'Externalising'
Factor 1 'Internalising-Depressive'	1.0		
Factor 2 'Anxious- Somatic'	.79	1.0	
Factor 3 'Externalising'	.63	.63	1.0

Table 7. Model F

Statistics, loadings & correlations					
		INT-Dep	Anx- Som	EXT	*p*
Model fit Statistics					
Chi-Square (ML)	1402.127				
Significance P	<0.01				
Degrees of freedom	429				
Comparative Fit Index	.92				
Tucker- Lewis Index	.909				
RMSEA [90% CI]	.068[.067-.072]				
Standardized factor loading					
Item 35: Feeling hopeless about the future		.49			.69
Item 50: Feeling of worthlessness		.41			.78
Item 17: Feeling blue		.53			.72
Item 16: Feeling lonely		.53			.66
Item 18: Feeling no interest in things		.48			.68
Item 52 : Feelings of guilt		.20			.71
Item 14: Feeling lonely even when you are with people		.42			.73
Item 22: Feeling inferior to others		.17			.73
Item 42: Feeling very self-conscious with others		.27			.77
Item 44: Never Feeling close to another Person		.36			.60
Item 36: Trouble Concentrating		.30			.72
Item 09: Thoughts of Ending Life		.42			.60
Item 15: Can’t get things done		.25			.69
Item 29: Trouble catching breath			.51		.53
Item 28: Afraid to travel			.33		.66

Table 7 continued. Model F		INT-Dep	Anx- Som	EXT	*p*
Item 02: Faintness or dizziness			.47		.48
Item 12: Suddenly scared for no reason			.27		.79
Item 31: Avoid certain places			.29		.71
Item 30: Hot or Cold spells			.59		.46
Item 08: Afraid of open spaces			.23		.66
Item 33: Numbness in body			.61		.39
Item 26: Having to Check and double check			.28		.66
Item 23: Nausea			.41		.52
Item 32: Mind gone blank			.35		.65
Item 19: Feeling fearful			.14		.85
Item 37: Feeling weak in parts of body			.50		.52
Item 01: Nervousness			.20		.75
Item 13: Temper outbursts that you cannot control				.62	.54
Item 41: Having urges to break or smash things				.66	.47
Item 46: Getting into frequent arguments				.60	.51
Item 40: Having urges to beat injure, or harm someone				.61	.32
Item 06: Feeling easily annoyed or irritated				.42	.59
<u>Factor Correlation</u>					
INT-Dep			.46	-	
Anx- Som				-	
Ext			.41	.41	

Figure 3. Diagram for CFA



* In the interest of graphical clarity, the number of manifest variables has been abbreviated.

Predicting Caseness from Latent Variables.

To determine and test the utility for the general factor's to predict caseness beyond the narrow factors, a logistic regression was run to test the utility of latent variables in the chosen bifactor model. Caseness categorized as either PD vs. Non-PD was regressed on 'General', 'Internalising-Depressive', 'Internalising-Somatic' and 'Externalising' factors. All factor scores for the independent variables were extracted using Maximum Likelihood Estimation and were tabulated. Initial estimates indicated that all Variance Inflation Factors (VIF) scores for the individual factors were below '10' indicating low inter-correlations (Field et al., 2012, P.343) and suitability for analysis. A model was requested with Pseudo R^2= .61. The general *p* factor was the best predictor for caseness following 'Externalizing'. Odds ratios for 'Internalising- Depressive' and 'Internalising-Somatic' were found not to be significant in terms of predictive validity in determining casensess (See Table 8).

Table 8. Model E: Logistic Regression Predicting Casness

Constant/ Factors	*B*	*(SE)*	Odds Ratio	VIF
			Odds ratio	
Constant	1.6	.20 * *		
General *P*	2.2	.20 * *	9.5	1.12
Internalising-Depressive	.40	.24 *	1.6	1.72
Internalising-Somatic	.38	.37	1.4	1.22
Externalising	.95	.28 * *	2.6	1.24

* Significance Value P = .05
* *Significance Value P < .001
Pseuod R^2= .61.

Discussion

Preliminary CFAs were requested on the BSI in both the control and patient populations at the item level to test the fit for a single factor of psychopathology against a three factor model (Externalising, Internalising and Thought Disorder) and a bifactor Model where a single factor is extracted while remaining variance for the aforementioned factors were taken into account. The process of selecting and testing models was meant to mirror the process used by Caspi et al. (2013). None of the three models displayed adequate fit indices (Hu & Bentler, 1999), and were therefore rejected. Furthermore, a data driven EFA was run to determine the relationship between the items and to define the factors they represent. EFA yielded an ideal fit for three Factors that were interpreted as 'Internalising- Depressive', 'Internalising-Somatic', and 'Externalising'. A three factor solution remained below par in terms of model fit adequacies, however when a General factor was requested to form a bifactor solution, the fit reached acceptable conditions. The primary strength of the current investigation is the use of EFA, which avoids the confirmation bias replete in previous studies that have looked into the bifactor structure. Such bias is inherent in CFA, as EFA allows for a data driven process. The findings depart from Brodbeck et al. (2014) who also fitted a bifactor model using the BSI, in that they had located an eight factor solution using EFA: 'Depressed Mood', 'Suicidal Ideation', 'Information Processing', 'Phobic Fear', 'Nervous Tension', 'Interpersonal Security', 'Aggression' and 'Somatic Symptoms' using EFA as opposed to the three factors located in this study. The researchers highlighted that the number of factors extracted was higher than anticipated. They attribute this,

as well as the absence of an ‘Externalising’ factor, to their sample consisting exclusively of treatment-seeking psychiatric patients.

The current findings are comparable to other studies that found no evidence for the emergence of an independent Thought Disorder factor. It is comparable with Caspi et al. (2013) who found that items prescribed to The ‘Thought Disorder’ factor were subsumed by other factors. Also similar to their findings, the Internalising factor was shown to be most related to the General factor and that the Externalising factor is less indicative of general *p*. Interestingly the structure of the narrow factors are analogous to Kreuger’s (1999) three-factor model in that two domains were interpreted as ‘Internalising’ along with a third Externalising domain, where the ‘Internalising- Somatic’ domain match well with Kreuger’s ‘Fear’ construct. However, the Internalising-Depressive domain is most analogous to Kreuger’s ‘Anxious-Misery’ domain, save for items pertaining directly to generalized anxiety disorder or its feature presentation, ‘worry’. A reason for this may be due to the BSI’s focus on somatic and physiological symptoms of anxiety and holding insufficient measures within the BSI to account for ‘worry’, with the exception of some items that are analogous to the DSM V (APA, p.222) criteria for generalized anxiety loading on our ‘Internalising –Depressive’ factor such as ‘Trouble concentrating’ and ‘can’t get things done’. It must also be noted, in part as a limitation of this investigation and in part due to the exclusive use of the BSI, that the items constituting the Externalising factor e.g. ‘Having urges to break or smash things’ or ‘Having urges to beat injure, or harm someone’ pertain to emotional reactions and not behaviours in the strictest sense. They arguably hold tenuous links to the manifest variables:

alcohol Dependence, drug Dependence and antisocial personality disorder that constitute Externalising factor specified by Kreuger et al. (1998), Lahey (2012) and Kim & Eaton (2015).

Also, similar to the findings of Kim & Eaton (2015), the transdiagnostic factor summarised by *p* predicted severity in psychopathology over specific factors. The scope of the current investigation yields further evidence to the descriptive nosology of general psychopathology in an adult population, and provides further evidence for the utility considering transdiagnostic factor. In keeping with the postulate that personality disorders represent an extreme form of psychopathology along a continuum or continua, there is a positive relationship between the transdiagnostic factor and the severity of psychopathology. This had been noted as far back as Clark et al., (1995) who noted severity and comorbidity are positively correlated in that individuals with severe disorders where they were more likely to meet criteria for other disorders than others. At face value, the data suggests that, the greater comorbidity among several diagnostic entities, the greater the likelihood that a severe personality disorders would emerge. An alternative view on the findings may suggest that a severe representation of psychopathology influences a myriad of difficulties and dysfunction in many domains, consistent with the *Dynamic Mutualism Model*.

Limitations

The major limitation of this investigation is the cross-sectional view of the data. It falls short in providing further information on the developmental trajectory of the general transdiagnostic factor, and as such we are unable to comment on the directional and or functional aetiology of the *p* factor. Despite

the statistical, well-fitting of the model and its predictive validity, further questions remain about *p*, most notably, its validity as a construct, beyond a mere statistical artifact. This would suggest that it could potentially be measuring subjective distress among all items measured, as a consequence of mental ill-health and dysfunction. As with *g* for general intelligence, despite its pragmatism, there is little to delineate the functional path of how *p* emerges as a liability factor as suggested by Caspi et al. (2013). To date the efforts of Patalay et al. (2015) and Murray et al. (2016) are able to indicate that *p* in early life is predictive of future mental ill-health, through longitudinal models, to suggest that it can be summarized as a yet unnamed liability factor for psychopathology. What's more the longitudinal design of their investigations offers the first element to infer a causal relationship. It is possible that *p* represents specific genetic, or environmental factors, established or as yet unknown. However, the presence of multi-modal and longitudinal data was not available in the current investigation.

A further limitation to be considered is that the vast majority of the patient group were women presenting with BPD. This presents a bias in the generalizability of the model across genders. Caspi et al. (2013) highlighted that high scorers on the Internalising factor were represented by woman in contradistinction to high scorers on the Externalising factor captured mostly by men; implying that the factors represent gendered personality styles. At its current stage of collation, the database and sample size is too small and would be underpowered to investigate gender group differences. Furthermore, a strength and arguably a limitation as well, is the use of both control and treatment-seeking patient participants. Following from the recommendations

of Mansell and colleagues (2009) who had set what they consider as "arbitrary but challenging criteria" (p. 9) to determine the suitability of a process to be considered transdiagnostic; they specify that transdiagnostic processes be assessed in both clinical and nonclinical samples. However, this is based on the working premise that symptoms of psychopathology are spectral and continual, and would require formal investigation into the metric and structural invariance between the two groups, for which this dataset is currently too underpowered.

Clinical Application

Despite equivocal claims to the 'true' structure of psychopathology, which may render the process of classification little more than a pedantic exercise, the Internalising-Externalising conceptualisation is still useful. Although Clinical Psychology is a discipline led more by a holistic and integrative Bio- Psychosocial Model, *essential* and *pragmatic* nosology is indispensable and go beyond semantic discourse. LeDoux (2015, p.11-13) highlights the defining clinical example of how the terms 'Fear' defined as physical manifestations of dread towards a dangerous situation and 'Anxiety', defined as worry about the future became distinguished. The observations relayed by Donald Klein in the 1980s, who had been studying the effects of (the then experimental Selective Serotonin Re-uptake Inhibitor or SSRI) Imipramine and found that anxiety levels in patients remained unchanged, while staff noticed a decrease in the frequency of complaints around racing heart, and shortness of breath (later known as panic attacks). By contrast the class of drugs known as Benzodiazepines that act on GABA receptors in the brain reduced anxiety but had no effect on panic attacks. This illustrates how

two diagnostic entities, Panic Disorder and Generalized Anxiety Disorder, both of which may easily be conflated in a clinical setting, are in fact differentially distinguishable along neural pathways.

Research Implications

There are several questions that remain unanswered and were beyond the scope of the current dataset. First, investigation is required to ascertain the stability of the structure between the two groups, specifically, how the structure of *p* varies between control and clinical populations. A second and interesting question is how loadings on the general factor vary after intervention. Furthermore, what kind of intervention? The current structural models for psychopathology along with other cross-sectional investigations and studies, only relied on phenotypic presentations, require validation to illustrate that psychobiological structure exists and that changes in those processes lead to salient changes in the latent variable (Borsboom et al., 2003). As the dataset stands, it is too underpowered to investigate model invariance between the control and patient groups even at a single time point. To our knowledge there are continued efforts to further recruit participants in both the control and patient groups within this database. As nearly all participants within the patient group were treatment-seeking across the two evidence based therapeutic modalities to treat personality disorders namely, Dialectical Behavioural Therapy (DBT) and Mentalizing Based Therapy (MBT), the database is fertile ground for further investigation into how the latent structure of psychopathology may alter following treatment. A final and pertinent question is the functional exploration of *p* as a 'valid' concept underlying a specifically defined liability factor leading future psychopathology.

To infer a functional and possibly causal pathway would require longitudinal data across the lifespan.

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Part 3: Critical Appraisal

Introduction

The journey through my thesis was by far one of the most challenging and rewarding experiences. I originally had the advantage of using a pre-existing database and was set to finish ahead of time. Unfortunately, I had to take a year out on sick leave. When I returned, another trainee had tackled the topic I had intended on discussing. Nevertheless, I was able to build on the subject, for which I was pleased. For many of my colleagues, trainee Clinical Psychologists, the research process represents a break from clinical practice, and it represents a sub-profession within a profession. Though there seem to be few psychologists who practically engage in both research and clinical practice, research is always in the back of my mind as it is necessary to guide psychologists in their practice through evidence-based methods. Despite the labour, the topic I chose was one that I am deeply passionate about and one that summarises and culminates years of contemplation of a discipline that remains in its infancy.

My previous theoretical orientation definitely played a role in choosing the topic. I had previously completed a Master's degree in the Psychology of Individual Differences. I graduated with a strong scientific grounding in academic psychology with a passion for latent modelling and extinguished fear of writing computer code. What I enjoy most about latent modelling is the opportunity to define and discover underlying processes that streamline the understanding of human behaviour. Since my early days as an undergraduate psychology student, I had always been dissatisfied by the multiplicity of competing theoretical models of personality, psychopathology and therapy. As a scientist in training, and a self-defined *functionalist* (in the informal sense

that I think of concepts in terms of their function), I believe in scientific truth and that psychology should be no exception. Throughout my training as a clinical psychologist I was keen to look for the commonalities in therapeutic models, such as the role 'exposure' in both Acceptance and Commitment Therapy (ACT), classic Cognitive Behavioural Therapy (CBT) and possibly Psychodynamic therapy (if one considers 'insight' a form of exposure). What was interesting to me was the apparent similarities between Structural Equation Modelling and the formulation process in clinical work. In SEM you have a matrix of data points, where all items are representative points reflecting truisms in the world and are organized in a matrix. Models are constructed, compared and selected to represent a thesis on the world that is primarily useful. Likewise in clinical formulation or conceptualisation, concrete assessment data from any given service user leads to a model that is grounded on theory, accounts for relevant psychological attributes and paves the way for practical application in the clinic or a wider system. This brought me to, what I was delighted to find, is known as the school of '*Pragmatism*' within the philosophy of science. I was also pleased to learn that the progenitor of *Pragmatism*, John Dewey was a Psychologist in profession (Hookway, 2006). It seemed that I had found my intellectual home, and was able to resolve or rather, develop the capacity to tolerate the cognitive dissonance of "picking a side".

Formulating *p* (Depression and Anxiety, a case example):

Another major theme in mental health that often has individuals 'picking sides' is the use of diagnostic entities vs. formulations. Having come from a psychiatric background, where in my home country, clinical psychology is

under-developed, I was surprised to learn that diagnostic procedure was largely ignored as either irrelevant or, at most, seen as something that is within the purview of psychiatrists. I have even encountered individuals who subscribe to ideas of *Social Constructionism* who outwardly reject the concept of psychiatric diagnosis as on political grounds.

It was difficult for me at first, to communicate psychological formulations independent of a known diagnostic entity, that I had been used to for so long. Looking at reviews of methods of psychiatric research and the paradigm shift from categorical to dimension-based classification, inevitably brought up the debates on the use of diagnosis versus the process of clinical formulation. The British Psychological Society (BPS) highlights the process of 'Psychological Formulation' as a core competency of all member psychological practitioners. A publication on the 'Good Practice Guidelines on the Use of Psychological Formulation' by the Division of Clinical Psychology (DCP) of the BPS (2011) concedes that there is no universally accepted definition for 'formulation'. It is best described as a process of "assessment, discussion, intervention, feedback and revision" of a client's psychological difficulties. (P.10). Although the term is not exclusive to the bio-psychosocial grand model of clinical psychology, within this paradigm a 'formulation' describes a given problem and highlights its predisposing, precipitating and functionally maintaining factors, as well as highlighting further risk and protective factors to the problem.

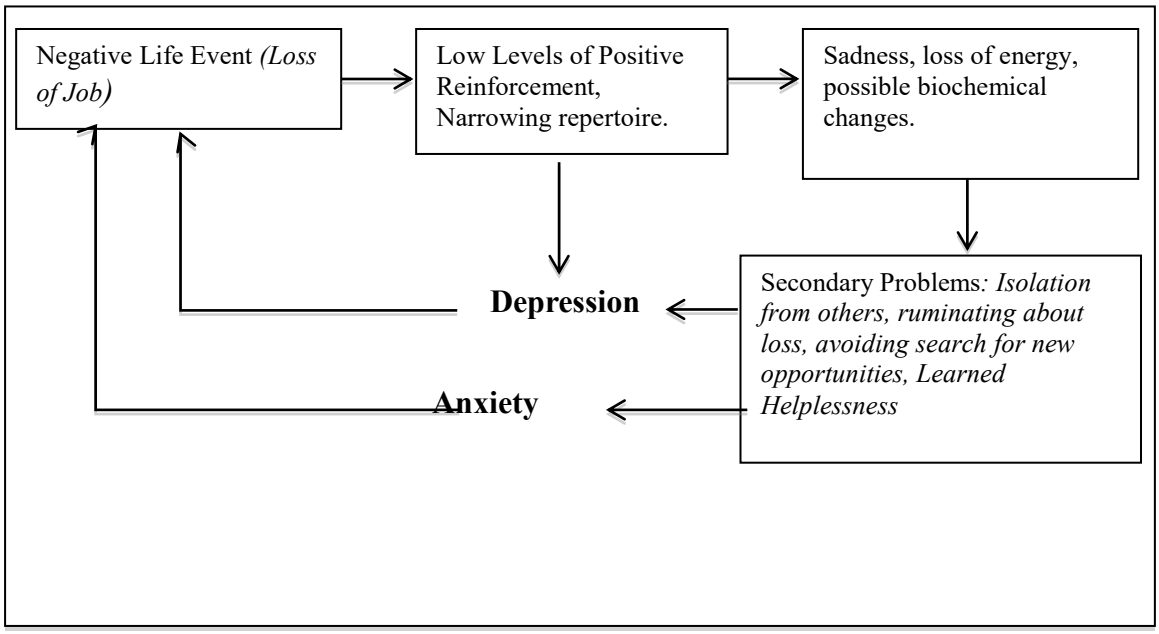
The publication acknowledges, but does not fully address the contentious, often exclusionary positions among various mental health practitioners between use of 'formulation' based models or diagnostic systems

of mental health such as the Diagnostic Manual of Mental Disorders –IV- TR (DSM-IV-TR) and its subsequent publications in the United States, and the International Classification of Diseases -10 (ICD-10) by the World Health Organization (WHO). Many of the critiques of the of the processes of diagnosis in lieu of formulation may in fact be criticism of the categorical approach to diagnosis, including but not limited to, lack of within-disorder heterogeneity and construct validity, and are not critiques of the process of classification per se. What I found with transdiagnostics and the various latent modelling techniques, is that it helped me reconcile the process psychological formulation and the importance of diagnostic entities

Figure 1. Represents a generic model formulation by (Martell et al., 2001) that accounts for the comorbidity between anxiety and depression, using behavioural theory. In this case an individual, K encounters a negative life event e.g. the loss of employment. This may leave her with low levels of reward and the narrowing of behavioural repertoires to seek positive reinforcement, as she no longer receives the enjoyed intrinsic or practical benefits of her former job. According to behavioural theory, the loss of positive reinforcement may lead to depression (Jacobson et al., 2001). Additionally, the low levels of positive reinforcement may lead to sadness and loss of energy; this could be moderated by a genetic predisposition to low mood that could lead to secondary problems. K may isolate herself and avoid others and ruminate about the loss and implicate shortcomings on her part and her abilities that lead to the loss. At this point a prolonged period of avoidance may engender in her a fear of approaching others and taking practical measures to seek further employment, similar to Learned Helplessness (Miller

et al. 1975). Concern over shortcomings may manifest itself as anxieties that may or may not tangentially meet a diagnostic for mental health disorder. Rumination over negative life events may exacerbate her low mood. K's anxiety proves to be further debilitating and leaves her with the practical disadvantage, feeling to anxious and low to take the necessary steps to get another job, this may leave her stuck in what is known as a 'vicious cycle'.

Figure 1. Behavioural Formulation of K's problem based on (Martel et al.,2001)



The clinical formulation is idiosyncratic to an individual and her circumstances but is based on and accommodates established bio-psychosocial theory and principles. In K's formulation RDoC processes are highlighted such as *Positive Valence System* and are represented in the extent to which loss of reinforcement lead to Depression. Additionally, *Cognitive systems* such as K's propensity to ruminate played a role in maintaining her Depression and engendering anxiety. The account as a whole explains comorbidity for one individual. However, clinical research is by its

very nature nomothetic as it aims to use deductive methods to uncover many of the other processes and their patterns of interaction that apply to this individual and many others.

The Process

Among the usual confusion at the early stages of choosing a research topic, I approached a number of staff members with the intention of 'doing anything… a long as I can do it well'. I was turned to the 'Probing Social Exchanges- a Computational Neuroscience approach to the Understanding of Borderline and Anti-Social Personality Disorder', group who were operating from the Functional Imaging Labs (FIL) nearby Queen's Square. The project was an internationally led, multi-site research endeavour. It was very exciting and indeed very humbling to be part of it. I thought that it would provide me with a unique opportunity to explore and be part of something ground breaking. The research team seemed ideal. Other trainees had previously worked with the team: the agreement was that in exchange for use of the database, each trainee would have to perform 30 testing sessions with both patient and control participants. Testing for each participant took place over two days. Participants had to complete an extensive battery of psychometric tests from which I extracted the BSI data. They also completed *Game Theory* tasks, some of which took place under fMRI. Part of my role was also to administer complete testing sessions in the Adult Attachment Inventory (AAI) for all participants, in addition to the Structured Clinical Interview for DSM IV (SCID-II) for the patient group. This was an incredibly rewarding experience as it allowed me the opportunity to learn how to administer psychometric tools

that few trainees would have the opportunity to do so on placements. In total I completed 30 testing session.

The Literature Review

The literature review was by far the most challenging aspect of my thesis. Initially, when I had started the project in late 2015 I was interested in *p* factor. Unfortunately, the topic was in its infancy and there were not enough publications on the topic to satisfy the breadth of a literature review. Instead, I settled on looking at the general structure of psychopathology, which included *p* factor models. Accordingly, I took a broader scope by looking at the general structure of psychopathology. The sheer volume of the results from the database search was overwhelming, and it was not clear whether I would be able to follow through a logical or coherent system to identify the logical flow between the different papers reviewed. I initially hoped to look at the general factor of psychopathology across all age groups, but that was not practical for the scope of a single 8,000- word literature review, and in the end I decided to focus on adults. Unfortunately, that meant excluding a lot of interesting papers, particularly those that used wider ranging age groups such as Patalay et al. (2015) and Murray et al. (2016), who used longitudinal data and elicited meaningful conceptualisations of the *p* factor.

The second biggest challenge with the literature review was familiarising myself with all the various modelling techniques. I believe this in itself was the most challenging part. Again, including all latent modelling techniques would require an entire doctoral thesis in itself. I did however learn a lot beyond my original knowledge base around structural equation

modelling. It was also at this point that I learned of (but not necessarily about) *Network Analysis*, and how they pertain to research in psychopathology and nosology. In Network Analysis Modelling, psychological variables as they would be known in latent modelling as manifest variables, are not treated as an underlying common cause that explains perceptible manifestations. Instead, there is an underlying assumption that psychological attribute is a complex system of perceptible components. This is in line with the *Dynamic Mutualism* model (Markon, 2006). I believe the narrowing of the scope of my literature review presents the biggest limitation in my thesis, as I struggled greatly with trying to conceptualise and communicate the narrative of the papers reviewed, while maintaining research fidelity. I wondered whether the 'shortcoming 'lay within me, being unable to understand the extent of the technical knowledge base. I managed to grasp on to clarity when I came across Krueger & Makron's (2006) conceptual paper that outlined the various mathematical models for which 'comorbidity' or the inter-relations between common mental health disorders. At this point I decided to look at papers that used multivariate models in order to narrow my search. As I mentioned earlier in my literature review, I felt that this represented the highest level or rather the broadest scope of analysis of phenotypic presentations of adult psychopathology within RDoC .Later on, as I was updating my search I came across another conceptual publication: "*Modelling Psychological Attributes in Psychology – An Epistemological Discussion: Network Analysis vs. Latent Variables"* by Guyeron et al. (2017). The paper explicitly tackled the epistemological concepts that I had previously thought about but was not able to articulate, specifically regarding *realism,* or the position of objective truths

versus *pragmatism.* I found the fact that this had been addressed very validating and reassuring that I was not overthinking matters.

The Empirical Paper

Despite the challenges of learning new and complex statistical techniques, the empirical aspect of my research was by far the most enjoyable and rewarding part. I enrolled on a an M Plus course to 'master' the technique, and although it was useful, I soon learned that I had an underlying assumption or rule that I have to learn or be knowledgeable about everything before I could undertake any research endeavour. However, I soon learned that research, much like clinical training, involves a lot of learning on the job. I had previously done a Master's thesis that I thought was challenging, but in retrospect I had a lot of support and guidance from my supervisor. Finding myself on a Doctoral course, I learned that the culture involves a lot of independent work and auto-didactic training, which has enhanced my self-sufficiency in tackling research problems.

In practice, empirical research involves becoming very familiar with your data, and I found M plus to be too rigid. After much hesitation, I switched back to R, a programme I was much more familiar with. I had previously been very hesitant to do so because none of my supervisors were familiar with it, and I had been anxious to go at the challenge alone. In the end, learning Laavan, the package designed for latent modelling proved to be another transferable skill I picked up and a reward to facing the challenge.

Unfortunately, after I had come back from interruption of studies, having learned that the question I had initially proposed had been partially

tackled by Gibbons (2017) on the same dataset, I thought I had to change my research topic completely. I was left feeling stuck and despondent and this lead to an impasse. However, after carefully looking through her work, I realised that she had tackled the question differently to how I would have, and so I went forward with my own method. Having struggled, with the various methods and techniques across the different papers in the literature review, I felt that I wanted to do something that could be easily reviewed by other researcher in line with work and methods that had already been tried and tested. To do so I aimed to follow Caspi et al. (2013) as closely as possible. When my initial models failed to meet cut-off criteria, I was discouraged and wondered what I had done wrong, knowing full well that it was not the appropriate attitude for research. Falling back to the methods used in my review papers, I chose to use EFA, a procedure that I was familiar with. The results were very 'heartening' I was pleasantly surprised that the Internalising factor had bifurcated similarly to earlier works, namely the seminal work of Krueger (1999). The fact that data driven analysis yielded results that were comparable to the research base gave me much needed confidence in the methods and research decisions that I had made, and I strongly encourage future researchers to embrace impasses with null findings and not feel the need to 'prove' themselves original.

Criterion Validity of the Construct

As the validity of the *p* factor as a theoretical construct depends on its pragmatic utility, a useful clinical criterion had to be established, beyond a descriptive account of its structure. A major shortcoming of the current empirical efforts is that it lacks longitudinal data, depicting the progression or

regression of psychiatric symptoms. Other studies have demonstrated the utility of the *p* factor in demonstrating that, as a measurable construct, it retains temporal reliability and lends predictive validity to further psychopathology across the lifespan (Patalay et al., 2015 ; Murray et` al., 2016). In the current investigation I was able to demonstrate that at least within a contemporary time frame it remains useful in predicting casensess between 'normal' presentations of mental ill health and severe presentation thereof, or at least be able to say that, 'for a yet unknown cause' severity in psychopathology is associated with higher levels of `comorbidity among various different symptoms.

Logistic Regression vs. Discriminant Analysis?

Another learning point for me came when it was time to decide which statistical test to use for the second part of the empirical project. My supervisor had told me to extract factor scores and go forward with Linear Discriminant Analysis (LDA). LDA was another modelling technique that I was familiar with and found very interesting but had never used myself before. I learned how to use it and understood its underlying principles, however despite being told what to do, the more I learned about it the more I realised that it was not the appropriate method to test and communicate my findings. Generally, when the criterion validity of the *p* factor in its relation to PD or non PD- crassness was considered, the question whether to use Logistic Regression (LR) or a Linear Discriminant Analysis (LD) arose. Apart from the heuristic method of classifying outcome and predicting measures as continuous vs. ordinal to choose the modelling technique of choice, it was necessary to refer to the logical implications of either choice. Ultimately an LR

was chosen as it treats as consider independent variables as continuous, and best suits the postulate that the severity of psychopathology is a continuum that traverses diagnostic entities. Ultimately, I brought the subject up with my supervisor and made the case for the use of LR, which was accepted. Although this may seem like a fatuous point to mention, it was in fact a very important and major learning experience for me. To do so, I had to overcome trepidation and a lifelong pattern of unquestioning reverence to authority. It was a small step that paved the way for me to discover my own confidence and the importance of critical thinking.

Future Directions

Moving on from this research project, I think that the topic of the general structure of psychopathology has been sufficiently investigated and that INT-EXT paradigm has stood the test of many investigations across different populations and datasets. The *p* factor is a burgeoning concept that has shown its utility in making sense of how comorbidity is understood and has practical clinical research applications. However, it seems that further along, research should focus further on narrower questions within the hierarchy set by RDoC. Personally, I hope to continue my research career with specific focus on the interplay between the RDoC processes.

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Appendix A

Table 1 Means and Standard Deviations for Items and Subtotals for PD, Control (non-PD) and Pooled Samples.

	PD		Control (non-PD)		Pooled sample	
Item/Subscale	Mean	SD	Mean	SD	Mean	SD
SOM	1.4	1.0	.43	.5	1.1	1.0
2	1.3	1.3	.5	.7	1.0	1.2
7	1.2	1.4	.2	.6	.9	1.3
23	1.8	1.3	.6	.9	1.4	1.3
29	1.2	1.3	.2	.5	.9	1.2
30	1.5	1.4	.3	.8	1.1	1.3
33	1.4	1.4	.4	.7	1.1	1.3
37	1.8	1.4	.5	1.0	1.4	1.4
OC	2.3	1.0	.8	.7	1.8	1.2
5	2.1	1.3	.8	.9	1.6	1.3
15	2.4	1.4	.8	1.1	1.9	1.5
26	2.2	1.4	.6	.9	1.7	1.5
27	2.5	1.3	.7	1.0	1.9	1.4
32	2.3	1.3	.7	.9	1.7	1.4
36	2.7	1.2	1.0	1.1	2.1	1.4
I-S	2.3	1.1	.4	.6	1.7	1.3
20	2.3	1.3	.4	.8	1.7	1.5
21	2.2	1.3	.4	.7	1.6	1.4
22	2.1	1.4	.4	.7	1.6	1.5
42	2.5	1.3	.5	.8	1.9	1.5
DEP	2.4	1.1	.5	.7	1.8	1.3
9	1.6	1.4	.08	.3	1.1	1.4
16	2.6	1.4	.8	1.0	2.0	1.5
17	2.7	1.3	.8	1.0	1.2	1.5
18	2.5	1.3	.6	.9	1.9	1.5
35	2.6	1.4	.6	1.1	2.0	1.6
50	2.7	1.3	.4	.9	2.0	1.6

Table 1 continued. Means and Standard Deviations for Items and Subtotals for PD, Control (non-PD) and Pooled Samples.

ANX	1.9	1.1	.4	.5	1.4	1.2
1	2.1	1.3	.8	.9	1.7	1.3
12	1.7	1.4	.2	.6	1.2	1.4
19	2.0	1.3	.4	.7	1.5	1.4
38	2.4	1.3	.5	.8	1.8	1.5
45	1.7	1.4	.2	.6	1.2	1.4
49	1.7	1.4	.4	.8	1.3	1.4
HOS	1.6	1.1	.4	.5	1.2	1.1
6	2.6	1.2	1.0	1.0	2.1	1.3
13	1.7	1.5	.2	.6	1.2	1.5
40	1.0	1.4	.1	.5	.72	1.25
41	1.4	1.5	.2	.7	1.0	1.1
46	1.4	1.4	.2	.6	1.0	1.3
PHOB	1.6	1.1	.2	.4	1.2	1.2
8	1.1	1.3	.1	.5	.8	1.2
28	1.9	1.4	.1	.5	1.2	1.5
31	1.7	1.5	.1	.6	1.2	1.5
43	2.3	1.4	.4	.8	1.7	1.5
47	1.5	1.4	.2	.7	1.1	1.3
PAR	1.9	1.0	.4	.6	1.4	1.1
4	1.3	1.3	.4	.9	1.0	1.2
10	2.3	1.4	.5	.8	1.7	1.5
24	1.9	1.4	.3	.8	1.4	1.4
48	2.0	1.4	.4	.8	1.1	1.3
51	2.3	1.5	.6	1.0	1.8	1.6
PSY	1.9	.96	.3	.5	1.4	1.1
3	.6	1.1	.1	.5	.4	1.0
14	2.5	1.4	.4	.9	1.9	1.5
34	1.5	1.5	.1	.4	1.0	1.4
44	2.0	1.4	.4	.9	1.5	1.5
53	2.8	1.4	.4	.8	2.0	1.6
Other						
11	1.7	1.4	.4	.9	1.3	1.4
25	2.6	1.4	.9	1.1	2.1	1.5
39	2.1	1.5	.2	.7	1.5	1.6
52	2.2	1.5	.4	.8	1.	1.5