ON STUDYING THE RELATIONSHIP BETWEEN PERSONALITY AND MENTAL DISORDER – SUGGESTIONS FOR FURTHER STUDY

BY

WILHELMINA JO BERRY

A thesis

submitted to the Victoria University of Wellington

in fulfilment of the requirements of the degree of

Master of Arts

in Philosophy

Victoria University of Wellington

2021

Abstract

Links between traits and mental disorders have been postulated off and on for centuries, but since a recent revival of interest within psychology, there has been a consistent and expanding field of research concerned with studying what is now widely accepted as the 'personality-psychopathology relationship'. This thesis explores that field of research, considering what has led to its stagnation and apparent difficulty in reaching robust and useful conclusions. In doing so, I provide an overview and critique of the study of the personality-psychopathology relationship. Several limitations of recent research are identified and explored, specifically concerning its focus of inquiry, statistical methodology and conceptual foundations. Throughout the thesis, I discuss the appropriate scientific method(s) and necessary conceptual considerations involved in studying the relationship between personality and mental disorder, and make some suggestions about what is required for robust and reliable research in this area.

Acknowledgements

In completing this thesis I have been fortunate enough to have both Simon Keller and Ed Mares as supervisors. Thank you both for the education, support and academic insight you have provided, not only for this work, but also throughout my entire tertiary education at the university.

I am particularly thankful to Ethan Carter. Your support and partnership in all aspects of my life is invaluable. I have especially appreciated this over the past few years. Thank you for your patience, insight and ideas.

Thank you also to Thomas Berry and Cassidy Dewar. Your food, discussions and calming input are greatly appreciated. Kaitlin Martin-Feek, you have been amazing in more ways that I can list, thank you! Thank you Steve and Em for your friendship and everything you put up with. And Mum – without your input and wisdom nothing in my life would go as smoothly as it does. I am very lucky to have your support.

Finally, my biggest thanks go to my Dad. The interest and time that you have invested my work has made this endeavour possible. You have answered endless phone calls, edited many poorly written sentences and thought through ideas upon ideas with me. Thank you truly for everything you have done. This thesis is for you (no give backs).

Table of Contents

Introduction

From a surprisingly young age, my cousin has been fascinated with music and mathematics. At the age of 2, he could list more prime numbers than I can today. He has perfect pitch and plays a range of different instruments, but piano seems to be where he really excels. As a young child, he used numbers to express his emotions, assigning 'good numbers' to happy feelings and people he was pleased with, and 'bad numbers' to unhappy feelings and people who he felt negatively towards. His social interaction methods were limited, mostly learnt from playing computer games, and he was shy and gentle, task-focused, risk-averse and notably picky about what he did and didn't enjoy.

To a large extent, these, and similar attributes, make him who he is. Over time, his outward tendencies have shaped, and continue to play a substantial role in defining, *him as a person*. They have formed the basis of his interactions with other people, and with the world more generally. His complex personality and particular set of interests and skills have a clear impact on his behaviours, relationships, and life choices, and through the combination of these different facets, he has formed a sense of self, or a self-identity, contributing to his understanding of who he is and how he fits in the world around him.

Yet interestingly, many of these factors that appear to make him, him, are closely aligned with common, almost stereotypical, descriptors or characteristics of autism spectrum disorder (ASD). Some of his most prominent outward tendencies, including social abnormalities, speech tendencies and particular and peculiar interests, might be seen as typical *symptoms* or *indicators* of autism.

Having never had an ASD diagnosis, the question of whether or not he is autistic presents us with a challenge that is faced constantly not only by psychologists and psychiatrists, but also by parents, teachers, politicians and the wider public: how can we, do we, or should

we identify mental disorders in comparison to individual personal differences? In the case of my cousin, how can his person (his traits, behaviours and so on) be separated from a potential disorder? Would it be responsible or wrong to diagnose, and potentially treat, his 'autistic-like' attributes? And how might his personality influence, or be influenced by, the presence of ASD?

This intersection, where personality and mental disorder meet, is rapidly moving to the forefront of psychopathological research. The potential role of personality in our understanding of the development, presentation, diagnosis and treatment of mental disorder has recently become a substantial and expanding area of study. This thesis explores that domain further. We start by considering mental disorder more broadly, before bringing personality into the mix, and narrowing the focus specifically to their relationship and the studies that explore that relationship.

Mental disorder

The idea that people can be psychologically 'ill' has been around for centuries, but our understanding of what mental disorder actually is and how to deal with it has changed drastically over time. Although confusion and uncertainty still remain, mental disorder is both prevalent and considered an important phenomenon in today's society.

The World Health Organisation has reported that mental disorder now affects one quarter of the global population (World Health Organisation, 2001). New Zealand, like many other countries, legally permits the compulsory assessment and treatment of individuals, including admission to hospital against the patients' will, on the grounds of a preliminary mental disorder diagnosis (Mental Health Act, 1992, s.10(1)(b)(ii)). Medical, legal, social and political discourse all identify a fundamental and radical difference between the statements *I am unhappy* and *I suffer from depression*. Behaviours, traits and mental states labelled 'mental disorders' are considered importantly different from those that are not (Arpaly, 2005, p. 282).

Moreover, many consider a diagnosis of mental disorder to be importantly different from a diagnosis of some sort of physical condition (Cooper, 2013, p. 487). Mental disorder, it seems, is a unique concept deeply embedded within (Western) society.

Yet academically, the notion of mental disorder is considered problematic, and its meaning presents a subject of extensive disagreement (Cooper, 2013, p. 487; Boorse, 1976, p. 61). The legitimacy of there being any mental disorder concept *at all* has been challenged (for example, by Szasz, 1960 and Sarbin, 1967), but even insofar as it is accepted that some conditions do constitute mental disorder, there is little consensus as to what unites those conditions as a kind, or how we might even begin to find that out.

The study of mental disorder

With the concept of mental disorder being subjected to ongoing scrutiny and re-evaluation, determining what, if anything, makes a condition a mental disorder has become a prominent concern in recent psychological discourse (see McNally, 2011, ch. 1; Wakefield, 1992, p. 373-374; Perring, 2010, s1). Philosophical studies have been particularly interested in investigating the *nature* of mental disorder (Davies & Levy, 2017, p. 1; Perring, 2010, s0), and moreover, in addressing related issues of definition and classification (Perring, 2010, s3; Ramirez, n.d., s2). Perhaps the most common approach to these sorts of conceptual problems, at least from within the philosophy of mental health and disorder, has been to focus on the distinction between normality and disorder (Perring, 2010, s3; e.g. Wakefield, 2007, p. 149). Such an approach acknowledges that many mental conditions can manifest at subclinical levels, and sets out to distinguish normal problems in living from genuine medical disorders (Wakefield, 2007, p. 149; Rettew, 2013, p. 85; McNally, 2011, ch. 1). Several academics have suggested that a clear understanding of the delineation between normal behaviours and pathological conditions is required for effective clinical practise, as well as playing an important role in addressing a

variety of political, legal and societal matters (Wakefield, 1992, p. 373; see also McNally, 2011, ch. 2 & ch. 8; Wakefield, 2007, p. 149).

However, explaining the boundary between normality and mental disorder poses a difficult task. Different (categorical) accounts of mental disorder have identified different distinguishing features that might be thought to separate normal from disordered mental states. Examples include "disunity, irrationality, the presence of suffering and disability, and several forms of dysfunction" (Radden, 2019, s4; see also Widiger & Clark, 2000, p. 949-952). But despite an extensive body of literature, definitive explanation as to what separates normality from mental disorder seems to be a complex and distant goal.

Dimensionality of mental disorder

Recently, mental health and disorder discourse has been influenced by an increasing endorsement and adoption of a dimensional approach to conceptualisation and classification (Lilienfeld, 2014, p. 130; Kozak & Cuthbert, 2016, p. 287). A dimensional perspective of mental disorder primarily entails acknowledgement that human functioning and mental states fall along continuous dimensions. Dimensional conceptualisations and classifications involve understanding mental disorders as phenomena that merge continuously with non-disorders and with each other. This perspective is widely thought to be the most empirically accurate interpretation of mental disorder (Lilienfeld, 2014, p. 130; Widiger & Gore, 2014, p. 4; Kozak & Cuthbert, 2016, p. 287-288), as well as having valuable social and clinical implications (Rettew, 2014, p. 43; Brown & Barlow, 2005, p. 552; Kozak & Cuthbert, 2016, p. 287-288).

The recent move towards a dimensional framework of mental disorder has highlighted inadequacies with the 'categorical' or 'taxonic' systems. This previously dominant approach towards conceptualisation and classification seeks to identify discrete and qualitatively distinct conditions, characterising mental disorder as an all-or-none medical phenomenon (Widiger &

Gore, 2014, p. 3-4; Rettew, 2014, p. 42; Coghill & Sonuga-Barke, 2012, p. 469). Increasingly, research is beginning to suggest that there are no (or at best, very few) clear-cut boundaries that can accurately demarcate normal from disordered mental states (Lilienfeld, 2014, p. 131). Such findings are undermining the categorical goal of identifying some unique distinguishing feature(s) that can define 'mental disorder'. Consequently, research suggests there is a potential for real progress to be made by exploring dimensional conceptualisations that understand mental disorder as falling on a continuum with other phenomena (such as traits and characteristics, or other disorders) (Brown & Barlow, 2005, p. 553-555).

This has involved a radical shift in the way that mental disorder is classified and conceptualised. A growing body of literature is now seeking to understand and explain mental disorder in terms of its link to broader biological and environmental variables (examples include Kozak & Cuthbert, 2016; Brown & Barlow, 2005; Lilienfeld, 2014*;* Widiger & Gore, 2014). This process involves beginning to think about mental disorder in terms of its *relationships* with other 'non-disordered' states, as opposed to looking for *distinctions* that appear to be not empirically supported, nor present in clinical contexts.

The role of personality

As research progresses, our mental disorder concept has rapidly changed, and particularly in the past few years we have seen a substantial shift in the way mental disorder is understood. Alongside the increasing endorsement of dimensionality there has been a move away from the 'medical model' – which sees mental disorder as bodily dysfunction to be delt with by medical research and practice (Murphy, 2015, s1) – towards a more 'psychosocial' understanding. These recent changes have made room for new work investigating the development and presentation of mental disorder, particularly as it relates to other phenomena. Personality research has been central in this area.

The apparent merging of mental disorder with *personality* has played a fundamental role in justifying recent dimensionality arguments (examples include Van Leeuwen, Mervielde, De Clercq & De Fruyt, 2007, p. 63-64; Widiger & Samuel, 2005, p. 496-498; Eysenck, 1975, as cited in Busfield, 2011, p. 119-120). Some sort of link between personality and mental disorder (either as phenomena or specific traits and symptoms) is often provided as a paradigm example of the dimensionality of our mental disorder concept. Similarly, in early attempts to move the Diagnostic and Statistical Manual of Mental Disorders (DSM)[1] towards a more dimensional framework, personality disorders were proposed as the best starting point, with the proposition that if that were to be successful, it would then be appropriate to explore implementing similar changes for other mental disorders (Widiger & Smith, 2008, p. 749). Specific attention has been paid to the complex, but likely dimensional, relationship between personality disorders and general personality (Widiger & Smith, 2008, p. 30).

Moreover, personality-centric studies of mental disorder are becoming increasingly common, and literature repeatedly reports the increased utility, if not the indispensability, of incorporating the role of personality in understanding and explaining individual cases of mental disorder (examples include Krueger & Eaton, 2010, p. 98; Krueger & Tackett, 2003, p. 121; Lahey, 2004, p. 88). Clinical observations that personality is linked in meaningful ways with mental disorder have also had a significant influence within recent literature (for more see Brown & Barlow, 2005; Van Leeuwen et al., 2007; Kozak & Cuthbert, 2016). A number of academics have endorsed the proposition that integrating personality research – both on the underlying structure of personality and on specific personality traits – with psychopathology research will significantly advance and help explicate the current understanding of the aetiology of mental disorder, and have important implications for its conceptualisation,

[1] One of the main mental disorder classification handbooks used in the mental health field.

assessment and treatment (Depue, 1995, p. 435; Durbin & Hicks, 2014, p. 362; Frick, 2004, p. 5; Krueger, 1999, p. 41; Lahey, 2004, p. 88; Watson, Clark & Harkness, 1994, p. 28; Watson, Gamez & Simms, 2005, p. 47; Widiger & Smith, 2008, p. 762). According to psychiatrist and researcher, David Rettew, "… questions about the relations between [personality] and psychopathology constitute a new frontier in psychiatric research that could change not only how we define illnesses but also how we treat or prevent them" (Rettew, 2013, p. 102). Within psychopathological research, there is thought to be much to gain from exploring a personality-based approach to mental disorder.

The relationship between personality and mental disorder

The potential role of personality as a non-disordered mental state in understanding and conceptualising mental disorder is now widely recognised, and its investigation and endorsement are becoming increasingly common.

Over the past few decades, numerous psychological studies have been devoted to exploring this apparent link between personality and psychopathology (mental disorder[2]). A predominant focus of such studies has been on determining whether personality and psychopathology are in fact empirically related, but there has also been a significant body of work committed to investigating what the relevant literature refers to as the "nature" of the apparent relationship (Watson & Clark, 1994, p. 4; Watson & Clark, 1995, p. 361; Tackett, 2006. p. 584; Krueger & Eaton, 2010, p. 111; Durbin & Hicks, 2014, p. 363). This has involved delving further into the connection between the two phenomena, focusing on the aetiology, structure and/or neurobiology of personality and psychopathology, and attempting to study the

[2] I will mostly use the term 'psychopathology' in this thesis, only because it is in keeping with the relevant literature. However at times I switch between the terms 'psychopathology' and 'mental disorder' (using them interchangeably). Their difference is discussed in part 1 below.

complexities of the relationship between them (Widiger & Smith, 2008, p. 743, p. 756; Akiskal, Hirschfeld & Yerevanian, 1983, p. 808; Rettew, 2013, ch. 6; Tackett, 2006, p. 584, p. 588-594; Kreuger & Tackett, 2003, p. 110-112).

If we return to the case of my cousin to help illustrate the types of questions that these studies have been asking, rather than merely considering whether or not he should be considered Autistic and why, they might question the complexities of the apparent relationship between his personality and autism spectrum disorder.

For example:

- could his shy, gentle, risk-averse personality traits also be symptoms of ASD?
- Might his personality and the disorder ultimately be the same thing? Or variations of the same thing?

Questions about evidence and prediction might include:

- could his particular obsessions and task-focused behaviours be evidence of his having a disorder?
- do those or some of his other autistic-like traits give us reason to believe that he will meet criteria for an ASD diagnosis, or develop the disorder later in life?

Further still:

- could his particular personality cause him to have ASD?
- could an underlying disorder be causing him to be picky and struggle with typical social interactions?

These are the sort of question considered within recent studies of *the nature* of the apparent relationship between personality and psychopathology. Their answers have the potential to inform not only how we understand mental disorder theoretically, but also how we approach it practically, including for purposes of diagnosis, treatment, education and policy.

It is those studies of the relationship between personality and psychopathology – particularly its nature – that provide the focus of this thesis.

On studying the relationship between personality and mental disorder

Psychological and clinical literature concerned with the apparent 'personality-psychopathology relationship' is expansive. New research continues to build quickly upon previous work, drawing on the findings of others in order to progress. With such excitement and expectation surrounding the potential benefits of incorporating personality into mental disorder research, findings and conclusions seem to be expeditiously shared. As this work begins to extend outside of the realm of academic research, pushing into wider discussions via clinicians, media, and general literature, it has the potential to significantly impact social and societal understandings of mental disorder, as well as having implications for the way individuals are diagnosed and treated by professionals.

Interestingly, this exciting and influential new realm of research has received little scrutiny and evaluation of the foundations that underpin it. Specifically, there appears to have been no extensive and substantial analysis of the study's aims, objects and methods of inquiry, nor of the conceptual, ontological and methodological considerations that are involved with studying the apparent relationship between personality and psychopathology.

This thesis is centred on considering how we go about studying the relationship between personality and psychopathology. As will be discussed, personality and psychopathology are so complex and difficult to grasp as phenomena and concepts that they provide tricky objects of inquiry for empirical study, with their intersection being even more tricky. That is certainly not to say that their study isn't worthwhile; in fact, the phenomena and their relationship are particularly interesting subjects, and the need for their further exploration is clear. But there is also a need to consider the way that study is achieved. Studies and

discussions of the relationship (especially its nature) appear to have gained substantial footholds in both academia and practice without rigorous scrutiny and analysis of the theory and scientific enquiry that underpins that work. This is problematic, especially due to the impact that this body of research has on our mental disorder knowledge and practice. Findings in this field have the potential to radically change the way that mental disorder is understood, including its diagnosis, treatment, policy and so on, (Rettew, 2013, p. 102-104; Lahey, 2004, p. 88; Tackett, 2006, p. 593-594) and thus it is important that the research informing that understanding be both robust and reliable. Recently, a great deal of work has been done in reviewing and challenging the way that psychological phenomena are studied, especially in respect to the conceptualisation of phenomena and the empirical methods of their study[3]. There is a current need for this sort of work to be done in the realm of personality-psychopathology research, and that is what this thesis begins to address.

The central question of this thesis is:

What is required for robust and reliable study of the relationship between personality and psychopathology?

This involves considering: *what are the limitations of current attempts to study the relationship (specifically its nature)?* and *what are the appropriate scientific method(s) and necessary conceptual considerations involved?* Thus, this thesis is not an attempt to answer the question *what is the nature of the relationship between personality and psychopathology?*. It is instead concerned with *how* we might go about achieving that understanding.

In answering the above question, this thesis performs two main tasks. The first is providing a comprehensive review and analysis of the work studying the relationship between personality and psychopathology. This involves providing an overview of the body of work,

[3] For example see Haig, 2014.

but also discussing the research project more specifically, considering the studies' aims and general approach. The second is evaluating and critiquing the scientific process of that research. Here, I am concerned primarily with studies of the nature of the relationship. Specific attention is paid to illuminating the indeterminacy, vagueness and assumptions that are present in the growing body of work, and emphasising the fact that addressing, or at least acknowledging, those areas will have significant implications for the study.

From this evaluation I make some suggestions about requirements for improved personality-psychopathology research. Broadly, I conclude that robust and reliable study of the relationship between personality and psychopathology requires:

(a) a clear research project – by that I mean that it should be apparent what the study is seeking to achieve; there should be a clear and unambiguous focus of inquiry (or multiple) and the distinct tasks involved should be recognised and engaged with appropriately;

(b) caution around the use of empirical and statistical methods of inquiry – especially in relation to identifying, representing and measuring personality and psychopathology;

(c) consideration of the conceptual foundations or context of the study, and more specifically, the conceptualisations of personality and psychopathology as objects of study.

The thesis is divided into 3 parts.

Part 1 discusses the concepts, history and theories that underlie recent attempts to study the relationship between personality and psychopathology. I start (§1.1) by looking at personality and psychopathology as concepts, including their role in the personality-psychopathology literature and various attempts to define or demarcate their boundaries. Following that (§1.2) is an overview of the work involved in determining that personality and psychopathology are in fact empirically related. I explain the emergence of the body of literature, the relevant studies' methods, and their general findings. Then parts 2 and 3 get to the heart of the matter.

Part 2 is an overview and analysis of current attempts to study the nature of the relationship between personality and psychopathology. Here, we start to look at the scientific method(s) and conceptual considerations involved in that study. I present the general approach of the study (comprising two key stages) (§2.1), discuss each of those stages in turn (§2.2 and §2.3), and argue that the focus of inquiry of the study as a whole is undefined and reasonably unclear (§2.4) – there is not a clear research project to engage with, and multiple distinct tasks are being confused and conflated.

Following that, part 3 is an evaluation and critique of that work, particularly its aims, objects and methods of inquiry, and a discussion about *what is required for robust and reliable study of the relationship between personality and psychopathology.* I continue to discuss the study's focus, particularly as it relates to the tasks of description and explanation, and the importance of having a clear research project (§3.1). I also discuss the empirical methodology, and the capacity of statistics as a tool for exploring relations between personality and psychopathology (§3.2). Finally, I return to discussing personality and psychopathology concepts, and their role as objects of inquiry in empirical study (§3.3).

PART 1: Personality, Psychopathology, and their Relationship

Before delving straight into the complexities of studying the relationship between personality and psychopathology, we briefly consider the core concepts involved, as well as the history and theories that underlie present work. Understanding what is meant by 'personality' and 'psychopathology' is fundamental to understanding the studies' aims, objects and methods of inquiry. Thus, we start by looking at personality and psychopathology as concepts, and how they are distinguished from each other (§1.1). We then move on to considering how the influential, almost axiomatic, link between the two concepts came to be established (§1.2), as this has the potential to shed light on the intentions of current research. I present a short overview of the personality-psychopathology studies (continuing in §1.2), before moving onto discussing the work surrounding its *nature* (in part 2).

1.1 'Personality' and 'Psychopathology'

Exploring the relationship between personality and psychopathology has the promise of helping to resolve complex issues in our understanding of mental disorder. Yet the concepts personality and psychopathology are complex in and of themselves. While most of us have the ability to quickly and confidently employ the terms, explaining their meaning tends to be more difficult. Despite the feeling that it should be a simple task to identify the distinguishing features of both personality traits and psychopathological symptoms, attempts to actually articulate those features suggest otherwise. Pointing to an instance of a personality trait, such as the extroversion of your friend, may be quite straightforward, but explaining what makes that a matter of personality is more challenging. The same applies to instances of

psychopathology, where we can say, for example, that suicidal thoughts tend to be a symptom of a psychopathological condition, yet face uncertainty in trying to explain why that is the case. Equally challenging is identifying what distinguishes the two concepts from each other. For instance, figuring out when introverted and antisocial behaviour should be considered a product of an individual's personality versus a symptom of a psychopathological condition can pose a problem for even the most experienced medical professionals.

Studying the *relationship* between two variables generally requires conceptual clarity around their *distinction*. Without that distinction, the two variables aren't individually and uniquely identifiable from each other. Consequently, analysing the patterns and details of their co-occurrence, such as their order of presentation or their points of convergence and divergence, is difficult to achieve.

This is clearer if we consider less colloquially differentiated phenomena, such as hope and optimism. To determine that optimism precedes hope, for example, or that optimistic individuals are more likely to be hopeful, requires understanding what makes an individual optimistic versus hopeful.

In general, it's tricky to measure links between phenomena for which we lack definitions of some sort, or at least rough parameters for their identification (individually) and segregation (relatively). This section discusses the terms 'personality' and 'psychopathology', and more specifically, how the terms are used within the literature that explores their apparent relationship.

1.1.1 Personality

Personality *traits* have a prominent role in conceptualisations of personality. Explaining what is roughly meant by the term 'personality' appears to be easily achieved by providing examples

of individual traits, such as friendliness, narcissism and enthusiasm. Traits are complicated in that they don't necessarily belong to personality alone. However, traits are directly associated with temperament and character, and are also closely related to other psychological phenomena such as mental states and behaviours. Distinctions between traits and temporary moods, for example, can be hard to pin down, despite the important colloquial understanding that being in a grumpy mood is different to being a grumpy person. While some qualities such as extraversion and conscientiousness are well defined as personality traits, many others, such as aggression, impatience, generosity and sensitivity, are not so easily categorised as personality (as opposed to temperament, character, etc.) or as traits (as opposed to some sort of state).

Trait models, such as the Five Factor Model, have been the dominant way of conceptualising or quantifying personality in the personality-psychopathology literature (Durbin & Hicks, 2014, p. 363). Trait models conceptualise personality as "dimensions of individual differences in affect, behaviour, and cognition that are relatively stable across time and situations, and can be hierarchically organized from relatively narrow constructs at lower levels to broad constructs at higher levels" (Durbin & Hicks, 2014, p. 363). Under this conceptualisation, a defining feature of personality is its 'stability' – while moods, behaviours and mental states come and go, an individual's personality is less fickle. Most predominantly, trait models view personality as being made up of a series of broad trait dimensions (e.g. the Five Factor Model's 'big five' are neuroticism, extraversion, conscientiousness, agreeableness, and openness to experience). The higher level traits are umbrellas for lower level traits (e.g. volatility and withdrawal are sub-traits of neuroticism, and each sub-trait subsumes even lower level traits called 'facets'[4]). This trait organisation is said to make up the structure of personality (Fajkowska & Kreitler, 2018, p. 5; Durbin & Hicks, 2014, p. 363).

[4] See DeYoung, Quilty & Peterson, 2007.

The extent to which personality is defined within individual personality-psychopathology studies varies across the literature. Many studies restrict their scope (either explicitly or implicitly) to trait models of personality. Others fail to provide any sort of definition of personality, they simply state the tool or 'instrument' that their study uses to measure the personality of their sample. Very few researchers of personality-psychopathology discuss the concept of personality in greater depth. Those who do (e.g. Klein, Kotov & Bufferd, 2011, p. 270-271; Tackett, 2006, p. 585-587), tend to focus on the way that personality is classified and measured. To my knowledge, none of the literature presenting personality-psychopathology studies addresses the core conceptual and ontological issues surrounding the personality concept. Researchers appear not to engage with, or even acknowledge, the controversial and defining issues associated with conceptualising personality. The core issues include questions such as: what is the relationship between personality traits and behaviour?; what is the ontology of personality (and/or personality traits)?; are personality traits merely descriptive, or do they have explanatory power? (Fajkowska & Kreitler, 2018, p. 5-9). These are longstanding and complex issues, and it is not surprising that literature on the relationship between personality and psychopathology does not delve deeply into them. However, it is noteworthy that the assumptions the studies make in relation to these issues are unclear and, as will be discussed later, this has implications for studying and interpretating the link between personality and psychopathology.

Finally, aside from a single definition of personality, distinctions between personality, temperament and character are also disputed. Different positions on the matter include: arguments that such distinctions are 'artificial' or merely historical; various viewpoints in which temperament and character are subcomponents of personality in some way or another; and those that view temperament and personality as being primarily distinguishable in relation to age, with temperament being defined as more biologically determined, providing the basis

from which an individual's personality develops later in life (Rettew & McKee, 2005, p. 15-17; Tackett, 2006, p. 585). Character has a far lesser role in the psychopathology literature than both personality and temperament, and thus does not play any role in the following discussion. Moreover, any distinctions between personality and temperament have little relevance within recent psychopathology literature, with the terms often seen as interchangeable, and literature tending not to differentiate between work concerning personality versus that concerning temperament. This thesis is primarily interested in the relationship between *personality* and psychopathology, but due to the similarity and overlap of personality and temperament studies, the distinction is not preserved here, and work surrounding temperament is incorporated where relevant. In what follows, temperament is encapsulated under the term personality for simplicity and continuity of ideas.

1.1.2 Psychopathology

Psychopathology is generally thought to refer to 'abnormal' or 'disordered' behaviours, cognitions and experiences (Haywood & Raffard, 2017, p. 3), however no single definition is agreed upon. Terms such as abnormal are particularly controversial, and are heavily associated with the medical model, which views psychopathology primarily as the product of dysfunction within the brain (Murphy, 2015, s1). According to Murphy, there is agreement that psychiatry "now adheres to the "medical model"" (2015, s1), and the use of the term 'psychopathology', as opposed to 'mental disorder', 'mental illness' or 'mental difference', appears to be more common in psychiatry and some fields of psychology than in other areas of discourse. A less medical definition is offered by Radden, who describes mental disorder as "deviations from normal thoughts, reasoning, feelings, attitudes, and actions that are by their subjects, or by others, considered socially or personally dysfunctional and apt for treatment" (2019, s0). The

appropriate terminology (e.g. ‘psychopathology’ versus ‘mental disorder’) is contentious, with different terms having different connotations (e.g. ‘psychopathology’ indicates a more medical perspective than ‘mental disorder’). Nonetheless, such terms are often seen as analogous in their main respects. Attempting to best represent the literature that I am engaging with, I mainly use the term ‘psychopathology’ in this thesis.

Individual disorders have a prominent role in understandings and discussions of psychopathology. Some of the more well-known and commonly accepted examples include schizophrenia, bipolar and depression. Many other disorders, such as attention deficit hyperactivity disorder (ADHD), are less clear cut, pushing the boundaries of what we consider to be a psychopathological condition. To some extent, possible conceptualisations of psychopathology are assessed based on the consequences they would have for these various disorders, as well as for well-known ‘non-disorders’. For example, it is important for any definition, conceptualisation or classification of psychopathology to be able to draw a line between depression and grief, and to categorise schizophrenia as a disorder, but not hysteria. What makes those common mental disorders ‘disordered’, while other behaviours, cognitions and experiences are not, remains an unresolved and ongoing question.

As discussed in the introduction of this thesis, the conceptualisation and classification of mental disorder is complex and debated, presenting many unresolved issues. Key conceptual and ontological questions are wide ranging:

- What distinguishes mental disorder from normal problems in living?
- What is the ontological status of mental disorders?
- Are they medical phenomena, socially constructed value judgements, or something else?
- Can mental disorders be latent?
- Is a medical diagnosis a necessary and/or sufficient condition for an individual having a mental disorder?

The issue of the conceptualisation of psychopathology is rarely touched upon in the personality-psychopathology literature. Instead, researchers tend to discuss particular classifications of disorders, such as the DSM, or simply identify individual disorders that they will focus on. Again, as in the case of personality, this in itself is not an issue, but the uncertainty surrounding where the individual studies sit on those many issues outlined above has implications when studying the phenomena and interpreting the findings and discussions. This is discussed in depth towards the end of this thesis.

1.1.3 Distinguishing personality and psychopathology

The distinction between personality and psychopathology may seem obvious. They are not two concepts that are easily confused in everyday discourse. Hallucinations are clearly not a personality trait, and enthusiasm is not a typical symptom of mental disorder. The boundary starts to get blurred, however, when talking about the extreme ends of particular traits and states. It is not always clear at what point, for example, orderliness becomes a symptom of obsessive-compulsive disorder; or how we can tell the difference between one person's susceptibility to stress and another's anxiety. The apparent overlap here is partly what makes studying the relationship between personality and psychopathology so interesting. It does seem that in many cases personality and psychopathology are closely linked. But it is when we come to study that apparent relationship that their distinction becomes particularly important. One issue is that with such inadequately distinguished phenomena, an observed or apparent relationship is hard to interpret (Rettew, 2013, p. 105; Frick, 2004, p. 4). It is possible that any observation or empirical finding of a seemly meaningful link is merely the product of our poorly defined overlapping and conflated concepts. A second issue is that when the language for describing personality traits is so inherently similar to the language used to describe related

psychopathological symptoms or conditions, it is difficult to understand what justifies the assumption that the two concepts really are distinct (Rettew, 2013, p. 105-106). Understanding the assumed and imposed distinction, and what justifies that distinction, is integral to understanding the apparent relationship.

Attempts to distinguish personality and psychopathology can be seen throughout the literature, however these have been heavily critiqued. For example, several researchers have discussed the possibility that the primary difference between the two phenomena comes down to impairment (e.g. Rettew, 2013, p. 106; Lahey, 2004, p. 90; Widiger & Clark, 2000, p. 951). Roughly, this view maintains that behaviours, cognitions, experiences and so forth should be considered disordered so long as they present a clinically significant level of impairment (Widiger & Clark, 2000, p. 951). However, there are many issues with this distinction, such as difficulty of deciding how the presence of impairment is determined, or of accounting for the role of one's environment in determining whether or not they are considered impaired (Rettew, 2013, p. 106-107), and thus many researchers exploring the idea have readily regarded it as problematic.

Perhaps the most central issue here is whether personality and psychopathology are best understood as qualitatively distinct phenomena (differing in kind), or as varying manifestations of a single underlying phenomena (differing in degree) (Rettew, 2013, p. 103; Tackett, 2006, p.588; Frick, 2004, p. 4). Unfortunately, this issue remains largely unresolved. It is not apparent whether or not a resolution to this is required for fruitful personality-psychopathology research, but this will be discussed in more depth throughout the following sections.

1.2 The Apparent Personality-Psychopathology Relationship

1.2.1 Emergence

Some sort of crucial link between personality and psychopathology has been postulated in medical research since at least as early as c. 400 BCE (Maher & Maher, 1994, p. 73; Watson & Clark, 1994, p. 5; Widiger & Smith, 2008, p. 743). Attempts to relate personality to pathology were conspicuously evident in ancient Greek medicine[5], and subsequently, as medical knowledge progressed, many other conceptual theories of the postulated relationship emerged (Akiskal, Hirschfeld & Yerevanian, 1983, p. 801; Maher & Maher, 1994, p. 73; Watson & Clark, 1994, p. 5; Kreuger & Tackett, 2003, p. 109). Although the extent to which personality and psychopathology have been linked in theory and research has varied over time (Watson & Clark, 1994, p. 5; Kreuger & Tackett, 2003, p. 109; Watson, Gamez & Simms, 2005, p. 47), since a revival of interest in the 1990s (Kreuger & Tackett, 2003, p. 109; Watson, Gamez & Simms, 2005), there has been a consistent and expanding body of literature concerned primarily with developing and evaluating empirical evidence in support of the theorised relationship. This revival was largely motivated by the perceived value of preceding conceptual theories, but also by clinical observations that appeared to suggest an important, empirically observable, link between individuals' personality and their presentation of certain psychopathological conditions (Akiskal, Hirschfeld & Yerevanian, 1983, p. 801). A clear focus of this growing body of work has been to draw on statistical methods and empirical data to provide more solid 'evidence' of the theorised and clinically observed relationship. Research has now presented a range of empirical studies that aim to measure the extent to which various personality traits or profiles are related to particular psychopathological disorders.

---

[5] The doctrine of the four humours is often cited as a paradigm example (e.g. Maher & Maher, 1994, p. 73; Rettew, 2013, p. 5; Rettew & McKee, 2005, p. 15; Watson & Clark, 1995, p. 351; Watson & Clark, 1994, p. 5; Widiger & Smith, 2008, p. 743).

1.2.2 The personality-psychopathology studies

Recent empirical studies exploring the link between personality and psychopathology, although plentiful, are notably uniform in both their methods of inquiry and their findings. Basically, such studies examine the level of association between broad trait profiles (or in some cases, individual traits) and specific psychopathological disorders, such as ASD, ADHD, depression or anxiety. The studies' samples are made up of at least two key cohorts – those who have received a recent diagnosis for the relevant disorder(s), or are at least deemed to meet the necessary diagnostic criteria at the time of the study, and a comparison group of people who do not meet those criteria. Various psychological scales are used to gather measures of personality in the samples, and statistical analyses are performed on the data to test for the presence of a relationship between those two key variables. The statistical analyses are predominantly of association and/or variance. Their findings tend to indicate that either high or low levels of certain personality traits or profiles are significantly correlated with particular disorders. For example, low levels of reactive control have been found to correlate with symptoms of ADHD (Martel & Nigg, 2006, p. 1180-1181) and high levels of neuroticism/negative emotionality have been found to have strong to moderate, positive correlations with numerous symptoms of depression (Watson, Gamez & Simms, 2005, p. 61-62). In sum, the numerous empirical studies have consistently reported *systematic* and *significant* relationships between personality traits/profiles and psychopathological symptoms (Tackett, 2006, p. 584; Rettew, 2013, p.101-102)[6].

[6] For examples of such studies, see: Garon, Bryson, Zwaigenbaum, Smith, Brian, Roberts & Szatmari, 2009 (on ASD); Martel & Nigg, 2006 (on ADHD); Watson, Gamez & Simms, 2005 (on depression and anxiety); and Ivarsson & Winge-Westholm, 2004 (on obsessive compulsive disorder (OCD)).

1.2.3 An established personality-psychopathology relationship (PPR)

The 'personality-psychopathology relationship' (henceforth *the PPR*) is now well established as a conceptual tool in psychological discourse. The link between the two phenomena is now generally taken to be axiomatic[7], and although many studies still seek to investigate that link, the field is now moving away from merely trying to validate or empirically 'prove' the presence of a meaningful relationship. Recent empirical findings, alongside the long history of conceptual theories, have led to the recognition that there are important theoretical, clinically observable and empirically supported links between the two phenomena. Those links, however, are complex, with their strength, direction and specificity (i.e. the preciseness of the relationship) varying across traits and disorders. While a multitude of data supports that there is a link between personality and psychopathology, the relationships that appear to hold between the various different traits and disorders are not particularly uniform or clear-cut (e.g. see Watson & Clark, 1995, p. 363). For example, some personality traits and profiles relate to almost all common mental disorders, while others relate to only a specific few; some traits and disorders/symptoms are positively correlated, others are negative, and sometimes both positive and negative trait-symptom associations are seen within a single disorder; and in some cases the associations relate to the presence of a symptom or disorder, while in others they concern the severity. Nonetheless, it is now widely maintained that understanding the PPR will have important implications in both theory and practice, particularly for the treatment (including choosing appropriate options), prevention (including early identification and intervention) and

[7] As evidenced within the work of: Krueger & Eaton, 2010, p. 97; Lo et al., 2017, p. 152; Watson & Clark, 1995, p. 363).

advanced understanding (including aetiology and taxonomy) of mental disorder (Frick, 2004, p. 5; Lahey, 2004, p. 88; Tackett, 2006, p. 593-594; Krueger & Eaton, 2010, p. 97-98; Rettew & McKee, 2005, p. 24).

PART 2: Attempts to study the *nature* of the apparent personality-psychopathology relationship

The idea that personality and psychopathology are *systematically and significantly* related is well-established in psychological and psychiatric research. With both the presence of the PPR and the importance of better understanding it being widely recognised in recent work, an extensive body of literature concerned with studying the relationship's *'nature'* is now developing. That emerging body of literature presents and discusses numerous empirical studies that seek to explore the confirmed relationship between personality and psychopathology. More or less, the focus of those studies is on determining what is going on within the PPR, investigating the structures and/or mechanisms that might underlie or account for the observed relationship, but that will be discussed further in the following sections.

Part 2 of this thesis is an overview and analysis of the work that attempts to study this 'nature' of the PPR. It includes a summary of the general approach towards the study, and a discussion of what it is to be involved with that study. Specific attention is given to the expression 'the nature' of the PPR, as I seek to clarify the intended research project – particularly the aims or focus(es) of inquiry of the collective study. There are two main objectives of part 2.

The first is to outline and review the prominent approach to the study, which I think is best characterised as comprising two key stages:

1) considering potential theories or 'models' of the nature of the PPR (outlined in §2.2);
2) assessing the accuracy or 'fit' of those models (outlined in §2.3).

The second objective is to determine the intended research project. Ultimately, I argue that the focus and scope of the study as a whole is both undefined and unclear:

- what it means to ‘study the nature of the PPR’ is not only vague, but seemingly ambiguous (discussed in §2.1);
- there are distinct possible tasks within the single body of research;
- and these appear to be confused and conflated in the individual studies, and in the study as a whole (discussed in §2.4).

The aim here is to review the work that has been done studying the relationship between personality and psychopathology, and to start to consider the scientific method(s) and conceptual considerations involved in that work. Following on from part 2, part 3 will then provide an evaluation and critique of the study of (the nature of) the PPR, and make suggestions about *what is required for robust and reliable study of the relationship between personality and psychopathology*. Section 3.1 will consider the difficulties and implications associated with the intended research project being inadequately defined, after which sections 3.2 and 3.3 will explore other methodological and conceptual complications involved with studying the nature of the PPR.

2.1 Approaching the study of the nature of the PPR

A large amount of work has been done in the area of studying the nature of the PPR over the past few decades. This has resulted in an abstruse body of literature. In my experience, that body of literature and its associated studies can be difficult to engage with, to the extent that at times it is difficult to figure out precisely what the research is seeking to achieve, and which studies are contributing to a common goal. The aim here is to present an overarching summary of that work and attempt to elucidate its methods of research – specifically what its goals and focus of inquiry are, the methods of study, and how findings are concluded.

2.1.1 The prominent two-stage approach to the study

Several possible theories of the *nature* of the PPR have been presented, re-presented and discussed throughout psychological and psychiatric literature of the past few decades[8]. These theories are generally presented in the form of 'models'[9], which aim to outline possible answers to the question: "what is the nature of the relationship between personality and psychopathology?". The core ideas of these models have developed from various different assumptions and hypotheses that gradually emerged across a range of schools of thought within psychological research (Akiskal, Hirschfeld, & Yerevanian, 1983, p. 801). Around the 1980s to early 1990s, following the resurgence of interest in the link between personality and psychopathology, Akiskal, Hirschfeld and Yerevanian, (1983), Klein, Wonderlich, & Shea (1993) and others began to articulate these long-standing assumptions and hypotheses as a set of potential models of the nature of the PPR. Together, these models now tend to be considered a plausible and comprehensive set of possibilities. Over time, they have been developed, refined and expanded and have continued to be extensively discussed.

The consideration of the set of proposed models comprises a key stage in studying the nature of the PPR. Research papers on the topic typically begin with considering possible answers to the question at hand by stating, reviewing or developing the existing possible theories (viz. the models), then turning to empirical methods of research to evaluate or compare them. Such empirical methods make up a second key stage in studying the nature of the PPR. Generally speaking, this involves the collection of observational or self-report data for a sample

[8] The vast majority of literature of this sort has been published since approximately 1980.

[9] Both what the 'models' are and what is meant by 'model' are discussed further in section 3.2.

of subjects, focusing on the history or presentation of specific traits and disorders. Statistical tests[10] are used to analyse the data, and the results are framed with respect to the aforementioned set of models. That is, the statistical results are considered support for or against the given theories of the nature of the PPR, with the accuracy or 'fit' of the models assessed *against* the empirical evidence.

In general, the prominent approach to studying the nature of the PPR can be characterised as being comprised of those two major stages. The approach roughly aligns with a hypothetico-deductive method of scientific inquiry, in which hypotheses are formulated and empirical observational research is used to test the hypotheses (Haig, 2014, p. 7).

The first stage – the consideration, development and amendment of a range of models (which will be discussed in §2.2) – is primarily hypothesis based. The models are considered 'conceptual models' (Durbin & Hicks, 2014, p. 363). Their postulation is founded on theory, and while their development is informed or influenced by other measures – such as statistical findings, clinical observations, prior understanding, knowledge and experience, and so forth – conceptual soundness is of primary importance.[11]

The second stage, to 'test' those conceptual models, is founded on empirical work. In this stage, coherence and consistency play a role, but assessing the accuracy or validating the 'fit' of a model based on empirical observation is the priority.[12] An overview of the ways in which the conceptual models are commonly 'tested' is presented in section 2.3.

---

[10] Discussed further in section 2.3.

[11] Inferred from the work of: Durbin & Hicks, 2014, p. 363-366; Kreuger & Tackett, 2003, p. 110-120; Rettew & McKee, 2005, p 20-23; Tackett, 2006, p. 588-593, Rettew, 2013, p. 108-118.

[12] See previous footnote.

This two-stage conceptual-empirical approach, as outlined above, is the focus of this thesis. It has dominated the literature on the topic over the past few decades and continues to be prominent in ongoing discussions and research studies.

2.1.2 The research project

While the general approach to studying the nature of the PPR is reasonably clear and consistent, articulating the precise research project (particularly the aims, scope and focuses of inquiry of the study) is not so straightforward. To my knowledge, none of the relevant literature ever sets out a clear and unambiguous research question, or a description of precisely what they are trying to investigate by carrying out the study. Beyond stating that the study is on 'the nature' of the observed relationship between personality and psychopathology, little clarificatory information about the study's focus or objectives is provided. And unfortunately, what is meant by 'the nature' of the PPR is non-specific and not inherently informative. For instance, without further specification, attempts to investigate the nature of the PPR may be best understood as being about any or all of the following: causal processes; predictive relationships; associations or covarying patterns. Some of the literature gives the impression (or directly states) that 'the nature' refers to *how* personality and psychopathology are related[13], yet other literature does not establish that link[14]. Considering *the nature* of the relationship between personality and psychopathology is not *consistently* seen as interchangeable with considering *how* personality and psychopathology relate. Moreover, even if we were able to specify that 'the nature' directly refers to 'how' the phenomena relate, this does not fully resolve the ambiguity of the statement.

[13] E.g. De Pauw and Mervielde, 2011, p. 279; Kopala-Sibley, Klein, Perlman & Kotov , 2017, p. 1030-1031.

[14] E.g. Rettew and McKee, 2005, p. 22.

To illustrate this, consider a scenario in which someone reports a relationship between belief B and value V. If we inquire *how* they are related, we could get many different *types* of answer. We might find out about:

- the associated or covarying surface patterns or the way the phenomena jointly present – for example, it could be that most of the change observed in B is accounted for by change in V, or that a certain strength of B is strongly linked with the presence of V;
- the temporal patterns – it may be that people with V tend to develop strong B after a certain amount of time, or that people only ever hold B if they previously had V;
- the causal processes – in which case, we may find out about how B results in neurostructural changes that lead to V, or about how some other variable, X, is responsible for the presence of both V and B, or how people with V interact with the world in such a way that they are exposed to things that lead them to develop B.

Now of we were to inquire about the *nature* of the relationship between B and V, it seems even less clear what *type* of answer we may be after. We may want to know one of those things above, but we also might be asking about why we observe the relationship. Again, that could be a question of mechanisms and causes of the two phenomena, but it also might be that the definition of V entails or requires that you must present with B in order to meet the criteria for having V; or that B and V are just different presentations or degrees of a single phenomenon.

Presumably we could inquire into other aspects of the relationship when we seek to determine its nature, but the hypothetical list quickly gets long. The point is that there are many different *types* of interest one could have when inquiring about the nature of the relationship. The underlying, and most problematic[15], issue here is that the recent body of literature

[15] Later, in part 3, I argue that this lack of clarity is problematic, underpinning and intertwined with several methodological and conceptual issues within recent attempts to understand and explain the nature of the apparent personality-psychopathology relationship.

concerning the nature of the PPR is, on the whole, vague as to whether the study is focused on the ***descriptive*** *surface patterns* of the relationship or the *underlying* ***explanatory*** *mechanistic processes* (discussed further in §2.4). Consequently, there is a significant level of ambiguity involved in discerning precisely what is being undertaken when academics purport to be 'studying the *nature* of the relationship' between personality and psychopathology.

2.1.3 The difficulty in discerning a single clear research project

It is uncommon in scientific inquiry to have an extensive field of research without a clear understanding of exactly what is being studied. That is not to say that the research question must always be well defined, or even that the possible outcomes of the study, need to be known from the outset. But what *is* important, is a clear understanding of what it is to be engaged with that research – that there is an unequivocal focus of inquiry, with reasonably clear criteria for success.

Of course, that sort of clarity can be achieved by precisely stating the research project – outlining the study's aims and objectives, without ambiguities or vague descriptions. But it could also be achieved just with consistency across the relevant studies and literature. Specifically, if the possible theories and outcomes of the research (in this case the models) all pointed towards a single research question, or all appeared to be possible answers to a common question (all one *type*), and the methods employed to perform the study all pointed towards a clear single goal, then the lack of an explicit research project would not be an issue. However that is not the case within studies of the nature of the PPR. The ambiguity that results from there being no consistent and unambiguous statement of the study's precise focus of inquiry – no *explicit* specification – is perpetuated by there seemingly being no clear *implicit* specification either. What I mean by no "implicit specification" is that analysing the prominent

approach to the study doesn't allow one to deduce a consistent and unambiguous understanding of the specific intended goal. This lack of specification (explicitly or implicitly) is referred to in the following as the lack of a precise or clear *research project*[16].

In what follows, I demonstrate the fact that neither the models nor the methods of testing those models elucidate a clear and succinct research project. I first present the models (§2.2) and methods of testing them (§2.3), and then (§2.4) return to discussing how understanding the approach of the research does not help to narrow the possibilities (outlined above) of what 'the nature' might be referring to. Thus, the research project of the study is both vague and ambiguous. I propose that two potential tasks are involved, one concerning description, the other concerning explanation. I illustrate how recent attempts to study and understand the nature of the PPR have, in general, been a mixed bag of such projects with their intentions very rarely being clearly distinguished, or even easily separable.

2.2 Stage 1: the proposed potential models

The theorised 'models' of the nature of the PPR are a major component of recent work. The models are *conceptual*; they provide "relatively idealised" conceptualisations of what the nature of the relationship might be (Durbin & Hicks, 2014, p. 366). Over time, they have formed the theoretical basis from which the recent empirical work has emerged, and they are generally taken to provide a set of testable hypotheses which can be supported or disconfirmed

[16] I'm not sure if this terminology is common, or strictly correct, but 'research project' is used here to denote a study's scope and direction – its focus(es) of inquiry, precise intentions or goal, criteria for success and so on.

by individual studies. As already discussed, consideration, development and revision of these models comprises the first of two key stages in recent attempts to study the nature of the PPR.

Roughly speaking, a 'model' in this context is an attempt at *representing* the nature of the PPR. There is an observed relationship between personality and psychopathology, and each of these models provides a different representation of what might be going on. Being more specific about what the models are representing is difficult, because what is meant by 'the nature' of the PPR is (as noted in §2.1) non-specific and it is not inherently informative or well defined within the literature. Different types of scientific models perform different functions (Frigg & Hartmann, 2020 s1). Some relevant examples include:

- phenomenological models, which "only represent observable properties of their targets and refrain from postulating hidden mechanisms" (Frigg & Hartmann, 2020 s1);
- different types of explanatory models, which represent causal or explanatory features that "produce, underlie or maintain" the target (Craver & Tabery, 2015, s3);
- and mechanical models, which provide descriptions "of the mechanism's behaviour [and] … of the mechanism that accounts for that behaviour" (Craver & Tabery, 2015, s3).

Again, because the target – 'the nature' of the PPR – is not clear, it is unclear what type of function or combination of functions, these models are thought to perform.

2.2.1 The theoretical models

Throughout recent literature many different models, and variations of previously existing models, have emerged. The most common and widely accepted of these are often referred to as the *theoretical models*[17]. The theoretical models are typically presented as a unique set,

[17] Terminology used in: Tackett, 2006, p. 594; Durbin & Hicks, 2014, p. 363.

treated as providing a reasonably complete or exhaustive range of possibilities about the nature of the PPR. Depending on the author discussing them, the number of models stated as members of this set seems to range from anywhere between three and seven, and the names and descriptions of the models often differ slightly. Nonetheless, the content of the set of theoretical models is interpreted as being largely consistent across the literature. The central defining claims of each model are relatively fixed regardless of who is discussing them, and the divisions drawn between the models (basically, whether the theoretical models are defined as a set of 3 models, 4 models, etc.) make much the same distinctions between the possibilities, just to different degrees. An example of this is that one author may consider there to be a group of six models, while another author may instead characterise this as three models, each with two possible variations. Below, I outline what seem to be the four, core theoretical models, and use a simplistic example of the relationship between Autism and extroversion to demonstrate the main ideas of each model.

The predisposition model

The 'predisposition' model, sometimes called the 'vulnerability' or 'risk' model postulates that personality traits (or dimensions) and psychopathological symptoms differ qualitatively – they are different *entities*, but particular personality traits work as predisposing factors for the future development of certain forms of psychopathology (Rettew, 2009, p. 3438; Tackett, 2006, p. 588; Rettew, 2013, p. 112; Rettew & McKee, 2005, p. 22). That is, certain personality traits make it more or less likely that an individual will develop certain psychiatric disorders. If the predisposition model were to apply to Autism and extroversion, then individuals with low extroversion might be more likely to develop Autism than individuals with high extroversion.

This model is sometimes said to theorise that personality traits are "antecedents and predictors" of psychopathology (Kopala-Sibley, Klein, Perlman & Kotov, 2017, p. 1030); but

others describe it as postulating that personality traits "play a causal role in the development" of psychopathology (Clark, Watson, & Mineka, 1994, p. 103). In that case, particular levels of extroversion might cause individuals to develop Autism.

The complication model

The 'complication' or 'scar' model also postulates that personality traits (or dimensions) and psychopathological symptoms are distinct entities that differ qualitatively, but that psychopathological symptoms can modify premorbid personality traits (Rettew, 2009, p. 3438; Rettew, 2013, p. 114; Tackett, 2006, p. 588; Clark, Watson, & Mineka, 1994, p. 103). For example, Autism might change the mental state or neurology of individuals in a way that leads to changes in how introverted or extroverted they are.

This model has been described as making similar predictions as the predisposition model, but with the aetiological causal role working in the opposite direction (Rettew & McKee, 2005, p. 22).

The pathoplasty/exacerbation/bidirectional model

According to the 'pathoplasty' or 'exacerbation' model, personality traits can modify the expression or course of psychopathological symptoms (Rettew, 2009, p. 3438; Clark, Watson, & Mineka, 1994, p. 103). They influence the manifestation of a disorder in "course, severity, presentation, or prognosis" (Tackett, 2006, p. 588).

This model has various different ways of being understood within the literature. Rettew (2013) defines the 'patholpasty' or 'bidirectional' model as "a hybrid of the scar and risk models" where "the direction of causality can work in either direction, depending on the disorder, the trait of even the individual" (p. 117). In this case, a child might be at risk of developing Autism due to their particularly introverted personality, and after developing

Autism later in life, that disorder has an influence with the way they interact with the world, causing them to become even less extroverted.

The continuum/spectrum model

The 'continuum' or 'spectrum' model holds that there are no qualitative distinctions between personality traits (or dimensions) and psychopathological symptoms (Rettew, 2009, p. 3437) – they share a common continuum, and a psychopathological symptom is thus an extreme (high or low) manifestation of a particular personality/temperament trait (Clark, Watson, & Mineka, 1994, p. 103; Rettew, 2013, p. 109). According to this model, it might be that Autism is simply an extreme level of particular traits (e.g. extroversion) and mental states. They are ultimately variations of the same thing: at certain extreme levels we move away from considering something an instance of a personality trait and instead classify it as a symptom of a disorder.

This model is often described as postulating a "common etiology and underlying neurobiology" between personality and psychopathology (Rettew, 2009, p. 3437).

2.2.2 Other developed models

While there are many different iterations of the set of theoretical models, the basic main points are universal. They make up the core set of considered possibilities of the nature of the PPR. Other models have also been developed that generally fall within one of two categories: 'structural models' and additional variable models.

Additional variable models

What I refer to as 'additional variable' models are ones which introduce one or more variable(s), aside from personality and psychopathology, in an attempt to model the nature of

the PPR. These are becoming common in more recent literature (i.e. post 2010), and are not always coined ‘models’, although they closely align with the other models in the literature. Examples of additional variable models of the nature of the PPR include:

- an evolutionary model, in which individuals’ “life history strategies” play a central role in the relationship between personality and psychopathology (Hengartner, Tyrer, Ajdacic-Gross, Angst, & Rössler, 2018, p. 450);
- several developmental models that consider the role of age-related norms, developmental context, history of disorder and developmental pressures, to create models informed by a “developmental framework” (Durbin & Hicks, 2014, p. 382); and
- stressful life events models that suggest the occurrence of significant events in individuals’ lives, such as job loss or partnership separation, play an important role in the observed PPR – the role of the stressful live events is normally represented either as influencing personality (which influences psychopathology) or by having a joint influence with personality on psychopathology (Kopala-Sibley et al., 2017, p. 1030-1031; Hengartner et al., 2018, p. 443).

While the additional variable models provide more complex and developed possibilities than the aforementioned theoretical models, they nonetheless roughly align with, or fall under, the main claims of the theoretical models. For example, the evolutionary model can be seen as a variant of the common cause model, with life history strategies constituting a core component of both psychopathology and personality. The developmental models are described by Durbin and Hicks as “modifications” of the traditional theoretical models (2014, p. 382), and the stressful life events models can be directly mapped onto the theoretical models – in fact, Kopala-Sibley and colleagues present the models as common variants of the well-known set of theoretical models (Kopala-Sibley et al., 2017, p. 1030).

Structural models

Structural models play a slightly different role in representing the possibilities of the nature of the PPR. Specifically, structural models organise or 'structure' personality traits and psychopathological conditions. A prominent example of a structural model in psychology is the 'Big Five' model of personality. Commonly, structural models are focused on the structure of one construct (e.g. personality *or* psychopathology). Yet their relevance here is the role they have in attempting to theorise and understand the ways in which personality and psychopathology relate. *Structural models of personality-psychopathology relations* model covariance and degrees of association and between traits (personality) and disorders (psychopathology) (Durbin & Hicks, 2014, p. 364). An example[18] is the internalising-externalising model, in which psychopathological conditions are structured in a way that aligns with their personality correlates, with mood and anxiety disorders organised under the label 'internalising' and antisocial behaviour and substance use disorders organised under the label 'externalising' (Krueger & Tackett, 2003, p. 121-122). According to Krueger and Tackett, this model fits within the theoretical spectrum model, as the "labels internalizing and externalizing refer to broad but coherent spectra of personality and psychopathology that transcend [common rigid] distinctions" between the two phenomena (2003, p. 122).

In general, these structural models are closely interwoven with the theoretical models. Importantly, structural models are restricted to patterns of cooccurrence and covariation between personality and psychopathology; unlike other models, they are generally thought of as not attempting to represent the processes or mechanisms of the PPR (Durbin & Hicks, 2014, p. 364). Nonetheless, discussions of structural and theoretical models regularly overlap and

[18] For further examples, see: Watson, Gamez & Simms, 2005, p. 50-52; Watson, Clark & Harkness, 1994, p. 26.

blur into each other[19]. Whereas I have made a sharp distinction between theoretical and structural models, this is often not the case in the literature.

Comparing the different types of model

As mentioned above, the theoretical models are the most commonly considered set of possibilities of the PPR. Yet these other (third variable and structural) models are intertwined with discussions of the theoretical models. The structural models in particular are interesting in that they are not thought of as making causal claims. Thus, they confuse the ability to deduce a clear focus of the PPR study as they do not make causal claims, but they are closely related to, and often not distinguished from, the theoretical models which, as illustrated above, are sometimes characterised as making causal claims and other times are not. I have made sharp distinctions between the different 'types' of model, but that isn't particularly representative of the way they are discussed in the literature. To my knowledge, all of these 'other' developed models fall within consideration of the theoretical models, and thus the following discussion considers all these models when discussing studies of the PPR.

2.3 Stage 2: the empirical statistical research

Statistical testing of empirical data is the dominant method of studying the nature of the PPR. In so far as the theoretical models (above) are seen as providing testable hypotheses, this second stage of the study involves testing those models in an attempt to determine their accuracy. Researchers draw on empirical evidence to assess the fit of the representations theorised by the

[19] See for examples: Watson, Clark & Harkness, 1994, p. 29; Krueger & Tackett, 2003.

models. The idea that at least some empirical support has been provided for each model is widely reported (e.g. Tackett, 2006, pp. 589-593; Krueger & Tackett, 2003, p. 112; Watson, Gamez, & Simms, 2005, p. 62; Watson & Clark, 1994, p. 4), although many also express doubt that unique support for a single model has been achieved (e.g. Durbin & Hicks, 2014, p. 366). The methods of these empirical studies are largely consistent across the body of research. Some studies are designed specifically to test a single model (e.g. De Pauw, Mervielde, Van Leeuwen, & De Clercq, 2011), while others take a more overarching approach (e.g. Clayton, Ernst, & Angst, 1994). Individual studies tend to investigate relationships between *specific* disorders and traits, but work has also been done around the links between *multiple* disorders and/or multiple traits (Krueger & Tackett, 2003, p. 112), supposedly aiming to gain a perspective on personality and psychopathology more generally. While the studies aren't identical, the tools and methods of research are notably consistent. This section (§2.3) provides a brief, technical overview of the empirical and statistical methods of these studies.

2.3.1 Empirical data collection

These studies of the nature of the PPR work with empirical data. In order to evaluate the theoretical models, relevant evidence is gathered from samples of human subjects. The type of information gathered varies between studies, but examples include variables such as medical and clinical history, school reports, history of critical life events, general demographic information, and measures of personality and psychopathology. Collecting data on complex phenomena such as personality and psychopathology is difficult. A large portion of the studies assess personality by means of self-report (Klein, Kotov, & Bufferd, 2011, p. 276), normally by completing a questionnaire. Other methods of assessing personality in the studies are informant report (where the informant is someone such as a parent of the participant),

observation (either clinical or naturalistic) and interviews. The prominent method for assessing psychopathology appears to be semi-structured interviews and/or clinical assessments. The type of psychopathology data often gathered relates to the presentation, development and severity of the disorder and its symptoms. For representative examples of data gathering methods see De Pauw et al. (2011), Clayton, Ernst and Angst (1994) and Hengartner et al. (2018).

The tools or 'instruments' of measurement for personality and psychopathology are often, but not always, personality inventories and diagnostic criteria (respectively). Examples of personality inventories used are the Freiburg Personality Inventory, the Eysenck Personality Inventory and the Hierarchical Personality Inventory for Children (Hengartner et al., 2018, p. 446; Clayton, Ernst, & Angst, 1994, p. 340-341; De Pauw et al., 2011, p. 202). These inventories comprise a set of 'items' (or questions) that are thought to ascertain information about peoples' personality profiles. The DSM is regularly used to provide the diagnostic criteria for measuring the presence of psychopathology. Sometimes other data pertaining to psychopathology, such as severity or symptom type, is measured according to DSM criteria as well. In other cases, however, separate instruments are used to gather more detailed data about the subjects' psychopathological conditions[20].

To my knowledge, all studies within the field to-date have measured both personality and psychopathology by relying solely on surface level manifestations of the phenomena. That is, while the two phenomena are widely thought to exist as more than mere outward presentations (e.g. behaviours), the methods of measurement within the studies focus solely on outward, directly observable or self-reported features, such as behaviours, mental states, traits

[20] For an example, see De Pauw et al., 2011, p. 201-202, who use the Social Communication Questionnaire to gather information on the autistic symptoms presented by participants.

and symptoms. The studies' measures are of surface level manifestations of the phenomena, as opposed to the underlying, internal, structures or processes.

Different theories and research present different ideas about exactly what personality and psychopathology are and how they should be understood, but a common view is that the phenomena are composite – they are made up of various different elements (some internal elements, e.g. genetics and neurobiology; some surface level, e.g. behaviours and mental states)[21]. Thus, it is important to note here that regardless of how personality and psychopathology are best conceptualised, within studies of the nature of the PPR, the measures of the phenomena capture only the surface level outward or observable manifestations.

2.3.2 Statistical tests and analyses

Statistical tests are used for data analysis, with various different types of test employed across the body of research. In many studies, multiple tests are run and reported. While the details of the individual tests aren't relevant here, it is useful to understand the types of analysis that are implemented, and the way in which the results are used to assess the theoretical models of the nature of the PPR.

A quick review of the literature shows the prominent role that *regression analyses* have in testing the empirical data – multiple regression in particular[22]. Multiple regression assesses the relationship between a dependent variable (the outcome variable; the thing that the researcher is trying to predict or explain) and multiple independent variables (variables that may predict or explain the dependent variable) (Allison, 1999, p. 1). For example, the analysis

[21] See, for example, Radden, 2019, s3.1.

[22] A few examples of the use of multiple regression for analysing data on the nature of the PPR include: De Pauw et al., 2011; Hengartner et al., 2018; Garon et al., 2009; De Pauw & Mervielde, 2011.

could be used to assess how well a dependent variable such as level of education is explained by two independent variables, e.g. age and income. Regression is used for both prediction and causal analysis. Very roughly, regression works by fitting a model (an equation) that predicts the dependent variable *with* the independent variable(s), in a way that reduces the errors of prediction to be as small as possible (Allison, 1999, p. 3-16). Regression is closely related to correlation analyses, t tests and analyses of variance (or covariance), all of which are seen in recent attempts to study the nature of the PPR. Notably, the statistical tests used to assess the empirical data are analyses of associations, covariances, mean differences and so forth. An important feature of regression analyses in particular is that, arguably, they enable researchers to 'control for' individual independent variables in order to measure the *effect* of other independent variable(s) of interest on the dependent variable (Allison, 1999, p. 16-17). For example, regression enables us to hold the variable 'age' fixed, in order to look at the effect that income has on level of education *after accounting for age*.

Another prominent statistical theme in the literature is the use of longitudinal studies. In order to better assess the interaction between personality and psychopathology, and specifically their interaction over time, researchers use longitudinal data, analysing repeated measures of a single group of subjects over an extended time-period. The use of longitudinal studies allows for a more comprehensive understanding of the observed data patterns, and is widely reported as being an important tool for inferring causal relationships (De Pauw et al., 2011, p. 210).

2.3.3 The use of test results to analyse the set of potential models and draw inferences

The hardest to summarise but perhaps most important aspect of the empirical studies is the way in which the statistical results are linked back to the models – specifically, the way that the

models are ‘assessed’ or evaluated using the empirical findings. The most important feature to recognise about this process is the *type of link* that is drawn between the empirical statistical evidence and the conceptual theoretical models: evidence of statistically significant *patterns* occurring between the presentations of personality and psychopathology is taken to provide *direct, empirical support* for the *individual* theoretical models. Which model is endorsed by the research depends on the type of pattern observed. For example, regression results from a longitudinal study that finds psychopathology to be a good predictor variable of personality changes later in life is taken to provide support for the complication/scar model (e.g. Tackett, 2006, p. 589; Shiner, Masten, & Tellegen, 2002, p. 1172). Measures of personality before and after the onset of the psychopathological condition are thought to be required in order to show that psychopathology *causes* the observed change in personality over time (e.g. Tackett, 2006, p. 589). Another example is statistical findings that mean-level differences account for the differences in disordered and non-disordered populations are reported as evidence for the spectrum model (Rettew, 2013, p. 118-119; e.g. De Pauw & Mervielde, 2011, p. 286).

The point here is that individual findings based on observational and self-report data and statistical tests (mostly ones of association) are reported as giving direct evidence of a particular account of the PPR. Rather than being noted as associations or other patterns that might provide relevant pieces of information about the PPR, they are reported as evidence supporting or challenging the ‘correctness’ of a specific model.

2.4 Discerning the research project – explanation versus description

Section 2.1 raised the observation that what ‘the nature’ of the PPR might mean is vague and ambiguous, and the precise research project is not well specified within the body of literature.

I explained that the literature lacks a consistent and unambiguous statement of the study's precise focus of inquiry, but I also claimed that close analysis of the body of research doesn't paint a clear picture of what it is to be involved with that research. Having provided a brief overview of the general approach to studying the nature of the PPR, I now aim to show that referring to the methods of recent research does not help to elucidate a single succinct research project. That is, there doesn't appear to be one clear *implicit* research project within the body of work. In fact, analysis of the literature points towards *two* potential and apparent goals. Specifically, recent attempts to study 'the nature' of the PPR involve both *descriptive* and *explanatory* goals[23]. These descriptive and explanatory tasks are two importantly distinct focuses of inquiry, which are deeply intertwined within the single body of research.

2.4.1 Descriptive and explanatory goals inherent in the literature

Determining whether the goal is one of describing or explaining the observed relationship is central to understanding what is meant by 'the nature' of the PPR[24]. Both seem to be plausible clarifications of what studying the nature of the PPR actually involves, but what the studies would seek to achieve under each of those goals fundamentally differs.

On one hand, 'the nature' of the PPR could reasonably be interpretated as being about *description*. Studying the nature of the relationship in this sense would involve examining the way in which the PPR presents. The focus of inquiry would be the observable patterns,

[23] What I mean by 'description' and 'explanation', particularly in the context of PPR research, is discussed in the upcoming section (§2.4.1).

[24] I am including prediction under the task of describing. Although a distinction is often made between predictive and descriptive models, in the case of studying *the nature* of the PPR, the key distinction lies in the difference between describing patterns versus explaining processes of the relationship (this is discussed further in what follows).

including correlations, temporal associations, variances, dependencies and so forth. To describe the observed relationship between personality and psychopathology would be to investigate and represent the details of their complex joint presentation.

Alternatively, 'the nature' of the PPR might just as reasonably be interpretated as being about *explanation*. In this case, studies would be focused on determining what accounts for the observed relationship – what is the 'reason' for the observed relationship, or what is causally or 'aetiologically' responsible for the way in which personality and psychopathology relate? To explain the nature of the PPR would be to provide an account of the mechanisms or processes that underlie that relationship.

Descriptive or explanatory studies would (in theory) each give rise to very different research questions, methods and findings. But not only do description and explanation seem to be reasonable interpretations of what 'the nature' of the PPR might refer to, trying to determine their role within the recent body of work is difficult. Aspects of both descriptive and explanatory goals are inherent in the models, tests and inferences that are discussed and implemented across the various individual studies of 'the nature' of the PPR.

In the models

The models of the nature of the PPR display evidence of both descriptive and explanatory elements at play in the study. Attaining an overarching perspective of the various proposed models, and their various ways of being presented and discussed throughout the literature, gives the most prominent indication that both goals are inherent in the studies' methods.

The set of possible theoretical models are outlined and discussed by many different researchers throughout the body of literature. While their statements of the central defining claims of the models remain rather consistent across all of those discussions, the details of the models aren't so uniform. To start with, it is important to note that some researchers explicitly

state that the models are attempts to explain the causes of the PPR[25], while others make no such claims[26]. Some have made a distinction between causes of the relationship and the nature of the relationship[27]; others refer to the models as theories of the possible mechanisms[28]; and in some cases[29], such as within discussions of structural models, it is directly stated that the theories refrain from making statements of explanation. Moreover, in some instances, the terminology used to discuss the set of models is indicative of an *explanatory focus*, while in others, the discussions are restricted to much more *descriptive language*. Descriptive language is found in cases where the models are discussed with terminology focused on *patterns*. For example, they discuss the possibility that personality is an "antecedent" or a "predictor" or "precursor" of psychopathology (Kopala-Sibley et al., 2017, p. 1030), or that one puts an individual at "risk" of the other, or that the manifestations of personality and psychopathology differ in "degree" (Rettew, 2013, p. 103 & 112). Others use explanatory terminology centred around *causes*. For example, many papers consider whether personality "plays a causal role" in the development of psychopathology (Widiger & Smith, 2008, p. 761; Clark, Watson, & Mineka, 1994, p. 103), or whether they share a common "aetiological factor" (Watson, Gamez, & Simms, 2005, p. 62; Watson & Clark, 1995, p. 361). This mixed terminology used to articulate the set of models indicates that the models aren't uniformly seen to be strictly *descriptive* or *explanatory* in their function.

Similarly, the content postulated within the models appears to be a mix of descriptive and explanatory theories or accounts. The models (across the whole body of literature) are

---

[25] E.g. Tackett, 2006, p. 588.

[26] For example, some discuss them as attempts to *characterise* the interrelationship, making no mention of explanation or cause (Kopala-Sibley et al, 2017, p. 1030).

[27] E.g. Tackett, 2006, p. 584.

[28] E.g. Rettew & McKee, 2005, p. 22.

[29] E.g. Akiskal, Hirschfeld & Yerevanian, 1983, p. 804.

taken to make claims about the *types of associations and variations* (patterns) that occur between personality and psychopathology, as well as claims about *the causes or mechanisms* of their relationship. This is best illustrated in the summary table provided by Durbin and Hicks, in which the main claims made by the theoretical models are summarised. The important thing to note here is that the summary identifies the predictions made by the models as being about *surface patterns, predictions and causes*.

Table 1. Predictions of traditional personality–psychopathology models

Predictions	Spectrum/continuity	Precursor/prodrome	Common cause	Predisposition	Pathoplasty	Concomitants/state	Scar
Magnitude of trait–disorder associations	Very large	Smaller than in spectrum model; larger than in predisposition model	Smaller than in spectrum and precursor models	Smaller than spectrum, precursor, common cause models; depends on the proportion of cases with the predisposition	Unrelated to onset, but potentially large associations for predicting course	Unclear	Unclear
Trait and disorder have the same correlates and outcomes	Yes	Some	Some, but accounted for by shared etiological factors	No	No	No	No
Trait and disorder have the same causes	Yes	Yes, but additional causes contribute to the disorder	Yes, ranges from sharing some to all causes	No	No	Perhaps	No
Those with the disorder all have extreme trait levels	Yes	Yes	No	Yes, if there is only 1 trait predisposition and most with the disorder had the predisposition	No	Maybe	Unclear
Extreme trait level predicts time to disorder onset	No	Yes	No	No	No	No	No
Controlling for common causes eliminates trait–disorder associations	Yes	Yes	Yes	No	No	Unclear	No
Trait scores become more extreme when symptoms worsen	Yes	Maybe	Yes, if common causes operate to influence course of the disorder	No	No	Yes	No
Trait scores predict symptom severity, course, or treatment response	Unclear	No	Maybe	No	Yes	No	No

Figure 1: Table of "Predictions of traditional personality-psychopathology models" (from Durbin & Hicks, 2014, p. 367).

Returning to the earlier discussion about determining exactly what the models are representing (§2.2), it remains difficult to narrow the scope. The function that the models of the nature of the PPR are intended to perform appears to vary across the literature. Some presentations and discussions of the models point toward a phenomenological function, with the focus on representing the observable patterns and properties of the relationship, while others appear to be explanatory or mechanical, focusing on the mechanisms and causal/explanatory features that account for the relationship. Moreover, in some instances, the models are portrayed as performing each of those different functions concurrently (e.g. in Durbin & Hicks, 2014, p. 367).

Thus, the articulations and discussions of the models, when considered altogether, point towards both descriptive *and* explanatory aspects within attempts to study the nature of the PPR. The terminology used to discuss the models, and the predictions that they are said to articulate, both demonstrate this.

In the empirical research

A similar situation can be seen in the empirical research, although it is not so overt. The types of statistical analyses used across the body of research don't clearly belong to a descriptive or explanatory goal, nor do the inferences and discussions that emerge from the findings of those analyses.

Many of the statistical analyses used to study the nature of the PPR are ones that produce statistics identifying structural patterns (e.g. co-occurrences, variances, mean differences), such as ANOVAs or Wilcoxon Tests[30]. The findings from these sorts of analyses would aid only in attempts to *describe* the relationship. For example, the finding from De Pauw and Mervielde (2011, p. 282-286) that there are substantial differences in the means of particular traits when comparing ADHD versus control groups identifies a *structural* pattern that contributes to a *description* of the relationship between the traits and disorder. Structural patterns such as differences in group means, no matter how strong or significant, don't alone provide explanatory data. Yet there are also indications that regressions, longitudinal studies, and similar analyses[31] are being used in an attempt to control for specific variables in order to understand what accounts for the relationship. Some of these methods are more indicative of an explanatory goal. It is thought that such studies' findings could provide evidence about what

[30] See, for examples, Clayton, Ernst and Angst, 1994 and De Pauw & Mervielde, 2011.

[31] See, for example, Hengartner et al., 2018, p. 446-447.

causes the observed relationship between personality and psychopathology, and what mechanisms underlie that relationship. For example, several researchers (e.g. Watson & Clark, 1995, p. 361-361; Watson, Gamez & Simms, 2005, p. 62-63) have reported that statistical studies (such as that of Clayton, Ernst & Angst, 1994) have produced findings that give evidence of *causal* or *aetiological* factors causing the relationship.

Researchers who present summaries of the research in the field, or who draw on other's results in order to support a claim they are making, sometimes present those results using causal, aetiological and explanatory language[32]. However, within the original studies, when researchers report the results of their tests, they tend to refrain from making such claims, instead using purely non-causal and descriptive language[33]. The fact that researchers do not seem to draw causal conclusions from their own studies gives further reason to believe that the research should not be simply construed as an attempt to explain, as opposed to describe, the PPR.

To summarise, aspects of both descriptive and explanatory goals are apparent in the methods of the studies of the nature of the PPR. Although what 'the nature' of the relationship refers to is not well defined, both description and explanation have a central role.

2.4.2 Description and explanation interwoven within attempts to study the nature of the PPR

It is not unusual for a body of literature to engage with both descriptive and explanatory tasks, and I am not trying to suggest that research must be about only description or only explanation. Attempts to describe and explain a phenomenon are closely related, and it is absurd to say that

[32] For examples: Watson & Clark, 1995, p. 361-361; Watson, Gamez & Simms, 2005, p. 62-63.

[33] One example is Hengartner et al., 2018, p. 449-450. There are many other examples, but Hengartner and co. are a good example as they explicitly state that their findings fall short of providing robust causal evidence.

they must be kept separate, or that only one task can be achieved within a single body of research or literature.

The noteworthy point here is how deeply interwoven and muddled those two tasks are within the present body of work. Studying 'the nature' of the relationships appears to involve two different focuses of inquiry – one concerned with describing the relationship, the other with explaining it. But those two tasks are so intertwined within the single body of literature that, not only is it hard to determine what is going on (i.e. to determine that both description and explanation are in fact focuses of inquiry), but there isn't really any delineation between the two – the boundary between description and explanation is blurred.

Interestingly, the distinction between seeking to describe versus explain the PPR has been acknowledged within the literature. Rettew, for example, recognises that we can consider the relationship between personality and psychopathology "*both* at a surface level of description *and* with regard to their underlying neurobiology" (2013, p. 84, my emphases). Similarly, Nigg emphasises the importance of pursuing both the study of the aetiological mechanisms underlying the apparent relationship, and the study of the structural patterns of the relationship (2006, p. 407). Durbin and Hicks further recognise that different methods are required for studying "illuminating processes, rather than patterns of covariance" (2014, p. 364). Nonetheless, that distinction is not conformed to with the study as a whole, and in some cases, within the individual studies themselves.

As was evident in the preceding discussion about both descriptive and explanatory goals being evident in the models and empirical studies, there is no solid divide between the two projects. It is difficult to summarise, however, the extent of the mingling that has developed over time. Above, I gave examples of various cases from the literature which, altogether, illustrated the presence of both descriptive and explanatory goals, methods and claims being involved within the single body of work. It is more difficult to give direct evidence of the two

tasks being intertwined and not able to be differentiated from each other. The issue is not as simple as one paper stating that the goal is to explain the mechanistic process of the PPR, and then proceeding to use correlative data in order to draw a conclusion about mechanisms. Researchers in the field are certainly not confusing correlation with causation or stating that they aim to achieve one clear goal and then proceeding to do work towards an entirely different goal. The confusion and interwovenness of descriptive and explanatory tasks is more subtle and complex, and thus it is difficult to pinpoint specific examples. In order to demonstrate and give evidence of this, I will describe some of the common ways that the two tasks are intertwined, and then give one detailed example to show the complexity involved, and that it occurs within the literature.

In many papers, it is simply not clear which task is being engaged with. In such cases, there is no mention of either descriptive or explanatory tasks, yet the study is nonetheless empirically testing a hypothesis about 'the nature' of the PPR[34]. Another case involves papers switching between descriptive and explanatory statements about what 'the nature' of the PPR might be. Sometimes specific work (including studies, statements, statistics, inferences, models) appears to either group together or switch between descriptive and explanatory tasks[35]. Finally, as will be discussed in the upcoming example, it is not uncommon for one researcher, who appears to be engaged with explanatory tasks, to reference the work of another researcher who makes no mention of explanatory, causal, or aetiological findings.

An example: Watson, Gamez and Simms (2005) provide a brief summary of the empirical support that has been provided for various models across the body of literature. They describe the models as theories about *aetiology*, stating that they are possible accounts of "what

[34] An example of this is the study of Van Leeuwen et al., 2007.

[35] An example of this found in Durbin & Hicks, 2014, p. 367.

factor or factors are causally responsible" for the PPR (p. 62). They cite Clayton, Ernst and Angst's (1994) finding that "high premorbid levels of [neuroticism/negative emotionality] predicted the subsequent development of depression" as evidence in support of the vulnerability model (Watson, Gamez and Simms 2005, p 62-63). They (Watson, Gamez and Simms) articulate the vulnerability model as postulating that "[personality] exerts a causal influence on psychopathology … by increasing the likelihood that a person initially develops a disorder" (2005, p. 62). Yet Clayton, Ernst and Angst (1994), as far as the literature allows us to discern, only performed a Wilcoxon test, which at face value does not provide a stringent test of causation. That is not to say that it can't be done. Clayton, Ernst and Angst's study used longitudinal data to compare differences in means between different populations over time, and so it isn't impossible that they could provide causal findings. However Clayton, Ernst and Angst themselves do not report any causal results, or make any claims about an underlying mechanism or aetiological relationship between personality and psychopathology being supported by their findings. Within Watson, Gamez and Simms (2005), there is a discrepancy between the way that they describe the model and the type of evidence that they provide in support of it. In this case, descriptive and explanatory tasks are intertwined. Watson, Gamez and Simms describe the models as ones of explanation, they refer to predictive findings as evidence for a given explanatory model, and the study from which they draw that evidence appears to be intended to contribute to the goal of describing the PPR, not explaining it.

To reiterate, I am not suggesting that individual studies explicitly mistake description for explanation, and certainly not correlation for causation, and I acknowledge that this is just one example of the type of confusion between description and explanation that is characteristic of PPR research. Nonetheless, as the collection of various examples in this section have demonstrated, the body of PPR literature is abstruse with different tasks involved, and the

above scenario gives one example of how those tasks are intertwined and not kept separate *within the body of literature as a whole.*

Summary: attempts to study the nature of the PPR

An immense amount of research has now contributed to the study of the nature of the PPR. Generally speaking, the method of study has been comprised of two key stages: (1) consideration, development and revision of possible theoretical models; and (2) collection and statistical analysis of empirical data to assess the accuracy or fit of those models. Although studying the nature of the PPR is clearly a recognised research pursuit within psychology and psychiatry, and a great deal of work has been done in this area, it is difficult to be more specific about what that research project entails. I have argued that what is meant by 'the nature' of the PPR is non-specific and not inherently informative, and that upon analysis of the recent research, it appears that two fundamentally different tasks are involved within the single study: one concerned with describing the PPR; the other concerned with explaining it. This presents several difficulties, and the associated implications are discussed in section 3.1.

Having provided an overview of the study of the nature of the PPR, including its aims or focus of inquiry and the general two-stage approach, section 3 moves on to evaluating the scientific process of that research, and discussing what is required for effective and reliable inquiry in that area.

PART 3 – Critical evaluation: Limitations of attempts to study the nature of the apparent personality-psychopathology relationship and what is required for more robust and reliable study

There have now been extensive analyses of the nature of the relationship between personality and psychopathology, but comparatively little *conclusive* progress has been achieved. Where empirical findings have been thought to provide support for a particular model, they have been with respect to *individual* relationships between *specific* personality traits and *particular forms* of psychopathology (Tackett, 2006, p. 587; e.g. De Pauw & Mervielde, 2011; Depue, 1995; Rettew, Doyle, Kwan, Stanger, & Hudziak, 2006; Rettew, 2013, ch. 6). According to a recent summary of the literature presented by Durbin and Hicks, "there is no consensus that any one of these models [of the nature of the PPR] has been disconfirmed or any one has been uniquely supported" (2014, p. 366). While a great deal of valuable work has been done looking into specific links between traits and disorders, the field has largely stagnated at the level of providing broader accounts of the structural links, mechanisms and/or causes of the observed relationship between the two phenomena. Several PPR researchers have noted this lack of general and conclusive progress[36], highlighting the fact that questions about the processes underlying the joint presentation and connection of personality and psychopathology remain largely unresolved.

Parts 1 and 2 (above) provided a review of the work that has been done studying the nature of the PPR and gave a brief overview of the scientific methods that have been involved, as well as some of the conceptual elements at play. Part 3 now evaluates that work, focusing on the limitations of the research, and considering the main barriers to the study's progress. In

[36] For examples, see Durbin & Hicks, 2014 and Rettew, 2013, ch. 6.

doing so, I make suggestions about what is required for robust and reliable study of the relationship between personality and psychopathology.

In recent literature, a few issues have been raised that are thought to have contributed to the evident difficulty in studying the nature of the PPR (see Frick, 2004, p. 3-4; Rettew, 2013, ch. 6; Tackett, 2006, p. 587). I discuss what seem to be the most problematic barriers identified in the literature so far and introduce additional considerations about the theory and methods that inform and underpin recent studies. I propose that several factors have hindered the project of studying the nature of the PPR and will continue to do so unless they are adequately addressed, and in some cases, adequately resolved.

Throughout the following evaluation of the study of the nature of the PPR, I address three core aspects of the study: the research project (§3.1); the empirical and statistical methodology (§3.2); and the conceptualisation of the phenomena/objects of inquiry (§3.3). Each section discusses the barriers that are likely to have contributed to the evident difficulty in studying the relationship and considers the requirements of work in this area.

Broadly, I argue that: (a) a more precise articulation of the intended research project is essential for the success of future work, as well as for meaningful and valid interpretation of work already done; (b) statistical analyses (of association) to *test* potential models of nature of the relationship between 'personality' and 'psychopathology' provide a limited method of study as long as there continues to be a substantial lack of clarity concerning the conceptualisation, including the delineation and definitional parameters, of the two concepts themselves; and (c) current barriers and limitations of the study, concerning the study's design and aims, methods, and objects, stem from underdeveloped conceptual foundations.

Ultimately, I suggest that robust and reliable research in this area requires consideration and development of the *conceptual foundations* upon which it has emerged. In addition, careful consideration is required concerning the methods and design of the study at hand. Progress in

studying the PPR requires consistency and clarity around precisely what is being studied and what the resulting findings can tell us (discussed below). Thus, as Durbin and Hicks have argued,

> *"If real progress is to be made in discovering causal processes rather than in confirming and making progressively minor refinements to the conceptual space described by individual differences in traits at the population level, the field needs to critically evaluate the methods, designs, and hypotheses upon which it has become reliant" (Durbin & Hicks, 2014, p. 381).*

3.1 The research project: Explanation and description as distinct tasks not adequately demarcated in the current study

As pointed out in section 2 (mainly §2.4), within the single body of research studying the PPR, there are two fundamentally distinct tasks/focuses that appear to be intertwined and confused with each other. One is explanatory, concerned with causality or aetiology, including the mechanisms or processes that *explain* or account for the observed relationship. The other is descriptive, concerned with *describing* the surface patterns and properties of the relationship, such as the order of presentation, degree of association or shared correlates of personality and psychopathology. Thus, as was argued in part 2, the recent study of the nature of the PPR does not have a clear focus of inquiry. What is meant by 'the nature' of the relationship is neither inherently obvious nor well defined, but analysis of the research indicates a complex mix of *both* descriptive and explanatory goals.

Having established that these two different tasks – description and explanation of the PPR – are deeply intertwined and not adequately demarcated within the wider study, I now

suggest that this in itself is a significant factor contributing to the difficulty experienced by recent researchers of the PPR. Evidence and examples of their being intertwined was presented in part 2; this section now discusses the implications resulting from that and explains how it creates a barrier to fruitful research in this area. Specifically, I argue that confusing and conflating descriptive and explanatory tasks in this way has two *inherent* consequences.

The first stems from description and explanation being fundamentally different tasks with distinct goals, criteria for success and implications of their findings. When the two tasks are confused and confounded, it makes the body of literature as a whole difficult to engage with, interpret and apply – the study as a whole is largely undirected and unconfined (this is discussed in §3.1.1).

A second consequence is the conflation of research. With description and explanation not clearly distinguished, and the studies' results not sufficiently framed with respect to the relevant project, evidence across the two projects is conflated (discussed in §3.1.2).

Thus, I suggest that research that is able to provide well supported inferences or conclusions about the PPR requires better demarcation of these two tasks, and/or of the research project more generally.

3.1.1 Description and explanation of the PPR: two fundamentally different tasks

The distinction between description and explanation as individual tasks of research is recognised and endorsed in a substantial range of academic literature. The difference between describing and explaining the PPR has been discussed in a few places throughout this thesis so far, but a more comprehensive and direct outline of the distinction is useful here, so that we can better understand the implications of their entanglement.

Extensive arguments have been developed to support the view that explanation and description are not the same thing (Reese, 1999, p. 3-4). Although some have disputed this distinction, arguing that explanation should be understood as merely a different type or level of description, those arguments do not provide a challenge the view that explanation and description present *two importantly different research tasks*. The reason for this is that those arguments still endorse the same distinction that we see in the typical explanation-description distinction, they just purport that the two tasks should be understood as different forms of description, as opposed to labelling one of them 'explanation' (Reese, 1999, p. 3-4).

The difference between description and explanation is well illustrated by a simple example. Consider the relationship between a switch and a light: it is *description* to note that the light will turn on when the switch is flipped, but a wiring diagram of the process may offer an *explanation* of why the light turns on when the switch is flipped.

While it is an accepted idea that describing and explaining are fundamentally different tasks, articulating that difference can be complicated. Scientific explanation is a complex topic, and many different (sometimes conflicting) notions and accounts of explanation have been developed and debated over time, however, exploring those various theories and what they involve is out of the scope of this thesis. Regardless, what *is* seemingly consistent across the board is the idea that successful explanation achieves something over and above "mere description" (Woodward, 2014, s1). Roughly, description can be understood as providing an account of the way things are ('describing' *what* there is), whereas explanation provides an account of the cause or reason for the way that things are (it 'explains' *why* that thing is there) (Reese, 1999, p. 3-4; Woodward, 2014, s1).

Description of the PPR versus explanation of the PPR

Applied to the context of PPR research, that contrast between descriptive and explanatory outcomes remains. It is not realistic (or even appropriate) to provide a complete account of the different tasks of describing as opposed to explaining the PPR because there are many interpretations of what descriptive and explanatory tasks and their different types may involve. But while there is no list of the various descriptive and explanatory tasks involved with studying the PPR, certain tasks are categorically descriptive, while others are categorically explanatory. This is the case for the two tasks apparent within the recent PPR study, which I identified and labelled as *the descriptive task* and *the explanatory task* in part 2. Specifically, the first task, aiming to providing an account of *the way the relationship presents*, is strictly descriptive. This is because it focuses on *what there is* – the details and patterns of the manifested relationship. For example, identifying the magnitude of the association, or perhaps even shared correlates between the two phenomena, would be contributing to a description of the PPR. Elements of the relationship are described, and an *explanation* of these elements would then account for the reasons *why* – what causes the strong correlation or shared association with another variable? Thus, the second goal, aiming to provide an account of *the mechanisms or aetiological processes that underlie the relationship*, is clearly explanatory. It is concerned with *the causes of the relationship* occurring and/or being the way that it is. For example (although perhaps unrealistic), determining that psychopathological disorders alter individuals' brain chemistry in a way that causes changes in their personalities, would be an explanation of why personality and psychopathology are related.

Fundamentally different tasks, with different goals and criteria for success

The two intertwined research tasks involved in recent PPR research are importantly different because they seek to achieve different things. They are seeking to provide different *types* of

accounts of the PPR, and thus their research scope and possible outcomes are different as well. Thus, recent literature is hindered by the fact that it is not all working towards a common goal – the research is largely undirected and does not have a clearly defined scope. As was mentioned in part 2, in my experience, it can be difficult to figure out precisely what the research is seeking to achieve, and it now seems that this is due, not only to the fact that it is unclear what 'the nature' refers to, but also because two fundamentally different tasks are confused within the single body of work. Ultimately, their confusion and entanglement in the recent literature makes the study difficult to engage with. This is firstly because it is difficult for a body of research to make unified progress towards *either* goal without a clear understanding of what that work is seeking to achieve and which studies are contributing to which goal. But moreover, with description and explanation not adequately distinguished, it is difficult to engage with research already done. Reviewing the studies and interpreting and applying their findings is almost impossible without an understanding of whether the task at hand is descriptive or explanatory. Generally speaking, identifying the study's aims, considering what methods are best suited, and knowing what the findings can tell us and what they inform, are all influenced by an understanding of the research task at hand. This brings us to an associated issue: explanatory and descriptive findings have different implications, and thus it is important to be cautious not to confuse their applications.

Different implications of description and explanation

Descriptive and explanatory information have different uses. Descriptive information has the power to inform in some contexts but is limited in others. By definition, a description of the PPR is not able to provide information about *why* personality and psychopathology are related. Thus, description is uninformative for matters such as mental disorder treatment, where it is important to understand the causes or mechanisms of what is going on in order to be able to

mitigate or intervene in those, whereas explanatory information is able to provide evidence that is fit for the purpose of informing treatment options. On the other hand, a good description of the PPR might provide useful information for predicting the onset of mental disorder, or in making predictions about how children will develop over time. Treatment and prediction are two of the most clear-cut examples of how descriptive and explanatory information is used in different contexts in medical research, but others include policy, legal discissions and classification. The important point is that there are quite different implications and applications of the progress made in the two different projects. For example, it would be inappropriate to provide descriptive research as supporting evidence for a particular treatment plan, because that research does not have the necessary explanatory power that is required for that context.

In turn, it is important to ensure that the claims of implication are founded on findings from the appropriate or relevant research task. That is, if we are referencing the PPR research to inform 'real-world' decisions, it's necessary to ensure that those studies are directly applicable to the claims being made. However, with descriptive and explanatory tasks not adequately delineated in recent literature, their results aren't adequately segregated for these purposes. As was discussed in part 2, it is often difficult to determine which task a given study or paper is engaged with, and thus the appropriate use of that study's results or the statements made is unclear and *easily misattributed*. When implementing the findings of empirical research, it is crucial to understand whether it provides descriptive or explanatory information in order to know the scope of that information and what it accurately informs. Currently, general statements are frequently made about the implications and applications of the study's findings in general, which lump the two tasks together under one project and suggest that the study's findings are useful for purposes of treatment *and* prediction, along with other uses (see for example Watson, Gamez & Simms, 2005, p. 47).

Unfortunately, conflating the two projects involves confusing their implications. If we are unclear about the separation of the individual studies into their respective tasks, we risk being unclear and 'misattributitive' about the appropriate implications and applications of those studies' findings. Thus, confusing the two tasks is problematic in that it increases the risks of unfounded claims being made due to their not being supported adequately by *relevant* research. With the applications of such research being influential in the way they affect both individuals and the wider society, and the risk of getting things wrong carrying often heavy consequences, it is especially important to acknowledge the subtle differences between descriptive and explanatory findings.

Summary: confusing importantly different tasks of research

Altogether, current research is limited by the confusion and entanglement of descriptive and explanatory goals. It is important for research to distinguish descriptive claims and tasks from explanatory ones, not only because it makes the research focus and scope clear, but because generally speaking, they are different projects, and failing to adequately identify and discriminate them is failing to comply with this important and widely recognised distinction.

3.1.2 Conflating descriptive and explanatory evidence

The second, and more concerning, consequence of the entanglement of descriptive and explanatory projects is the associated erroneous conflation of the relevant studies' results.

With description and explanation being fundamentally different tasks, with different scopes and outcomes, the empirical data that provides relevant evidence for either one those tasks is not *tout court* extendable to the other. In fact, the type of evidence that informs one project is importantly different to the type of evidence that supports the other. A simple

example of this is correlative data: statistical correlations can provide direct and informative *descriptive* data about the PPR, but they are not informative about the *causes* or *mechanisms* of the relationship. Another example could be genetic evidence: certain types of findings about individuals' DNA might provide causal evidence that explains *why* there is a relationship between personality and psychopathology, while being limited in the information it provides about the way the relationship presents (the way that relationship will manifest in people, such the order or way that traits and symptoms present).

A significant result of confusing description and explanation within the single PPR study is that the empirical data pertaining to the two tasks is confused as well. With it unclear which individual studies pertain to which project, and some studies appearing to be either a mix of the two, or unspecified in exactly which task they are seeking to achieve, it is almost inevitable that those studies' empirical results and findings will be confused.

The main problem with this confusion of descriptive and explanatory research occur when (or if) research illegitimately draws on descriptions of surface level manifestations as evidence of the underlying causal or aetiological mechanisms of the relationship. Specifically, in cases where statistical results provide evidence of the way the relationship manifests and its observable properties and patterns, those findings are not directly extendable as conclusions about the relationship's underlying processes. This is briefly discussed within the PPR literature, with the recognition that structural and descriptive accounts of surface level associations are "not informative about *how* the associations were established" (Durbin & Hicks, 2014, p. 364). Yet although it is apparent that purely descriptive findings are not appropriately characterised as conclusions about causal, aetiological or mechanistic processes, when the studies are unclear about their intentions – whether they are providing descriptive or explanatory findings – those two types of findings aren't able to be kept separate.

There appear to be cases in the literature in which researchers provide descriptive findings (especially findings from studies which are not their own) as *direct explanatory evidence* of the underlying processes of the PPR[37]. However a more important point is that it is often difficult to determine where and when this happens as the two tasks and the associated findings aren't clearly distinguished or easily separated. This confusion is unsurprising given the literature's confusion of the two projects more generally, but it presents a significant issue when it leads to inferences or conclusions which are simply not supported by the right type of evidence.

This is not to say that descriptive findings give *no* insight into potential explanations (or vice versa). Relevant evidence pertaining to one of the projects *might* be relevant to the other in the sense that it contributes to our overarching understanding of the relationship. Research might even provide insight, or to some extent inform the possibilities, of the outcomes of the other project. But evidence pertaining to one of the projects is not one and the same thing as evidence pertaining to the other. The projects should not be seen as interchangeable. Even if evidence of one type is suggestive of outcomes in the other project, it does not entail that outcome, or provide *direct* evidence of it being the case.

With description and explanation so deeply intertwined within the single PPR study, evidence pertaining to the two tasks is mixed, and it becomes easy for evidence relevant to one task to be incorrectly presented as evidence of the other. Moreover, with all of the studies' research presented as findings of the unified study of 'the nature' of the PPR, with no clear distinction between descriptive and explanatory research, the different findings are conflated. As a result, the literature gives an inflated picture of the empirical findings, as it presents one

[37] See Watson, Gamez & Simms, 2005, p. 62-63 on associations as evidence in favour of a given aetiological model; or Rettew & McKee, 2005, p. 22 on proportion of variance as evidence of underlying mechanisms.

extensive set of results, whereas in reality, there are two tasks at play which do not provide information towards one single (either explanatory or descriptive) account of the PPR. Statements such as "[all of the theoretical models of the nature of the PPR] have received at least some support in the literature" (Watson, Gamez & Simms, 2005, p. 62) give the illusion of a large, cohesive body of empirical research, without taking into account the complex differences of the tasks that are involved with that research.

3.1.3 Suggestion for further study: a clear research project

I suggest that more robust and reliable research in this area requires better demarcation of the intended research project.

The lack of a clear and well-defined research project has had several negative consequences. What is meant by 'the nature' of the PPR is not inherently informative, its meaning is not specified, and consequently, the focus of inquiry of the body of research is not uniformly understood. The muddle of descriptive and explanatory tasks within the single body of research, in particular, has noticeably limited its success. It is difficult to understand what the study is seeking to achieve and what its findings are able to tell us or what they can be used to reliably inform. Recent work has failed to discriminate importantly different tasks of research. The scope of the study is not well understood or easy to determine, the implications and applications of the study's findings are problematically confused, and evidence pertaining to the two tasks, although not equivalent, is easily conflated.

Being clear about this will help to elucidate and separate the different potential goals or tasks of the research and ensure that their different scopes are adequately acknowledged. Most importantly, I recommend that when it comes to studies of the PPR, it is important to be rigid about the distinction between the descriptive and explanatory tasks. Not doing so limits

the reliability of the research and limits its progress. Without a more precise research project, the issues outlined above cannot be resolved and thus will continue to limit the success of the research.

Altogether, a more precise articulation of the intended research project is vital to the success of future work, as well as for meaningful and valid interpretation and application of work already done.

3.2 Empirical and statistical method of inquiry: complications and limitations of the statistical approach to evaluating the models of the PPR

Recent attempts to study the nature of the PPR have been distinctly uniform in their methods. Statistical analysis of empirical data has been at the forefront of that research. Beyond the brief and somewhat routine discussion of the set of potential models (the first stage of the prominent two-stage approach discussed in part 2), to my knowledge, empirical statistical research has been the only method of studying the PPR.

The overview (provided in §2.3 above) of the empirical data collection, statistical analyses and their results and inferences demonstrated what is involved in this methodology. Importantly, that overview identified two key aspects of the research. The first is that the statistical tests implemented are mainly ones of *association* – they analyse the way that multiple variables are associated with each other, by calculating correlations, covariances, differences in their means, and so on. Multiple regression and longitudinal studies are also regularly employed as methods of *causal* analysis. The second key aspect of the research is that the empirical data collection captures quite specific representations of personality and psychopathology. The measures of the two phenomena are entirely (as far as I have seen)

measures of surface level manifestations. Regardless of how personality and psychopathology might be best conceptualised, empirical studies collect information about individuals' personality and psychopathology by recording information about their outwardly 'observable' properties such as behaviours. Sometimes the measures will account for less clearly observable information, such as emotions, thoughts and cognitions, but that is 'surface level' information in comparison to what we might call 'internal elements', such as DNA structures, neurobiology and brain chemistry. That is, the recent studies collect information by measuring surface level manifestations of the two phenomena, *as opposed to* their underlying ('internal') structures or processes.

This empirical statistical methodology presents a second crucial limitation of current attempts to study the PPR. Specifically, the statistical analyses as they are currently used in the study don't provide a particularly robust method of inquiry for PPR research. They are limited in what they can tell us about the relationship between personality and psychopathology, largely due to the way that empirical data of the two phenomena is collected. This issue is further exacerbated by many of the complications of the methodology being overlooked in the relevant literature. There is currently a disparity between the information that the statistics can reliably provide, and what the literature purports them to indicate or prove about 'the nature' of the PPR.

The following discussion about the limits and problems of the empirical statistical methodology is split into two parts.

The first (§3.2.1) focuses on the use of statistics as a method of inquiry for the explanatory project of the PPR study. I argue that, at least as it is implemented in the current body of literature, the employed method of statistical analysis is inadequate for the purpose of ascertaining sound, robust evidence of causation/underlying mechanisms of the PPR.

The second part (§3.2.2) discusses the use of this methodology for either project (descriptive or explanatory). The focus here is on the *measurement* of personality and psychopathology. I argue that, causal and aetiological claims aside, any successful and informative statistical analysis requires precise and accurate measures, and that is currently not achieved within PPR research.

I suggest that robust and reliable study of the PPR requires caution around the use of empirical and statistical methods of inquiry. If that approach is to provide a useful methodology for studying the PPR, the issues discussed below need to at least be acknowledged and addressed, if not directly resolved.

3.2.1 Statistical analyses as a method of inquiry for the explanatory project

Statistical analyses tend to be uninformative about the aetiology and mechanistic processes underlying relationships between phenomena. This is especially true when the analyses are working with observational, as opposed to experimental, data. Roughly, this is because the statistical process involves analysing *patterns* of situations or events that have occurred, rather than investigating the laws or causal chains in the world that have led to that occurrence. It is difficult to use information about the *way* that things are to determine *why* those things are as they are. Sometimes statistical analyses can be insightful about causal processes, but stringent requirements need to be satisfied in order for this to be the case. Nevertheless, the current approach to studying the PPR relies heavily on statistical inferences as a main method of inquiry, with a common trend within the literature being the use of statistical data as *evidence* of causality or mechanistic and aetiological processes. So far, this use of statistical analysis has been largely inconclusive.

The problem is that statistical analyses, at least the ones employed within the literature, provide an ill-suited and reasonably ineffective method of inquiry for investigating the *explanatory aspects* of the PPR because the necessary standards for producing explanatory (causal, aetiological, mechanistic) evidence are currently not met. In what follows, I argue that the research fails to meet those necessary standards in two fundamental ways.

The first is that there are a number of *test parameters* that need to be satisfied in order for statistical analysis to be considered capable of providing causal information, and those are not adequately addressed within the literature.

Secondly, the study faces an issue of *representation*. It is vital to ensure that the tests are assessing robust representations of the phenomena and their relationship, in a way that currently cannot be achieved.

Test parameters: satisfying the standards for causal inference

With any statistical test, there are a series of parameters that need to be met in order for the test to be fit for purpose. Some of the most important of these parameters are the test assumptions. Test assumptions outline characteristics about the data (e.g. it is normally distributed) which need to be met in order for the relevant test to give reliable outputs. If those test assumptions are not met, reliable conclusions cannot be drawn from the results of the analysis. Test assumptions, while crucial to reliable analysis, are relatively straightforward and well adhered to. However in some situations, especially in complex ones such as when working with unusual data sets or aiming to infer causal mechanisms, there are further parameters beyond the standard test assumptions that need to be met in order for researchers to be able to draw reliable conclusions from the results of the analysis. It is those parameters that present an issue in the current study.

Many analyses are presented across the body of literature, and although it is quite possible that some of those meet the necessary parameters for causal inference, a common trend across the board is that those more complex parameters aren't sufficiently addressed[38]. That is, the research that performs the analyses tends not to acknowledge the necessary parameters and show how the study at hand satisfies those parameters. Moreover, in many cases it seems that those parameters simply aren't met – the statistical analyses have a series of small deviations from the necessary standards for causal inference. This is not an issue in and of itself, because meeting those parameters is not necessary if the tests belong to the descriptive project, and are only intended to be relevant to descriptive statements. However in so far as the results are reported as contributing to the explanatory goal, or not adequately distinguished from that, it is important that the analyses are shown to meet the necessary standards for causal/aetiological inference[39]. In my experience of the PPR literature, the studies seem to never quite reach the benchmark by which we could be confident that reliable causal conclusions can be drawn from the results of the analyses. Some examples from the literature are provided here to illustrate some of the ways in which the necessary parameters for inferring causality/explanatory information from the test's results either aren't satisfied or aren't shown to be satisfied.

Longitudinal regression: several researchers have used the results of regression analyses of longitudinal data for the purpose of investigating the PPR (for examples, see Katon et al., 1994 and Hengartner et al., 2018). Throughout the literature, findings from these sorts of studies have been reported as giving evidence of *explanatory* causes or mechanisms of the PPR (for example by Watson & Clark, 1995, p. 362; Klein, Kotov, & Bufferd, 2011, p. 271 &

[38] An example from the literature is provided below ('Longitudinal regression').

[39] As was argued in section 3.1.2, the two projects require different evidence and so, when making claims of explanatory processes, it is important that the evidence is fit specifically for that purpose.

280). This is unsurprising, as both longitudinal studies and regression analyses are thought to be well-suited for enabling causal inferences. However many different factors need to be controlled and accounted for if that causal inference is to be valid. Two examples of the types of factors that need to be addressed in order for the analyses to meet the necessary parameters for causal inference are to do with *multicollinearity* and *omitted variables*. There should be an absence of multicollinearity in the data, meaning that independent variables must not be highly correlated with each other. Further, important, potential confounder, variables must not be omitted, meaning that other variables that might be associated with both the dependent variable and other independent variables should be included in the analysis in order to avoid spurious correlations (Allison, 2014, para. 9). Without those sorts of parameters being addressed, the analyses don't provide robust means for causal analysis (Allison, 1999, ch. 3). However, in PPR studies those parameters aren't always shown to be satisfied. An example of that can be seen in Katon et al. (1994, p. 81-86), where regression analysis of longitudinal data investigated the effect that multiple variables, such as neuroticism, adherence to medication and chronic pain, had on depression symptoms. Yet the associated literature that claims the study provides evidence of the aetiological mechanisms of the PPR (Watson & Clark, 1995, p. 362) does not address, or even mention, whether or not those parameters are met. This is not because it is obvious that they are met, because it is quite possible, for example, that adherence to medication is strongly negatively correlated with neuroticism. Thus, the literature reporting that such studies provide evidence of explanatory process underlying the PPR unreasonably overlooks these factors.

In order for the empirical statistical methodology to be considered capable of providing reliable explanatory information about the PPR, the crucial test parameters need to be more adequately addressed within the literature. Specifically, for matters of reproducibility, interpretation of the findings and extendibility of the results, it is essential that evidence of the

test parameters being satisfied is provided in the relevant literature that presents the study's findings.

Representation: variables and phenomena of interest

Even in cases in which the necessary test parameters are satisfied, and are shown to be satisfied, the statistical data still cannot be assumed to provide reliable and *unique* evidence of causality or aetiology due to the way in which the phenomena are represented in the analyses.

Representation plays an important role in statistics. This is because when we are interested in using statistics to study something in the world, we represent that thing using data in order to be able to put it into an analysis. A simple example of this is age. If we are studying the age of a group of people, we gather that information as a set of numbers and input it as a 'variable' in the analysis. Age is well represented by a number.

Accurately representing phenomena is perhaps the single most important thing to get right when using statistical analysis as a method for studying causes and explanatory process that underlie the relationships between phenomena. If the patterns and properties that are found to exist between the two phenomena (from the statistical tests) are not based on precise and valid representations of those phenomena, then using that information to try to understand what explains those patterns is almost impossible (discussed below). However, whereas phenomena such as age are easily represented by numbers, other phenomena, such as personality, cannot be as simply represented by data sets.

As discussed above, in the current PPR study, the empirical method of research gathers personality and psychopathology data solely by measuring surface level manifestations of the phenomena. Thus, the personality and psychopathology 'variables' that are input into the statistical analyses represent those phenomena of interest using entirely surface level data. The issue with this is that the tests' variables do not necessarily provide robust or valid

representations of the phenomena of interest, and assessing that degree of representation presents issues in and of itself.

Assessing the degree of representation requires an understanding of what a good representation of the phenomena would look like. In the case of diabetes, for example, assessing the degree of representation is easily achieved because we have a clear conceptualisation of what it is to have diabetes. With its conceptualisation being largely uncontroversial and well defined, all we need to know is how a study has gathered information about the medical condition (e.g. by medical diagnosis, or by measures of blood sugar levels) in order to assess how well that variable in the study represents diabetes. Without a better conceptual understanding of what is really encapsulated by the terms 'personality' and 'psychopathology' – an issue that is explored in depth in section 3.3 – we are ill-placed to understand the accuracy of the representation between the statistical variables and the intended phenomena of interest. Although it is commonly thought that personality and psychopathology are composite constructs in that they are comprised of many different components, exactly what combines to make something an instance of personality or an instance of psychopathology is widely disputed and currently vague. Specifically, within the PPR studies, it is not made clear, for example, whether or not it is thought that neurochemistry is an important component of mental disorder. Without further refinement or explication of the study's concepts, we lack the necessary information to assess the disparity between the variables and the intended phenomena of interest. The link is unclear and seemingly unknown, or at least, it is very difficult to discern within current literature.

While it is difficult to determine the accuracy of the study's representation without a more articulated and endorsed conceptualisation of the phenomena, it is clear that personality and psychopathology are widely held to be phenomena that exist as more than mere clusters of surface level presentations, and so it's unlikely that the variables of current PPR research map

directly onto the intended phenomena of interest. It is unlikely that the variables of the study, which are based entirely on surface level manifestations, provide a complete, precise and accurate representation of the complex (and composite) phenomena that we are talking about when we discuss the potential *reason or causes* of the relationship between personality and psychopathology. Similar discrepancies between surface level manifestations and the underlying 'deeper' phenomena that they supposedly represent can be found in other areas of research. Assertions don't map clearly onto beliefs, symptoms don't map clearly onto diseases, and phenotypes don't map directly onto genotypes. Interestingly, in these cases, the degree of representation between the observed variable and the underlying phenomena is easily assessed because we have a reasonably clear picture of what a good representation of the phenomena would require. Moreover, the variable and the phenomena being unequal in this way is much less of a concern when the goal is not one of explanatory inference.

The consequence of the variables not mapping directly onto the phenomena of interest is that when we observe different interactions and processes at the surface, variable level, those patterns cannot be reliably *extrapolated* to the lower, mechanistic phenomena level. Without a better understanding of the link between the variables and the phenomena, the variables act only as *markers* of the phenomena. Simply put, the presentation of the observable marker of the phenomenon is not a sufficient indicator of the phenomenon itself in respect to time, origin, and so on. It may reliably tell us about the presence of a phenomenon – for example, someone is presenting with depressed mood – but not about the underpinnings of the manifestation – such as *someone is presenting with depressed mood due to low serotonin*. The presentation of an observable marker struggles to answer questions such as *when did the serotonin become sufficiently low?* or *does the depressed mood persist without the low serotonin?*. As a final illustration of what I mean by the variables acting only as 'markers', consider two different psychological phenomena: stuttering and extroversion. For stuttering, when you observe the

behaviour, you are observing the phenomena. You aren't observing the *causes* or the *mechanisms* of the stuttering, but stuttering and the surface level manifestation of stuttering are one and the same thing. For extroversion, when you observe extroverted behaviour, you cannot be said to be observing the phenomena. There is something *more* to the personality profile of extroversion than extroverted behaviour. To observe extroverted behaviour is to observe a *marker* of the phenomena – there is a level of abstraction between the surface level manifestation and the phenomena itself. There are of course surface level manifestations other than behaviour to consider here, but the point is that if personality and psychopathology are thought to exist as more than clusters of surface level manifestations, variables that only take account of surface level data provide markers, as opposed to complete representations, of the phenomena of interest.

With the variables of the study providing only markers of the phenomena, statistical analysis is greatly limited in the evidence that it can provide about underlying mechanisms or causes. To understand why the barriers of representation suggest that the tests are unable provide *reliable and unique* explanatory evidence (in the way that the literature purports them to), it is useful to consider an example.

Example case: longitudinal studies with outward behavioural manifestations as variables. Longitudinal studies are thought to be appropriate for inferring causal relationships. Causes generally precede effects. Yet this information doesn't seem to apply well to cases in which the studies' variables do not fully represent the phenomena of interest. It may be the case that there is a particular temporal pattern of presentation of the variables, for example, a certain symptom might tend to develop after the appearance of a certain trait, but that doesn't directly entail any specific causal relationship between personality and psychopathology. The variables that are indicative of the phenomena are *only* indicative – the degree of representation

is not one of equivalence, and thus the observed patterns cannot be assumed to be equivalent to the mechanistic patterns.

Scenario: suppose there were a longitudinal study, which found that all children who presented with low extraversion developed Autism later in their lives, say in adolescence, and all children who presented with high levels of extraversion did not develop Autism. It may be claimed – and this is the type of claim often made – that observation or data provides *evidence* in support of the model which theorises that personality causes or has a predisposing influence on psychopathology – viz. the predisposition model. Let's call this inference, from data to model, *inference A*. The problem is that, based on the study alone, this inference doesn't seem any more appropriate than any other.

The findings of the study do not rule out that there is an additional variable which is responsible first for the presentation of low extraversion, and later for the development of Autism, with there being no causal relationship whatsoever between the personality trait and the psychopathological condition themselves (*inference B*). That is, it may be that the common cause model is correct or 'the best fit' for the data. Not only is this explanation not ruled out by the findings, it would be *equally* consistent with the study's results as *inference A*. In turn, it is unjustified to infer that the study provides *unique* empirical support for the 'goodness of fit' or correctness of the predisposition model.

Further still, it would also be consistent with these findings to suggest that what is going on is that the children were born with a particular neurobiological structure (or even a particular gene) which is a latent manifestation or characteristic of Autism (note, this explanation requires that psychopathology can be latent), and it is the presence of this that causes them to have low extraversion, but the surface or 'outward' manifestations of Autism do not present until later in development – *inference C*. Another possible alternative (a variant of *inference C*) is that the individuals first had Autistic traits or symptoms, which caused low extraversion, and

only later in their lives they presented sufficient symptoms (perhaps with the presentation of low extraversion) to meet diagnostic criteria of Autism (or any scale used to measure Autism as a variable in the study) (*inference D*). Each of these explanations would be in line with the scar model.

Environmental factors, conceptual and classification considerations (such as the criteria for considering an individual to have Autism), and so forth, are possible explanatory factors that exist above and beyond the mere observation of the patterns of the 'markers' of personality and psychopathology. If the variables – the surface level manifestations – act only as markers of the phenomena of interest, mechanistic or causal process cannot be directly inferred. Thus, this statistical analysis *alone* is insufficient for providing *unique* data pertaining to the explanatory project.

In summary, the issue is that the tests' variables do not necessarily provide robust or valid representations of the phenomena of interest. As personality and psychopathology are widely thought to be made up of more than clusters of surface level manifestations, it seems that the current PPR study's variables act only as markers of the intended phenomena. Thus, while the studies' results *might be indicative* of the underlying mechanistic process, they are unable to provide unique evidence of a particular model. Specifically, symptom and behaviour clusters of surface level *manifestations* as *variables* of the *intended* phenomena lack the coherence, accuracy and precision required for representation in causal contexts.

Summary: empirical statistical methodology for explanation

Causation is very hard to prove with statistical analysis. Not only are we restricted by the types of tests that enable causal inference, but the data and theoretical underpinnings that are appropriate for causal analysis are limited as well. Within the current body of literature, the

employed method of statistical analysis is inadequate for the purpose of ascertaining sound explanatory evidence.

One simple barrier to the research providing sound explanatory evidence is that the necessary evidence of the statistical tests' parameters being met is not provided, and thus we are unable to verify that the statistical analyses are sufficient for providing causal data. More importantly, the study's methodology faces an issue of representation. This stems from the complexity of the phenomena being studied. The standard of representation that the test variables provide is difficult to assess without a more comprehensive conceptualisation of personality and psychopathology within the study (discussed further in §3.3). Further, the use of surface level manifestations as *variables* presents a more challenging representation issue – the variables act only as markers of the phenomena, and thus the observed patterns cannot be reliably extrapolated to explanatory mechanistic claims. Due to there being in sufficient reason to assume, that (a) the analyses satisfy the appropriate test parameters and (b) the variables provide valid representations the phenomena of interest, the explanatory goal of the PPR research is impacted by the currently dominant method of study.

This is not to say that the use of statistical methodology is misapplied or incorrect, just that its implementation in the explanatory project is, at this stage and without further development, largely unfruitful and uninformative. At best, the studies' results can provide data that are suggestive as to the most simplistic explanation of the relationship, but those inferences must be understood as tentative and should not be considered robust or unique evidence of causality or aetiology. Thus, while the analyses might provide interesting information about temporal associations between the variables, they are inadequate for providing *unique and reliable explanatory evidence* about the PPR. I am not arguing that the statistical findings are entirely unhelpful, or that they can't contribute to a bigger picture of research or be used for inference to the best explanation, but *currently* they do not provide

robust explanatory evidence in the way reported in the literature. If the different tasks of description and explanation were clearly separated within both the individual studies and the models that they are testing, then statistical analysis may be able to be used and interpreted more successfully.

A major limitation of recent attempts to study the PPR lies in the fact that the methodology is *currently* not well fit for the purpose for which it is being used. Section 3.3 will discuss what might be required in the body of research to enable the use of statistical methodology to produce more robust and reliable findings. A remaining issue, however, is that there is a current disparity between what the statistical results can tell us about the PPR, and what the literature is reporting that the results provide evidence of. The extent to which unjustified conclusions and inferences are stated in the literature is uncertain, as it is sometimes hard to tell whether the test results are thought to support the descriptive or explanatory goal of studying the PPR. Regardless, the statistical analyses (as they are currently used) are not a reliable tool for ascertaining unique explanatory evidence, and so in so far as a component of the research does involve making explanatory inferences, caution is required around the use of statistics as a method of inquiry to achieve that. The crucial issue is that it is important that the issues and limitations of the current methodology are acknowledged within the literature, and that their explanatory power is not overstated.

3.2.2 statistical analyses as a method of inquiry for either project – measurement: quantifying the objects of inquiry

Causal and aetiological claims aside, the empirical statistical methodology of current PPR research is limited in its effectiveness and reliability due to an issue of *measurement.* Specifically, any successful and informative statistical analysis requires precise and accurate

measures. Quantifying the objects of inquiry – being able to pick out and measure what we are studying in the world – is a crucial aspect to sound statistical research. Imagine for example that we are trying to study the relationship between bald people and the size of their feet. If we set out to study baldness in the world, we need to first understand how to measure it. It could be that it is an absolute, categorial state (e.g. bald versus not bald), in which case we need to know the cut-off point. Alternatively, it may be thought of as falling on a continuum, in which case we run into an issue if we aren't sure how to count bald people, and we face an even larger issue if all researchers go about counting baldness differently, with no standard of reference for what the literature considers to be a good measure of baldness. Without a clear understanding of precisely how objects of inquiry may be quantified, statistical analysis provides an imprecise and reasonably ineffective method of inquiry.

Thus, the use of statistical analysis as a tool to investigate the PPR involves the assumption that the study's tools of measurement successfully 'pick out' the intended distinct and individually identifiable entities: personality and psychopathology. Yet personality and psychopathology in the current body of literature are fuzzy and difficult to quantify. Their measurement is not only difficult to perform, but difficult to theorise.

This presents a multifaceted measurement problem. Empirical measurement of the phenomena gives rise to a range of methodological complications, but addressing those complications, and considering how to successfully quantify and 'pick out' personality and psychopathology in empirical study, leads to a further, theoretical, measurement problem. Regardless of how personality and psychopathology are best quantified, current measures fail to accurately and precisely pick out the individual objects. Let's consider these issues in turn.

The methodological measurement problem. Some objects are harder to measure than others. While some, such as age, are easily measured, others can present a host of complications. Unsurprisingly, personality and psychopathology are particularly difficult to

measure. Numerous methodological measurement issues arise when trying to gather empirical data of the two phenomena, such as item overlap, comorbidity, and false negatives and positives, and these have a negative impact on the validity of the study's results.

One of the methodological problems concerning the way the two concepts are measured in empirical studies which has been addressed in detail in the literature is *item overlap* (a problem in research in which questions, statements, etc. that are used to assess one phenomenon are also used to assess another phenomenon of interest). It is worried that although empirical studies typically treat the concepts of personality and psychopathology as distinct (able to be individually identified and measured), many of the individual instruments being used to measure personality traits and psychopathological symptoms in individuals confound the two concepts (Rettew, 2013, p. 104-106; Tackett, 2006, p. 587; Frick, 2004, p. 4; Rettew & McKee, 2005, p. 24). Specifically, it is often the case that the same items occur on scales that purport to measure personality dimensions as the scales that purport to measure the corresponding psychopathological condition (Rettew, 2010, p. 442; Rettew, 2013, p. 104-106; Tackett, 2006, p. 587; Frick, 2004, p. 4). An example of this from the literature on temperament is that items assessing where participants fall on a scale of "losing one's temper" are found on both trait measures of novelty seeking and psychopathology measures of oppositional defiant disorder (Rettew, 2013, p. 104-105). Other examples of methodological measurement problems can be found when analysing the tools of measurement of the study, but while these are not ideal and must be accounted for, such complications are characteristic of statistical analysis more generally, and in research outside the PPR domain, can usually be effectively delt with once they have been identified.

The theoretical measurement problem. The complex issue here is that it is not at all obvious what a good measure of personality or psychopathology would look like. Dealing with methodological measurement issues is almost impossible if we can't decide *how* to accurately

and precisely measure or empirically 'pick out' the phenomena of interest in theory. The problem is that the objects of inquiry of the study are simply difficult to quantify – personality and psychopathology do not have "clear and self-evident" boundaries; they are not "reliably observable" entities that "offer themselves up as raw data" (Freebody & Freiberg, 2006, p. 711). Typically, the 'natural science' approach of statistically and empirically analysing an observed or known link between two phenomena would have objects whose boundaries are straightforward and almost 'self-evident' (Freebody & Freiberg, 2006, p. 711). Here, we face issues in determining: *how* we can accurately 'tap into the objects' (how they can be accurately identified and *quantified* in the world); what should be done when methodological issues such as item overlap are identified; and generally, how we can, *in theory*, establish what would suffice as a good measure. We have no generic or widely accepted standards by which to assess or determine good measures of the phenomena. With such uncertainty surrounding how to observe the phenomena in the world we lack standards or reference points by which we can determine whether the measures pick out what we mean for them to pick out, and nothing else. When we face an issue such the measurement of a single behaviour acting as an indication of both a personality trait and a psychopathological symptom, it is unclear how we should even go about attempting to resolve that. The result is uncertainty about how to perform good measurement of the objects; it is unclear *how* to establish precise and accurate measures for data collection of these complex and non-specific phenomena.

Current measures are inadequate. While it is difficult to determine the appropriate measures of the objects, it is not difficult to see that the *current* tools of measurement (the scales) of the variables in the statistical analyses provide limited measures of complex and poorly understood phenomena. The psychological scales and diagnostic criteria seem to be *treated* as adequate measures for personality and psychopathology, yet this does not seem to align with *any* well endorsed interpretation of what an accurate quantification of the intended

objects of inquiry could be. Statistical analyses rely on valid and reliable measures. Without refinement of the intended objects of inquiry and development of their measures, statistical analysis provides a limited tool for assessing their relationship.

With the current barriers to measuring the objects of inquiry, the results of the tests fail to provide informative information about the PPR. The validity of the test results is dependent on the reliability of the measurement, and the appropriate interpretation of those results is highly dependent on a more detailed understanding of how to quantify personality and psychopathology in the world, which is simply not present within current literature.

Again, this is not to say that statistical methods cannot be used to study the PPR. Instead, it important to understand and acknowledge the complications and constraints of doing so. Addressing the issues with the methodology is an important element of achieving sound research. As was discussed is part 2, the results of the statistical analyses are commonly reported as direct evidence either for or against the various proposed models of the PPR. I suggest that a contributing factor to the inconclusiveness of the recent study and its lack of generalisable findings stems from these issues of measurement. Caution is required in the way that findings from the empirical research are reported, and consistent and explicit acknowledgement of the issues that the research faces when it comes to measuring personality and psychopathology has the potential to result in more realistic, consistent and sound inferences.

3.2.3 Suggestion for further study: caution around the use of statistics

Statistical analyses of association are limited in what they can tell us about the relationship between personality and psychopathology. There are specific requirements – conditions that need to be satisfied – in order for empirical statistical data to be considered robust support for

generalised theory. Those requirements are often not addressed in the current body of literature. Most notably, issues concerning the test parameters, representation of the phenomena and approaches to measurement need to be acknowledged and, where possible, addressed. PPR research in particular faces a complicated array of limitations when it comes to the use of empirical and statistical methods of inquiry, and this is predominantly due to the complexity of the phenomena that are being studied. The result is that the capacity of statistics as a tool for exploring the relationship between personality and psychopathology is *currently* limited. Section 3.3 will briefly discuss how the field might achieve more informative and well-founded PPR research while continuing to use the same statistical methodology – namely, by paying attention to the conceptual considerations involved with the study. In the meantime, there is a disparity between the information the tests can reliably be assumed to provide, and what the relevant literature purports that they are able to prove or indicate about the PPR. It is important to keep this in mind when reviewing the current PPR research.

I suggest that robust and reliable research in this area requires caution around the use of statistics as a method of inquiry. As illustrated above, the implementation of statistical analysis as a method of inquiry for the study faces multiple complications and uncertainties around the validity of the findings. This is not to say that the studies themselves are not providing useful information, and of course, many of the issues outlined above will be able to be resolved. Yet these issues appear stem from an underlying lack of clarity about the demarcation of and delineation between the related phenomena and what types of things ('objects') they are.

I propose that it is fundamental that PPR research:

- understands and states the limits of statistics for the purpose for which it is being used;
- is cautious not to overstate the findings or draw inferences that are not adequately supported by the empirical data;

- addresses the issue of how to identify, represent and measure personality and psychopathology in order to produce more robust and reliable findings.

This third point, about the consideration of the phenomena and how they can be researched, will now be discussed in section 3.3.

3.3 Conceptualisation: personality and psychopathology and their purported delineation

The final, most fundamental, limitation of current attempts to study the PPR concerns the conceptual foundations of their research. The issues outlined in section 3.2 demonstrate the shortcomings of *empirically* studying conceptually vague phenomena. They demonstrated how accurate identification, representation and measurement of the intended phenomena depend on robust conceptualisations of those phenomena. Without understanding what we really mean by 'personality' and 'psychopathology' it is hard to perform consistent and sound empirical research of the *'nature'* of their relationship. It doesn't seem reasonable to claim that we can accurately identify, represent or measure concepts or entities that we can't conceptually articulate. Of course, we can study things we can't perfectly define, but making complex claims *about* a *relationship* at least requires some clarity about the demarcation of and delineation between the related phenomena and what types of things (or 'objects') they are.

This critique is not aimed at the empirical statistical method of research. As discussed in section 3.2, statistical analysis can provide useful tools for analysing psychological phenomena, those tools just must be used appropriately. Rather, the research project itself, the currently (almost entirely) empirical study of the PPR, is hard to achieve without more open and direct consideration and specification of the study's *intended phenomena* (discussed further in what follows). This fundamental conceptual component of the project is almost

entirely overlooked within the current body of literature that sets out to study the PPR, and this is notably limiting recent empirical research and hindering the project's progression. There are two key considerations in terms of the role of better conceptual foundations within PPR research.

The first is that personality and psychopathology need demarcating – they are fuzzy, complicated phenomena, and what the terms actually pick out in the world, and why, is open to dispute. Their boundaries are not straightforward, and they are certainly not self-evident or clearly naturally defined by the way the world is. This in and of itself is not an issue. But it means that those who purport to study the phenomena, especially if doing so empirically, need to demarcate what it is that they purport to be studying – *their working conceptualisations* of personality and psychopathology. The importance of doing so and the relevance to the success of PPR research is discussed in section 3.3.1.

The second concerns disentangling personality and psychopathology from each other. As the research has found, they are deeply interrelated, and at times, very similar phenomena. Why PPR research requires better delineation between personality and psychopathology, and the implications of not doing so, are discussed in section 3.3.2.

I suggest that robust and reliable study of the PPR requires that these fundamental conceptual components of the study are addressed. Personality and psychopathology are conceptually vague and complex phenomena and attempts to make progress in terms of understanding the relationship between 'personality' and 'psychopathology' will be inherently limited so long as we are unclear about how to *conceptualise* the two phenomena in the first place.

3.3.1 Demarcating personality and psychopathology

There are numerous possible ways to understand what makes something an instance of personality, and what makes something an instance of psychopathology. They are conceptually nebulous; their general parameters are disputed and open to multiple different interpretations. Literature presents various different approaches to defining personality, and several different 'structures' are endorsed as possible ways to conceptualise personality traits. Moreover, there are an array of definitional problems with refining the phenomenon. Examples include: the distinction between character, temperament and personality; the role of developed versus innate tendencies; and taxonomic decisions (identifying traits and profiles) (see Lemery, Essex & Smider, 2002, p. 867; Rettew & McKee, 2005, p. 15-20; Boag, 2015, p. 36-37). Psychopathology is even more diverse and complicated, with conceptual questions concerning the most appropriate way to define mental disorder (an area of ongoing dispute) being characteristically hard to answer. Putting personality aside, what it requires for something to be a mental disorder is a difficult and unresolved problem in itself – independently of the problem of its interaction, or lack thereof, with personality. PPR research is disadvantaged in that there are very few already established and widely accepted definitional parameters for both personality and psychopathology (as discussed in section 1).

This complexity and disagreement associated with defining personality and psychopathology does not alone present an issue for personality and psychopathology research. As mentioned above, research studies can, and often do, face issues of definition and conceptualisation of their phenomena while nonetheless having a subject matter that is valuable, substantive and worthy of pursuit. But while it is both unrealistic and unnecessary to insist that research requires precise and robust *definitions* of the relevant concepts, refining the possible interpretations of their general parameters is pivotal in achieving a coherent and useful or practicable body of empirical research that is conducive to being built upon, developed and extended in future work. So far, studies of the PPR have routinely failed to articulate what

'personality' and/or 'psychopathology' refer to *within their work*. When the phenomena being studied are so widely disputed, with very few obvious boundaries or features, the burden falls on the researcher to specify how they conceptualise the phenomena they are working with – what their boundaries are (even if only roughly laid out) and what assumptions are made about the phenomena in order for them to be objects of inquiry in the study. Not doing so can lead to several difficulties.

The need for conceptual clarification. To start with, establishing good empirical methods of inquiry, and understanding the associated constraints and resolutions to issues that arise with those methods, depend on understanding the general parameters of the phenomena being studied. The ability to refer to these conceptual foundations is fundamental to developing and evaluating studies that can provide informative data about the intended phenomena of interest. Specifically, the methodological issues of section 3.2 are difficult to address and resolve without better demarcation of personality and psychopathology. For example, in establishing the 'degree of representation' (discussed in §3.2.2) of the studies' variables, the intended conceptualisations can provide a guide for determining what would suffice as a good variable. Without an understanding of what the *intended phenomena* of the study actually is, there is no reference by which we can determine the degree of representation that the study's variable(s) provides. Similarly, resolution of measurement issues, such as item overlap, (discussed in §3.2.2 as well) need to be informed by the general parameters of the phenomena of interest. We should be able to refer to the conceptual articulation or demarcation of the phenomena of interest to inform the empirical study.

Moreover, demarcation is central to developing a body of research that achieves core research requirements and goals. In particular, it is important for means of: *reference* – the ability to reliably refer to studies within the body of research as a way to provide evidence that doesn't require further justification; *reproducibility* – the ability to replicate the study and its

findings, perhaps with modifications or variations (e.g. to disorder type); *extendibility* – the ability for the study to be extended, and for it to *inform* future studies; and *applicability* – the ability for the study to be applied and for the relevance and appropriate applications of the findings to be evident (its usability). This is because without knowledge of how the phenomena have been understood within the study at hand, we don't have the full picture of what is being researched.

While the gold standard would be to have complete and well endorsed conceptualisations of personality and psychopathology, there is great value in the study simply stating the 'conceptual assumptions' of the empirical research, or of framing and openly discussing what personality and psychopathology refer to in the study at hand. For example, simply stating whether psychopathology is taken to be a categorical or dimensional phenomenon within each study has substantive impact on the appropriate methods and tools for research, and for determining the appropriate interpretations, extensions, and so on, of the findings of that work. DSM-III, for example, is not an appropriate tool for measuring psychopathology as a dimensional phenomenon, as it classifies mental disorder as an 'all-or-none' categorical phenomenon. If, in future, we were to universally conceptualise mental disorder as an entirely dimensional phenomenon, it would be difficult to find out which studies and findings remained relevant, and what we know about the link between personality and psychopathology conceptualised *in that way*.

A major limitation of current PPR research is that personality and psychopathology are not adequately demarcated within the study. Not only is this a problem it itself, but it underlies some of the most problematic issues identified in the use of empirical statistical methods of inquiry.

3.3.2 Delineating personality and psychopathology

A second conceptual issue lies in how we *separate* personality and psychopathology as concepts. Basically, we need to unravel personality from psychopathology and delineate between the two other in order to implement informed research of their relationship. This is for several reasons.

The first reason is that empirical research of how the phenomena are related depends on an understanding of their distinction. More precisely, empirical research of a relationship between two phenomena requires that they can be *reliably and discretely identified*. It is one thing to simply identify a relationship, but making claims about the way in which two phenomena are related is fruitless (or premature) if we are not able to identify how they are distinct. The fine grain analysis required for studying *how* or *why* the relationship occurs relies on the ability to precisely and accurately delineate the phenomena and individually identify them in the world. Thus, empirical studies that attempt to understand, describe and/or explain the relationship between personality and psychopathology depend on being able to tease the two concepts apart. Not only do we need to better delineate them, it is important to understand how they differ; to identify what it is that distinguishes them. Currently there are no adequate explanations for how we draw the line between the phenomena of interest (personality and psychopathology), and how the domains do and do not inform, influence or relate to each other *in theory*. Most obviously, we face a problem of how to sort behaviours – by degree, kind, aetiology, and so forth (see Lahey, 2004, p. 88), and this limits our ability to reliably *individually* identify personality and psychopathology in the world for empirical study. Currently, personality and psychopathology are so conceptually intertwined that empirically studying their relationship is difficult because they are difficult to individually identify.

Moreover, a second reason that personality and psychopathology require delineation is that the empirical data alone are inconclusive, and provide an incomplete picture of the relationship without conceptual support. A compelling and satisfactory theory or model of the PPR, will, to some extent, be determined by its alignment with the 'conceptual facts'. More specifically, interpreting and applying the empirical statistics is dependent on the type of interaction that is conceptually possible, and that is determined by how personality and psychopathology are *conceptually* similar and distinct. Without the conceptual foundations that outline what distinguishes personality from psychopathology, there is no way to establish what would count as good empirical evidence of a particular theory or model of their relationship. Regardless of what the empirical evidence tells us, a spectrum relationship between personality and psychopathology is *only possible* if personality and psychopathology are conceptualised as quantitatively, not qualitatively, distinct. To say that personality and psychopathology fall on a dimensional spectrum is to say that they differ by degree, not kind, and this is to make a *conceptual* claim about the phenomena. No degree of association, or any similar empirical statistic, can determine this alone. Thus, one of the key reasons that better conceptual analysis is required for fruitful study of the PPR is that the empirical data alone are inconclusive.

To illustrate this point, let's recall the example case of section 3.2.1: the longitudinal study of autism and extroversion in children and adolescents. That example was one in which a hypothetical study found extraversion in young children to be strongly negatively linked with the development of Autism, and the considered inferences were:

- *inference A*: extraversion causes or has a predisposing influence on Autism
- *inference B*: a common variable is responsible for both extraversion and Autism
- *inference C*: a particular neurobiological structure (or even a particular gene) is a latent manifestation of Autism and it is the presence of this that causes children to have low

extraversion, with but the surface or 'outward' manifestations of Autism not presenting until later in development

- *inference D*: the individuals first had Autistic traits or symptoms, which caused low extraversion, and only later in their lives they presented sufficient symptoms (perhaps due to the decreased extraversion) to meet diagnostic criteria of Autism.

Considering the latter two potential explanations *inference C* and *inference D*, gives rise to conceptual and methodological problems. In the first case, there are problems about defining psychopathology as a concept – can it be 'latent'? In the second case, there are problems with measurement (the scales used to assess for the presence of the variable) – can we really identify psychopathology in this way? Can we extract items that assess the personality trait in question from the measure of the psychopathological condition in question?

These alternative interpretations may be critiqued on several grounds. For example, it may be argued that mental disorders cannot be latent, and therefore *inference C* is not a reasonable inference based on the results of the study, but the point is that this is an argument that needs to be made, or at the very least a point that needs to be stated, and within current literature, that is not done. It is a genuine position to hold that mental disorders can be latent (Malgaroli, 2018, p. 3; The Canyon, 2020, para. 3; Borsboom & Cramer, 2013, p. 95) and thus in order to consider it a reason to infer support for any one model as any more likely than any other, one must, at the very least, specify that they believe we should not consider it possible for mental disorder to be latent.

These final two inferences are not obvious or straightforward interpretations of the findings; they give rise to many questions about what possibilities are consistent with our understandings of the phenomena. But my point is that it is important to acknowledge they are reasonable suggestions, and the questions that arise do not have obvious answers. Unless conceptual and ontological clarifications are made they are no less acceptable as interpretations

of the findings of the study than the more commonly assumed ones (in this case, *inference A*). There is not justification for assuming that one explanatory model is any more validated than any other based *solely* on these types of results. Unique support for a model, and conclusive progress concerning the 'nature' of the PPR, requires *conceptual* 'evidence' as well as empirical.

The lack of delineation between personality and psychopathology presents a second key conceptualisation-based limitation of current PPR research. Better conceptual consideration for how we draw the line between the two could have substantial positive implications for the ability of PPR research to progress.

3.3.3 Suggestion for further study: conceptual consideration

I have argued that the foundational issue that underlies the current study of the PPR is a lack of well-developed conceptual foundations. Some of the key barriers and limitations of the current study, concerning its design and aims, methods, and objects, stem from underdeveloped conceptual foundations. Most notably, there is a lack of clarity about the *distinction* between 'personality' and 'psychopathology', as well as an important lack of definitional parameters for how we *conceptualise* the concepts *individually*.

Although it is not uncommon for a field of research that faces issues of definition, distinction and inadequate tools of research concerning its concepts or 'objects of study', to nonetheless have a subject matter that is valuable, insightful (able to make advances), and worthy of pursuit, it is important that those issues are well understood, and that the field nonetheless has a clear understanding of what assumptions are made in order for empirical research to be possible. The problem in the case of recent PPR research is that the studies' results seem to be heavily contingent on the possible resolutions to those in-clarities, but it is

not made clear what assumptions and theories the studies adopt (particularly in respect to the definition and separation of personality and psychopathology) when they perform their research. We need to understand the study's conceptualisations of the phenomena in order to know how they work as variables within the study, how they should be measured, and what the study's results can tell us. Not adequately addressing this is limiting the success of recent research.

Unfortunately, it does seem that personality and psychopathology are just difficult to study. They have a range of properties which make them very difficult to grasp both empirically and conceptually. The way in which personality and psychopathology exist in the world – their ontological status as concepts or kinds – is unclear or often not addressed at all. This, along with the fuzzy and composite nature of the concepts, and their current overlap and interwovenness, makes for a confusing study of their relationship. Yet this inherent complexity of personality and psychopathology just means specific conceptual considerations are central to the study of their relationship. So far, this conceptual component is almost entirely overlooked in PPR literature.

Thus, I suggest that robust and reliable research in this area would benefit from development of the conceptual foundations upon which it has emerged, but most importantly, it requires that studies to frame their work (including their aims and results) with respect to the conceptual considerations required in their research. More open and direct consideration and specification of the study's *intended phenomena* would likely address some of the methodological barriers (outlined in §3.2) that restrict the ability of the statistical analyses to provide valid and informative finings. Empirical study alone is not well suited to achieving the aims of the research project. Thus, considering the conceptual possibilities has the potential to significantly advance our understanding of the possibilities of what is going on within the PPR.

In sum, the necessary conceptual tasks and considerations are, in my view, the following: *acknowledgement of the conditionality of the empirical findings* – that is, recognition of the fact that the findings are conditional on their conceptual foundations/underpinnings; *disclosure of the conceptual landscape that the empirical studies are embedded within* – the individual studies should be placed/framed within the conceptual context that they work within); and *a parallel conceptual study of the PPR*, considering the kinds of things personality and psychopathology are, how they can be quantified, how they are best delineated.

Conclusion

Much valuable work has been undertaken in an effort to understand the relationship between personality and psychopathology. With the establishment of a relationship now entrenched, focus has turned to its nature. It is that study into the nature of the relationship that has been my focus, with particular consideration as to *how* we go about studying the relationship. In doing so, I have had three key intentions: to provide an overview of the recent body of literature and research involved with the study; to evaluate and critique the scientific process of that work; and to make suggestions about what is required for robust and reliable study in this area.

An overview of recent literature and research discussed the way the PPR study has been undertaken so far, including the proposed models and empirical methods of research. Analysis of that recent work revealed a fundamental lack of clarity about what it means to study the nature of the relationship. There is no one clear research project to engage with, and multiple distinct tasks are being confused and conflated. I have provided an overview and analysis of what is involved with current studies of the nature of the PPR, but it has not been my intention to recommend any one model over the others or to suggest the most likely account of the 'nature' of the relationship. I have suggested that the question *what is the nature of the relationship?* is poorly understood and difficult to interpret, and thus I believe it would be unhelpful and contradictory for me to speculate an answer to that question. Instead, my aim has been to discuss *how* we might go about achieving a better understanding of the PPR. To this end, I have provided an evaluation and critique of the recent PPR study. I have argued that three key limitations have impacted the body of research to date, and from that, have made corresponding suggestions about what is required for more robust and reliable research in this area. These limitations and suggestions relate to the research project, empirical and statistical

methodology, and conceptual foundations of the study. I have recommended that successful PPR research requires: a) a clear research project; b) caution around the use of empirical and statistical methods of inquiry; and c) consideration of the conceptual foundations or context of the study.

My hope is that this evaluation does not come across as negative or unconstructive. The goal has been to review the process of recent research in order to identify what is required for future successful research. The focus has thereby been on the limitations of the recent study, but that is not to say I think the study has been poorly executed or is of little value. Ultimately, studying the PPR is an extremely difficult task. Personality and psychopathology are not simple objects of inquiry – they are conceptually vague and relatively poorly understood – and that alone makes their relationship a particularly complicated thing to research, especially with empirical methods. Robust and reliable study in this area will require addressing, and hopefully overcoming, each of the current limitations outlined above. Most importantly, the conceptual components of the research must be considered. Until we are clear about how to conceptualise the two phenomena, attempts to make progress in terms of understanding the relationship between 'personality' and 'psychopathology' will be inherently limited. PPR research would greatly benefit from extending its methods beyond purely empirical study in order to incorporate more conceptual methods – considering the kinds of things personality and psychopathology are, how they can be quantified, how they are best delineated.

Finally, collating and summarising the PPR literature has made it clear that the notion of there being a unique, analysable 'nature' of the relationship is misleading. It gives the impression of a single 'essential' thing to discover, despite the fact that almost all leading researchers in the field state that it is unlikely that there will be a single model able to account for the complex

interaction between the two fugitive concepts. My argument that both description and explanation are importantly different tasks inherent within the PPR study provides one reason to doubt that there is such a single 'nature'. Further undermining that view is the diversity and complexity of our current mental disorder concept. The difficulty of achieving one general understanding of how different disorders link to personality becomes apparent if we compare schizophrenia, anxiety and alcoholism. To start with, it is possible that those three disorders are caused by entirely different processes, and if that is the case, it seems unreasonable to assume that there will be one single process or mechanism that causes their relationship with personality. Thus, achieving a better conceptual understanding of personality and psychopathology as individual entities has the promise of providing a more comprehensive and nuanced picture of what is involved in beginning to understand the complexities of the PPR.

Gaining an understanding of the complexities and various elements of the relationship between personality and psychopathology is invaluable work. Achieving better understandings in this area would significantly advance mental disorder discussions, having an impact on the way that mental disorder is understood, not only in academia, but also in societal, medical and clinical contexts. Consequently, emerging research in this area has the potential to radically change the way that we do things in the field. For example, it could have implications for theories about the aetiology and taxonomy of mental disorder, preventing disorder, the way individuals are diagnosed and treated by professionals, and the way we approach educating and parenting children. It is for this reason that careful and well-informed research is especially important, with the appropriate scientific method(s) and necessary conceptual considerations openly discussed, scrutinised and, where required, amended. This thesis has discussed how that can be achieved, making three key suggestions about what is required for robust and reliable study of the relationship between personality and psychopathology. I believe that the single

most important thing to bear in mind as research in this area progresses is that PPR research is difficult to perform. It will likely face many challenges beyond the ones that I have discussed in this thesis, but that alone will not pose a serious issue as long as those challenges are well understood and openly disclosed, and research is presented in a way that does not overstate the strength or implications of its findings.

References

Akiskal, H. S., Hirschfeld, R. M., & Yerevanian, B. I. (1983). The Relationship of Personality to Affective Disorders: A Critical Review. *Archives of General Psychiatry, 40*, 801-810.

Allison, P. D. (1999). *Multiple Regression: A Primer*. Thousand Oaks, California: Pine Forge Press.

Allison, P. D. (2014). Prediction vs. causation in regression analysis. Retrieved from: https://statisticalhorizons.com/prediction-vs-causation-in-regression-analysis

Arpaly, N. (2005). How it is not "just like diabetes": Mental disorders and the moral psychologist. *Philosophical Issues*, *15*, 282-298.

Boag, S. (2015). Personality assessment, 'construct validity', and the significance of theory. *Personality and Individual Differences, 84*, 36-44.

Boorse, C. (1976). What a theory of mental health should be. *Journal for the Theory of Social Behaviour*, *6*(1), 61-84.

Borsboom, D., & Cramer, A. O. (2013). Network analysis: an integrative approach to the structure of psychopathology. *Annual review of clinical psychology*, *9*, 91-121.

Brown, T. A., & Barlow, D. H. (2005). Dimensional versus categorical classification of mental

disorders in the fifth edition of the Diagnostic and statistical manual of mental disorders and beyond: Comment on the special section. *Journal of abnormal psychology*, *114*(4), 551-556.

Busfield, J. (2011). *Mental illness*. Cambridge, UK: Polity Press.

Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, Personality, and the Mood and Anxiety Disorders. *Journal of Abnormal Psychology*, 103-116.

Clayton, P. J., Ernst, C., & Angst, J. (1994). Premorbid personality traits for men who develop unipolar or bipolar disorders. *European Archives of Psychiatry and Clinical Neuorscience*, 340-346.

Coghill, D., & Sonuga-Barke, E. J. (2012). Annual research review: categories versus dimensions in the classification and conceptualisation of child and adolescent mental disorders–implications of recent empirical study. *Journal of Child Psychology and Psychiatry*, *53*(5), 469-489.

Cooper, R. (2013). What's special about mental health and disorder? In S. M. Downes, & E. Machery (Eds.), *Arguing about human nature: Contemporary debates* (pp. 487-499). London: Routledge.

Craver, C., & Tabery, J. (2015). Mechanisms in Science. In E. N. Zalta (Ed.), *The Stanford encyclopedia of philosophy*. Retrieved from: https://plato.stanford.edu/entries/science-mechanisms/

Davies, W. & Levy, N. (2017). Mental Illness, Reasons-Responsiveness, and the Social Nature of Mind. Unpublished manuscript.

De Pauw, S. S., & Mervielde, I. (2011). The role of temperament and personality in problem behaviors of children with ADHD. *Journal of Abnormal Child Psychology*, *39*(2), 277-291.

De Pauw, S. S., Mervielde, I., Van Leeuwen, K. G., & De Clercq, B. J. (2011). How Temperament and Personality Contribute to the Maladjustment of Children with Autism. *Journal of Autism and Developmental Disorders*, 196-212.

Depue, R. A. (1995). Neurobiological factors in personality and depression. *European Journal of Personality*, *9*(5), 413-439.

DeYoung, C. G., Quilty, L. C., & Peterson, J. B. (2007). Between facets and domains: 10 aspects of the Big Five. Journal of personality and social psychology, 93(5), 880-896.

Durbin, C. E., & Hicks, B. M. (2014). Personality and psychopathology: A stagnant field in need of development. *European Journal of Personality*, *28*(4), 362-386.

Fajkowska, M., & Kreitler, S. (2018). Status of the trait concept in contemporary personality psychology: Are the old questions still the burning questions?. *Journal of personality*, *86*(1), 5-11.

Freebody, P., & Freiberg, J. (2006). Cultural Science and qualitative educational research: Work ‘in the first place’on the morality of classroom life. *International Journal of Qualitative Studies in Education*, *19*(6), 709-722.

Frick, P. J. (2004). Integrating research on temperament and childhood psychopathology: Its pitfalls and promise. *Journal of Clinical Child and Adolescent Psychology*, *33*(1), 2-7.

Frigg, R., & Hartmann, S. (2020). Models in Science. In E. N. Zalta (Ed.), *The Stanford encyclopedia of philosophy*. Retrieved from: https://plato.stanford.edu/entries/models-science/

Garon, N., Bryson, S. E., Zwaigenbaum, L., Smith, I. M., Brian, J., Roberts, W., & Szatmari, P. (2009). Temperament and its relationship to autistic symptoms in a high-risk infant sib cohort. *Journal of abnormal child psychology*, *37*(1), 59-78.

Haig, B. D. (2014). *Investigating the psychological world: Scientific method in the behavioral sciences*. Cambridge, MA: MIT Press.

Haywood, H. C., & Raffard, S. (2017). Cognition and Psychopathology: Overview. *Journal of Cognitive Education and Psychology*, *16*(1), 3-8.

Hengartner, M. P., Tyrer, P., Ajdacic-Gross, V., Angst, J., & Rössler, W. (2018). Articulation and testing of a personality-centred model of psychopathology: evidence from a longitudinal community study over 30 years. *European Archives of Psychiatry and Clinical Neuorscience*, 443-454.

Ivarsson, T., & Winge-Westholm, C. (2004). Temperamental factors in children and adolescents with obsessive-compulsive disorder (OCD) and in normal controls. *European child & adolescent psychiatry*, *13*(6), 365-372.

Katon, W., Lin, E., Von Korff, M., Bush, T., Walker, E., Simon, G., & Robinson, P. (1994). The predictors of persistence of depression in primary care. *Journal of affective disorders*, *31*(2), 81-90.

Klein, D. N., Kotov, R., & Bufferd, S. J. (2011). Personality and Depression: Explanatory Models and Review of the Evidence. *Annual Review of Clinical Psychology*, 269-295.

Klein, M. H., Wonderlich, S., & Shea, T. M. (1993). Models of relationships between personality and depression: toward a framework for theory and research. In D. J. Kupfer, M. H. Klein, & T. M. Shea, *Personality and depression: A current view* (pp. 1-54). New York: Guilford.

Kopala-Sibley, D. C., Klein, D. N., Perlman, G., & Kotov, R. (2017). Self-Criticism and Dependency in Female Adolescents: Prediction of First Onsets and Disentangling the Relationship Between Personality, Stressful Life Events, and Internalising Psychopathology. *Journal of Abnormal Psychology*, 1029-1043.

Kozak, M. J., & Cuthbert, B. N. (2016). The NIMH research domain criteria initiative: background, issues, and pragmatics. *Psychophysiology*, *53*(3), 286-297.

Krueger, R. F. (1999). Personality traits in late adolescence predict mental disorders in early adulthood: a perspective-epidemiological study. *Journal of personality*, *67*(1), 39-65.

Krueger, R. F., & Eaton, N. R. (2010). Personality traits and the classification of mental disorders: Toward a more complete integration in DSM–5 and an empirical model of psychopathology. *Personality Disorders: Theory, Research, and Treatment*, *1*(2), 97.

Krueger, R. F., & Tackett, J. L. (2003). Personality and psychopathology: Working toward the bigger picture. *Journal of Personality Disorders*, *17*(2: Special issue), 109-128.

Lahey, B. (2004). Commentary: Role of temperament in developmental models of psychopathology. Journal of Clinical Child & Adolescent Psychology, 33(1), 88-93

Lemery, K. S., Essex, M. J., & Smider, N. A. (2002). Revealing the relation between temperament and behavior problem symptoms by eliminating measurement confounding: Expert ratings and factor analyses. *Child Development*, *73*(3), 867-882.

Lilienfeld, S. O. (2014). The Research Domain Criteria (RDoC): An analysis of methodological and conceptual challenges. *Behaviour research and therapy*, *62*, 129-139.

Lo, M. T., Hinds, D. A., Tung, J. Y., Franz, C., Fan, C. C., Wang, Y., ... & Sanyal, N. (2017). Genome-wide analyses for personality traits identify six genomic loci and show correlations with psychiatric disorders. *Nature genetics*, *49*(1), 152-156.

Maher, B. A., & Maher, W. B. (1994). Personality and psychopathology: A historical perspective. *Journal of Abnormal Psychology*, *103*(1), 72-77.

Malgaroli, M. (2018). *The Centrality of Sadness: Networks of Depression, Grief, and Trauma Symptoms in a Spousally Bereaved Sample* (Doctoral dissertation, Columbia University).

Martel, M. M., & Nigg, J. T. (2006). Child ADHD and personality/temperament traits of reactive and effortful control, resiliency, and emotionality. *Journal of Child Psychology and Psychiatry*, *47*(11), 1175-1183.

McNally, R. J. (2011). *What is mental illness?*. Harvard University Press.

Mental Health (Compulsory Assessment and Treatment) Act, New Zealand Statutes. (1992).

Murphy, D. (2015). Philosophy of Psychiatry. In E. N. Zalta (Ed.), *The Stanford encyclopedia of philosophy.* Retrieved from: https://plato.stanford.edu/entries/psychiatry/

Nigg, J. T. (2006). Temperament and Development Psychopathology. *Journal of Child Psychology and Psychiatry*, 395-422.

Perring, C. (2010). Mental illness. In E. N. Zalta (Ed.), *The Stanford encyclopedia of philosophy.* Retrieved from: https://plato.stanford.edu/entries/mental-illness/

Radden, J. (2019). Mental disorder (illness). In E. N. Zalta (Ed.), *The Stanford encyclopedia of*

philosophy. Retrieved from: https://plato.stanford.edu/entries/mental-disorder/

Ramirez, E. (n.d.). Philosophy of Mental Illness. In J. Fieser and B. Dowden (Eds.), *The internet encyclopedia of philosophy*. Retrieved from: https://www.iep.utm.edu/mental-i/

Reese, H. W. (1999). Explanation is not description. *Behavioral Development Bulletin*, *8*(1), 3-7.

Rettew, D. C. (2009). Temperament: risk and protective factors for child psychiatric disorders. In B. J. Kaplan, V. A. Kaplan & P. Ruiz (eds.) *Kaplan & Sadock's Comprehensive Textbook of Psychiatry 9th ed* (pp. 3432-3443). Philadelphia, Pennsylvania: Lippincott Williams & Williams.

Rettew, D. C. (2010). Refining our diagnostic system—cake or comorbid bread and fudge? *Journal of the American Academy of Child & Adolescent Psychiatry*, *49*(5), 441-443.

Rettew, D. (2013). *New thinking about the boundary between traits and illness.* New York City, New York: W. W. Norton & Company.

Rettew, D. (2014). Do you have a disorder or just a trait? *Psychology Today, 47*(1), 42-43.

Rettew, D. C., Doyle, A. C., Kwan, M., Stanger, C., & Hudziak, J. J. (2006). Exploring the

boundary between temperament and generalized anxiety disorder: a receiver operating characteristic analysis. *Journal of anxiety disorders*, *20*(7), 931-945.

Rettew, D. C., & McKee, L. (2005). Temperament and its role in developmental psychopathology. *Harvard review of psychiatry*, *13*(1), 14-27.

Sarbin, T. R. (1967). On the futility of the proposition that some people be labeled" mentally ill.". *Journal of Consulting Psychology*, *31*(5), 447-453.

Shiner, R. L., Masten, A. S., & Tellegen, A. (2002). A developmental perspective on personality in emerging adulthood: Childhood antecedents and concurrent adaptation. *Journal of Personality and Social Psychology, 83*(5), 1165-1177.

Szasz, T. S. (1960). The myth of mental illness. *American psychologist*, *15*(2), 113-118.

Tackett, J. (2006). Evaluating mofels of the personality-psychopathoogy relationship in children and adolescents. *Clinical Psychology Review*, 584-599.

The Canyon. (2020). How LSD abuse complicates mental health issues. Retrieved from: https://skywoodrecovery.com/lsd-abuse/how-lsd-abuse-complicates-mental-health-issues/

Van Leeuwen, K., Mervielde, I., De Clercq, B., & De Fruyt, F. (2007). Extending the spectrum idea: Child personality, parenting and psychopathology. *European Journal of Personality, 21*(1), 63-89.

Wakefield, J. C. (1992). The concept of mental disorder: on the boundary between biological facts and social values. *American Psychologist*, *47*(3), 373.

Wakefield, J. C. (2007). The concept of mental disorder: diagnostic implications of the harmful dysfunction analysis. *World Psychiatry*, *6*(3), 149-156.

Watson, D., & Clark, L. A. (1994). Introduction to the special issue on personality and psychopathology. *Journal of Abnormal Psychology*, *103*(1), 3-5.

Watson, D., & Clark, L. A. (1995). Depression and the melancholic temperament. *European Journal of Personality*, *9*(5), 351-366.

Watson, D., Clark, L. A., & Harkness, A. R. (1994). Structures of personality and their relevance to psychopathology. *Journal of abnormal psychology*, *103*(1), 18-31.

Watson, D., Gamez, W., & Simms, L. J. (2005). Basic dimensions of temperament and their relation to anxiety and depression: A symptom-based perspective. *Journal of Research in Personality*, *39*(1), 46-66.

Widiger, T. A., & Clark, L. A. (2000). Toward DSM—V and the classification of psychopathology. *Psychological bulletin*, *126*(6), 946-963.

Widiger, T. A., & Gore, W. L. (2014). Mental disorders as discrete clinical conditions: Dimensional versus categorical classification.

Widiger, T. A., & Samuel, D. B. (2005). Diagnostic categories or dimensions? A question for the Diagnostic and statistical manual of mental disorders. *Journal of abnormal psychology*, *114*(4), 494-504.

Widiger, T. A., & Smith, G. T. (2008). In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of Personality Theory and Research (3rd ed.)* (pp. 743-769). New York, NY: The Gilford Press.

Woodward, J. (2014). Scientific Explanation. In E. N. Zalta (Ed.), *The Stanford encyclopedia of philosophy.* Retrieved from: https://plato.stanford.edu/entries/scientific-explanation/

World Health Organisation. (2001). World health report: Mental disorders affect one in four people. Retrieved from https://www.who.int/whr/2001/media_centre/press_release/en/