Causal explanations of mental illness and perceived credibility of psychological therapy

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UCL Doctorate in Clinical Psychology

UCL Doctorate in Clinical Psychology Thesis declaration form

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

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Overview

This thesis examines the impact of causal explanations of psychiatric disorders; specifically, how biological versus psychosocial explanations of psychosis impact treatment recommendations and how this is modified by presenting evidence of treatment effectiveness.

Chapter 1, the conceptual introduction, provides a narrative review of the evidence regarding the effects of biological conceptualisations of psychopathology on different beliefs and the use of malleability-focused interventions to challenge such thinking. The review expanded the existing understanding of factors such as neuroessentialism, stigmatising attitudes and prognostic pessimism, and identified additional areas relevant to this topic. These included the potential use of empirical neuroimaging findings to demonstrate the neuroplasticity of biological factors involved in psychiatric disorder, in addition to the measurement of constructs such as treatment appraisals and pre-existing illness perception beliefs. It also identified clinicians as a group that is under-researched and where the impact of a malleability-focused intervention is poorly understood.

Chapter 2, the empirical paper, investigates the impact of presenting neuroplasticity evidence following psychological therapy (compared to control information) using an online experiment with the general public (study 1) and clinicians (study 2). The study also attempts to replicate previous findings regarding the impact of biological versus psychosocial descriptions of psychosis. When the general population is presented with any type of evidence that CBTp is an effective treatment, it can reduce a bias toward medication elicited by biological explanations. The effect amongst clinicians is more varied, depending on their training background.

Chapter 3, the critical appraisal, considers how my previous experience led to an interest in the study, before reflecting on methodological issues which arose whilst conducting

it. Issues such as study design, recruitment and contextual factors which impacted data collection will be discussed, before personal reflections are finally offered on how the research has impacted my development as a researcher and clinician.

Impact Statement

Chapter 1 of this thesis presents a conceptual introduction that suggests that biological explanations of psychopathology can have a number of unintended negative implications for attitudes amongst the general public, clinicians, and patients themselves. The introduction also highlights that interventions aimed at challenging such attitudes have focused on providing information about the malleability of biological factors involved, in addition to effective treatments which are available. Emerging evidence of psychological treatments leading to measurable neurobiological changes is reviewed and concludes that such evidence may be useful in challenging the treatment biases which can otherwise emerge from essentialist and overly simplistic biological explanations of mental health difficulties.

Chapter 2 investigates the effect of biological compared to psychosocial descriptions of psychopathology on treatment assumptions and the possibility of challenging these assumptions by providing evidence of the neuroplasticity of biological factors involved. The study indicates that the perceived helpfulness of psychological therapy is open to influence and that presenting evidence of CBT effectiveness will make the public more likely to choose it as a treatment option. This has important implications for clinical practice where brief interventions which target perceptions of the helpfulness of psychological treatment and the possibility of change may increase active engagement in CBTp, thereby increasing the likelihood of a successful outcome.

Another key finding was that clinicians need to be aware that the way in which they conceptualise a patient's mental health difficulties can not only implicitly influence their own treatment recommendation, but also impact the type of treatment a patient perceives as helpful. This could then have implications for treatment adherence and a patient's willingness to try new treatments in clinical practice.

The study also shows that a person's pre-existing illness perception beliefs and even political beliefs can influence how amenable they are to new treatment evidence. This has implications for clinical practice, where the cultivation of incremental beliefs can be used as a mechanism for change. At relevant services such as early intervention for psychosis settings, highlighting the malleability of biological factors involved in one's condition can increase their sense of control over their condition and give them increased hope for recovery. Finally, the study found a clinician's training background has important implications for the type of treatment they recommend, with psychiatrists showing a preference for medication even when presented with evidence that psychological therapy targets the biology of the condition described. This has implications for clinician training courses, where an emphasis on the scientist-practitioner model can underline the importance of empirical evidence informing clinical practice.

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Chapter 1: Conceptual Introduction

Biological explanations of psychopathology: implications for attitudes and evidence of neuroplasticity as a possible intervention

Abstract

In recent years there has been an increased focus on mental health neuroscience, which examines psychopathology at the neurobiological and genetic level. Emerging from this movement, biological conceptualisations of psychopathology have become increasingly dominant, both at the societal level and in the clinical context.

This conceptual introduction reviewed literature investigating the impact of biological explanations of psychopathology on attitudes amongst the general public, clinicians and patients themselves. Relevant literature was collected from a variety of sources, including the databases PsycINFO and Web of Science, in addition to books, articles and policies relevant to the topic. The conceptual introduction provides a detailed introduction to the research topic, defining concepts which are central to understanding the impact of biological conceptualisations such as "biomarkers", "essentialist thinking", "mind-brain dualism" and "fervent monism". The review of literature identifies a number of unintended negative consequences of biological explanations and also examines research which has attempted to mitigate such effects. Gaps in the literature are subsequently identified, which the empirical research paper (see Chapter 2) aimed to address.

Section 1 Introduction

Biological explanations of psychiatric disorders have become increasingly dominant in recent decades, fuelling concerns there has been a disproportionate emphasis placed on biological factors within the biopsychosocial model of mental health difficulties (Engel, 1977)When psychopathology is explained in biological terms, research has shown that there are negative implications in both social and clinical contexts. A large proportion of the ramifications stem from the essentialist thinking that they unfortunately evoke; that psychiatric disorders are relatively immutable and are caused by biological dysfunction in a deterministic manner (Lebowitz & Appelbaum, 2019). Education interventions aimed at challenging such thinking have focused on providing information about the malleability of biological factors involved, in addition to effective treatments which are available. This project aims to build on the existing research in several ways. Whilst much malleability-focused research has focused on changing attitude constructs such as blame, prognostic pessimism and desired social distance, there has been a lack of studies targeting treatment recommendations, particularly amongst clinicians. Furthermore, specific individual difference factors which might modulate the effects of a malleability-focused intervention, such as illness perception beliefs and political attitudes, have not previously been investigated and so will be included in the present study. Finally, presenting recent neuroimaging evidence is an approach which has not yet been used to emphasise the malleability of biological factors involved in psychiatric disorders.

This project aims to test whether treatment assumptions which result from biological explanations of psychopathology can be impacted by presenting recent biological evidence of neuroplasticity following psychological therapy. It is possible that psychological therapy will be perceived as more effective when it is presented as targeting the neurobiology of the mental illness described. If effective, brief interventions which target perceptions of the helpfulness of

psychological treatment could possibly increase engagement in therapy in relevant settings. This conceptual review will consider the essential research and theoretical background motivating this study. It will begin by giving context to the prevalence of biological conceptualisations of psychopathology (section 2). In section 3, the implications of this phenomenon in both social and clinical contexts will be outlined. Recent evidence regarding neuroplasticity following psychological therapy will be covered in section 4, before the latest research in education interventions is reviewed in section 5, highlighting gaps in the knowledge that the present study aims to address.

Section 2 Background and increasing prevalence of biological accounts

History

There have been dramatic changes in how psychiatric disorders have been conceptualised over the years, from early explanatory accounts which attributed mental disturbance to demonic possession, to contemporary biological explanations which emphasise the role of neurobiology and genetics in psychopathology (Pietikainen, 2015; Deacon, 2013). Biological explanations, which have become increasingly dominant in recent decades, emerged from the so-called psychopharmacological revolution of the twentieth century (Lebowitz & Appelbaum, 2019). Beginning in the 1950s, psychiatrists used chlorpromazine to control the positive psychotic symptoms of schizophrenia, citing it as the first broadly effective and non-invasive form of treatment. In subsequent decades, monoamine oxidase inhibitors, tricyclics and later selective serotonin reuptake inhibitors (SSRIs) were used to treat depression, whilst sedative hypnotics such as barbiturates and benzodiazepines were prescribed for anxiety. Further research identified particular neurotransmitters as being targeted by selected drugs and as a result, psychiatrists increasingly came to view themselves as treating biological illnesses using

pharmacological interventions. The prevailing use of psychotropic medication to treat psychiatric disorders is a trend that continues today (Lebowitz & Appelbaum, 2019).

Shift toward biological explanations

Progress in the development of psychotropic medication, coupled with the reconceptualisation of psychiatric disorders as diseases involving neurotransmitters and receptors, resulted in a dramatic shift in treatment preferences by the close of the twentieth century (Lebowitz & Appelbaum, 2019). In the U.S., data from a nationally representative survey highlighted how in 1998, the majority of patients received a combination of psychological therapy and medication to treat mental health concerns whereas by 2007, most patients only received medication (Olfson & Marcus, 2010). This shift in treatment trends has been accompanied by a more favourable change in attitudes toward psychiatric medication over time. Between 1998 and 2006, although Americans' perceived risk of psychotropic medication did not alter significantly, they were found to hold stronger beliefs in the benefits of such drugs and were more likely to select such drugs in hypothetical scenarios (Mojtabai, 2009). This trend is echoed in other countries too, where members of the general public have become more accepting of psychotropic medication over the years and biological explanations of psychopathology have been associated with favourable attitudes toward psychiatric medication (Angermeyer, van der Auwera, Carta, & Schomerus, 2017).

The increased focus on biological explanations for psychopathology has also been reflected in U.S. funding, which has increasingly prioritised research on the neurobiology and genetics of psychiatric disorder (Hyman, 2007). In 2009, the National Institute of Mental Health (NIMH) launched the Research Domain Criteria (RDoC) in order to identify biomarkers of psychiatric disorders. The RDoC framework posits that disordered brain circuits are the cause of psychiatric disorders, strongly endorsing that targeting such brain circuits and

modifying them using medication is the correct approach to treatment (Wakefield, 2014). This represents a significant departure from the Diagnostic and Statistical Manual of Mental Disorders (5th ed.) and the International Classification of Diseases and Related Health Problems (WHO 1992), which rely on symptoms and client self-reports for diagnosis (American Psychiatric Association, 2013).

In addition to the NIMH, the National Institute on Child Health and Human Development, the National Institute on Drug Abuse (NIDA) and the National Institute on Alcohol Abuse and Alcoholism (NIAAA) have all increasingly turned their focus to neuroscientific research over the past decade. Such initiatives have fuelled concerns that there has been a disproportionate emphasis on biological conceptualisations of psychopathology at the expense of psychosocial factors (Schwartz, Lilienfeld, Meca, & Sauvigne, 2016). Furthermore, disputes between professions over the preferred level of analysis have ensued, with psychological therapists being funded in separate research "silos" to pharmacological researchers. This partitioned funding system prevents dialogue and collaboration among theoretically different healthcare professions, with pharmacotherapy and psychological therapy in competition despite evidence that a combined approach typically outperforms either treatment alone (Huhn et al., 2014; Moss et al., 2016).

Classification as brain diseases

The increased focus on neurobiology and genetics in the search for causes of psychiatric disorder appears to perpetuate a form of mind-brain dualism; the belief that the mind and the brain are separate entities (Kendler, 2005; Miresco & Kirmayer, 2006). In order for one to be the cause of the other, they must be considered distinct rather than different parts of the same entity (Lebowitz, 2014). In rejecting a mind-body dualism, we must accept that psychiatric disorders are indeed brain disorders at some level because all psychosocial phenomena are

mediated by the brain and the rest of the central nervous system, a scientifically undisputed fact (Kendler, 2005). However, it has been argued that psychiatric disorders can just as accurately be conceptualised as psychological disorders because psychological functioning is by definition impaired in psychiatric disorder (Schwartz, Lilienfeld, Meca, & Sauvigne, 2016). Indeed, conceptualising psychiatric disorders exclusively as diseases of the brain risks confusing biological mediation with biological aetiology. The fact that brain functioning enables psychiatric disorders does not necessarily mean that such disorders are caused by abnormalities in brain hardware (Schwartz, Lilienfeld, Meca, & Sauvigné, 2016). Rather, biological theories of psychiatric disorder provide a useful explanation as to how biological processes play a role in the production of abnormal psychological phenomena (Deacon, 2013).

Despite widespread claims of "biologically-based brain disease", researchers have yet to identify a simple biological cause of any major psychiatric disorder (Kendler, 2005). Indeed, Kendler (2005 pg. 1250) stated that: "the strong, clear and direct causal relationship implied by the concept of 'a gene for' does not exist for mental disorders. Although we may wish it to be true, we do not have and are not likely ever to". Neuroscience has significantly enhanced our understanding of the brain and sophisticated technologies such as molecular genetic testing and brain imaging techniques have confirmed the role of biogenetics in psychiatric disorder (Panksepp, 2004). However, despite such recent advancements, neuroscience and genetic research have failed to identify a single biological marker with enough sensitivity to reliably inform the diagnosis of any psychiatric disorder (Deacon, 2013). In fact, research has shown that major psychiatric disorders cannot be attributed to a few, but rather dozens, or potentially thousands of genetic variants (Dar-Nimrod & Heine, 2011). To illustrate the point, schizophrenia, a psychiatric disorder which has historically been conceptualised with a genetic foundation, has several thousand genetic variants associated with it (International

Schizophrenia Consortium et al., 2009). Thus, despite substantial evidence of heritability in the transmission of psychiatric disorders, neuroscience and molecular biology are far from replacing psychology in explaining psychopathology (Deacon, 2013).

Dangers of reductionism

Although there have been great advancements made from the increased focus and research on the biological level of analysis, conceptualising psychiatric disorders as brain diseases has been criticised as being reductionist (Bentall, 2006). Kendler (2014) warned against the dangers of "fervent monism" in the field of psychiatry and psychology. "Fervent monism" refers to the tendency to place a disproportionate emphasis on one explanatory level in the hierarchy of the sciences (Comte, 2002). The increased focus on biological research is a trend which seems set to remain and will further entrench beliefs about the helpfulness of biological explanations amongst clinicians and the general public (Lebowitz & Appelbaum, 2019). It raises a cause for concern because evidence suggests that both laypeople and clinicians perceive biological explanations as incompatible with other etiological explanations (Lebowitz, Ahn, & Nolen-Hoeksema, 2013; Miresco & Kirmayer, 2006). Such evidence implies that the popularity of biological explanations could result in other levels of analysis being neglected, an issue which has been raised in the field of psychology recently (Kagan, 2013).

An review of over 9,000 abstracts from international conferences on psychosis showed that 75% of presentations emphasised biological factors whereas just 5% addressed psychosocial factors (Calton, Cheetham, D'Silva, & Glazebrook, 2009). This is despite evidence that life stressors are major precipitating factors in the case of psychosis, particularly when there is a genetic predisposition (Jones & Fernyhough, 2007). Indeed, psychological problems are multifaceted and can only be fully understood by considering numerous levels of investigation beyond the neuroscientific, to including the cultural, social and motivational

(Schwartz, Lilienfeld, Meca, & Sauvigne, 2016). No part of the biopsychosocial model should be neglected or favoured over another in explaining the origins of psychological disorder (Deacon, 2013). This point not only applies to cases in which there is an over-reliance on biological factors, but is also relevant when there is an overweighting of psychosocial factors in explaining psychological disorder. To illustrate the point, the Power Threat Meaning Framework posits that psychiatric symptoms are responses to environmental threats, with such socially influenced responses serving protective functions for those affected (Johnstone & Boyle, 2018). Whilst this conceptual system has been praised for incorporating personal, social, and cultural meaning, it has also been criticised for excluding conditions such as dementia, intellectual disability and neurodegenerative disorders, in addition to the effects of hormonal imbalances, vitamin deficiencies, viral infections and autoimmune diseases (Johnstone et al., 2019).

Popularity of biological factors in wider society

The increased tendency to conceptualise psychopathology as caused by biological factors is not just restricted to the field of psychiatry and clinical psychology (Haslam & Kvaale, 2015). Over the past decade, the media has given increasing coverage to neuroscience, presenting neurobiological processes as the dominant source of mental illness (O'Connor, Rees, & Joffe, 2012). This trend has been accompanied by a corresponding shift in the public's understanding of psychiatric disorders. A recent meta-analysis found that the proportion of people who attributed psychosis and depression to genetic inheritance and brain disease dramatically increased between 1990 and 2006 (Schomerus, Matschinger, & Angermeyer, 2014). Neuroessentialism is the view that the definitive way of explaining human psychological experience is by reference to the brain and its activity (Schultz, 2018). Research has suggested

that biological explanations can elicit neuroessentialist thinking (Bennett, Thirlaway, & Murray, 2008; Kvaale, Gottdiener, & Haslam, 2013; Lam, Salkovskis, & Warwick, 2005) Once neuroessentialist beliefs have been activated, there are often negative implications for social attitudes and behaviour, which will now be explored in the next section.

To summarise: Beginning with the psychopharmacological revolution of the mid-twentieth century, biological explanations of psychiatric disorders have become increasingly dominant in recent decades (Haslam & Kvaale, 2015). By the end of the twentieth century, the reconceptualisation of psychiatric disorders as diseases involving biological dysfunction, coupled with advances in psychotropic medication, had resulted in a more favourable view of psychiatric medication to treat mental illness. In the U.S., funding bodies have prioritised the neurobiology of psychiatric disorder, with national institutions turning their focus to neuroscientific research (Hyman, 2007). Such initiatives have fuelled concerns that there has been a disproportionate focus on the biological level of explanation at the expense of psychosocial factors, with conceptualisations of psychiatric disorders as brain diseases being criticised as being reductionist (Kendler, 2014). Neuroscience has enhanced our understanding of the brain, yet it has failed to identify a biological marker which informs the diagnosis of any psychiatric disorder. Despite this, the increased focus on biological research is a trend which seems set to continue, reinforcing the degree to which clinicians depend on biological explanations in their practice (Lebowitz & Appelbaum, 2019). In addition, the media has given increasing coverage to neuroscience, thereby influencing the general public's understanding of the aetiology of psychiatric disorders (Racine, Waldman, Rosenberg, & Illes, 2010). When psychopathology is explained in biological terms it often evokes neuroessentialist beliefs about a given disorder (Haslam, 2011), the consequences of which will be considered in the next section.

Section 3 Societal and clinical implications of biological explanations

Given the exponential rise in biological explanations of psychopathology described in the previous section, an understanding of the implications in both social and clinical contexts is of paramount importance (see Lebowitz & Appelbaum, 2019, for a comprehensive review). Regarding social attitudes, research has suggested that as biological explanations have flourished, public stigma toward those affected by mental health difficulties has not abated over the years, and has possibly even exacerbated (Schomerus et al., 2012). In addition to public stigma, biological explanations may also affect clinical attitudes in a number of ways (Lebowitz & Appelbaum, 2019).

Prognostic expectations are particularly important regarding psychiatric disorders. Patients who are more optimistic about their prognoses tend to engage more in treatment, leading to an improvement in symptoms, whereas those who are more pessimistic engage less, preventing any alleviation of symptoms (Alladin, 2013; Meyer et al., 2002; Rutherford, Wager, & Roose, 2010; Tambling, 2012). Furthermore, clinicians' expectations of their patients' prognoses significantly predicts treatment outcomes (Byrne, Sullivan, & Elsom, 2006; Meyer et al., 2002). Thus, it is crucial to discern the impact that biological explanations can have on prognostic expectations among both patients and clinicians (Lebowitz & Appelbaum, 2019)

Treatment preference is another factor which might possibly be affected by biological explanations of psychopathology. As previously mentioned, there has been a surge in the number of psychiatric disorders treated with medication and a corresponding decrease in the number being treated with psychological therapy over the past few decades (Olfson & Marcus, 2010). This trend has been accompanied by a rise in the popularity of biological explanations (Pescosolido et al., 2010) and so researchers have proposed that the more clinicians and patients perceive psychopathology as being caused by biological irregularities, the more inclined they

are to choose treatments that are thought to target the biological phenomena underlying the symptoms (Lebowitz & Appelbaum, 2019).

The therapeutic alliance is another variable which is important to consider when measuring the impact of biological explanations in clinical contexts. Much research has demonstrated that a strong therapeutic alliance positively influences treatment outcomes while a weak alliance leads to attrition and poor outcomes (Horvath, Del Re, Flückiger, & Symonds, 2011; Sharf, Primavera, & Diener, 2010). The empathy a therapist feels for their patient and a patient's perceptions of their therapist's warmth and competence contribute to the strength of the therapeutic alliance (Ackerman & Hilsenroth, 2003; Elliott, Bohart, Watson, & Murphy, 2018).

Theoretical frameworks which predict the impact of biological explanations on attitudes

Much research has been conducted on the implications of the shift toward a biological understanding of psychiatric disorders. Three theoretical approaches have been proposed in the literature, each predicting disparate outcomes. Attribution theory (Schmidt & Weiner, 1988) posits that citing biogenetic causes which are beyond one's control will elicit an increase in emotions such as empathy and pity, and decrease blame associated with having a mental illness. Under attribution theory, mental illness would be perceived as comparable to a physical health condition, thereby leading to reduced stigma and discriminatory behaviours (Larkings & Brown, 2018).

Conversely, essentialism is based on biological differences between people, frequently leading to the endorsement of social stereotypes which have negative implications. Firstly, essentialism theory predicts that an endorsement of biogenetic causes might lead to individuals with psychiatric disorders being viewed as categorically different because they possess a pathological essence, resulting in a desire for social distance from those affected (Haslam &

Kvaale, 2015). In such scenarios, psychiatric disorders are viewed as relatively immutable, resulting in an increase in prognostic pessimism regarding recovery. Furthermore, as essentialist thinking is associated with endorsing social stereotypes, biological explanations are linked to the acceptance of the prevalent stereotype that those with psychiatric disorders are dangerous and unpredictable (Haslam & Kvaale, 2015).

Apart from essentialism, dehumanisation is another way in which biological explanations can have a negative impact on social beliefs and attitudes. Mechanistic dehumanisation takes place when the person is considered as lacking qualities which are indicative of human nature such as warmth, emotionality and vitality (Lebowitz & Appelbaum, 2019). Biomedically focused psychiatric practice has been criticised of mechanistic dehumanisation because it is purported to conceptualise patients' minds as malfunctioning machines, thereby adversely impacting empathy levels. (Haslam, 2006). Thus, if biological explanations result in mechanistic dehumanisation, it can lead to increased stigma in terms of social attitudes and also negatively impact the clinician-patient relationship because of reduced empathy (Lebowitz & Appelbaum, 2019).

The results of several meta analyses suggest that there is strong evidence for a 'mixed blessings' model, incorporating both potentially beneficial and harmful effects. On the one hand, biological explanations may alleviate public stigma by diminishing blame but on the other hand they can induce pessimism about prognosis and treatment response, avoidance and the perception that those affected are unpredictable and dangerous (Haslam & Kvaale, 2015). Much research has been conducted to analyse how biological explanations elicit such reactions from the general public, clinicians and the patients themselves. The empirical evidence for the effects on each cohort will now be considered in turn.

Effects among the general public attitudes

Dangerousness and unpredictability

In recent years, researchers have expressed concern that if biological explanations imply that people with psychiatric disorders do not have control over their symptoms, such people could be perceived as being unpredictable and dangerous (Read, Haslam, Sayce, & Davies, 2006). Indeed, many empirical studies have confirmed this fear, with perceived dangerousness and unpredictability being associated with biological explanations of psychiatric disorders (Bennett, Thirlaway, & Murray, 2008a; Jorm & Griffiths, 2008; Schnittker, 2008a; Schomerus et al., 2014; Walker & Read, 2002).

Blame and immutability

A long-established argument in favour of biological explanations of psychiatric disorders is that they result in reduced blame toward those who are experiencing symptoms. Much research has in fact supported this theory, with many studies linking biological explanations with reduced perceptions of personal responsibility and blame (Deacon & Baird, 2009; Gangi, Yuen, Levine, & McNally, 2016; Kvaale, Gottdiener, & Haslam, 2013b; Lebowitz, Rosenthal, & Ahn, 2016). Another area of public opinion which has important social consequences is how immutable psychiatric disorders are perceived to be. If those with psychiatric disorders are thought of as being categorically different from the rest of humankind, neuroessentialist beliefs or even mechanistic dehumanisation can be triggered (Lebowitz & Appelbaum, 2019). Studies have found that biological explanations do indeed elicit neuroessentialist biases and result in the notion that such conditions are immutable (Bennett et al., 2008a; Kvaale, Haslam, & Gottdiener, 2013; Lam et al., 2005; Magliano et al., 2016)

Social distance

Studies which have investigated how biological explanations of psychopathology impact the desire for social distance from those affected report mixed results (Lebowitz & Appelbaum, 2019). Whilst some research has shown an association between biological explanations and increased desire for social distance (Dietrich et al., 2004; Dietrich, Matschinger, & Angermeyer, 2006; Speerforck, Schomerus, Pruess, & Angermeyer, 2014), other studies have found such explanations have little impact on social distance or even reduce it (Bennett et al., 2008a; Jorm & Griffiths, 2008; Lebowitz & Ahn, 2012; Lebowitz et al., 2016). Meta analyses have found that in experimental studies, biological explanations do not have any significant impact on preference for social distance whilst in correlational studies, a small but inconsistent relationship exists (Kvaale, Gottdiener, et al., 2013b; Kvaale, Haslam, et al., 2013).

Effects among clinicians

Stigma

Although the link between causal beliefs and mental illness stigma has been thoroughly investigated on members of the general public, there has been less focus on clinicians and patients themselves (Larkings & Brown, 2018). One study conducted on mental health clinicians did not find any association between genetic explanations of psychosis and preference for social distance from people with psychosis (Grausgruber, Meise, Katschnig, Schöny, & Fleischhacker, 2007). In another study with mental health professionals, a biogenetic rather than psychosocial explanation of psychosis led to the patient being perceived in less human terms, with such conceptions resulting in stronger preference for restraint methods (Pavon & Vaes, 2017).

Empathy impacting the therapeutic alliance

As discussed, any influence which biological explanations have on the therapeutic alliance will have an effect on clinical outcomes (Horvath et al., 2011). In a study with mental health clinicians, biological explanations of various psychiatric disorders consistently elicited less empathy than psychosocial explanations. Even when explanations incorporated both biological and psychosocial attributions, clinicians reported less empathy when information was predominantly biological compared to psychosocial (Lebowitz & Ahn, 2014). In a subsequent study, laypeople consistently rated clinicians who read biological conceptualisations of psychiatric disorders as less warm than those who read psychosocial explanations (Lebowitz, Ahn, & Oltman, 2015)

Treatment selection

It has been shown that clinicians are susceptible to the mind-body dualism (Miresco & Kirmayer, 2006), in which the mind and body are perceived as distinct and separate. Considering this, researchers have proposed that biological explanations might cause a clinician to recommend treatment such as medication, which has a biological target, over psychological therapy, which is perceived as targeting the mind and possibly not as effective in treating biologically described pathology (Lebowitz & Appelbaum, 2017). In support of this theory, a correlational study found that the more a disorder was considered by clinicians to have a biological aetiology, the more effective medication was expected to be and the less effective psychological therapy was expected to be in treating it (Ahn, Proctor, & Flanagan, 2009).

In a subsequent study, clinicians perceived psychological therapy to be significantly less effective than medication when patient symptoms were described in biological compared to psychosocial terms (Lebowitz & Ahn, 2014). The biological explanations also elicited higher ratings of medication effectiveness (with the exception of schizophrenia, which yielded equally

high medication effectiveness ratings regardless of explanation; Lebowitz & Ahn, 2014). In another study, clinicians who conceptualised their patients' psychosis in biological terms were more likely to recommend medication, whilst those who favoured psychosocial explanations were more likely to rate CBT as effective (Carter, Read, Pyle, Law, & Morrison, 2017). Thus, such results suggest that biological explanations have a significant impact on patient care as they directly influence the type of treatments clinicians recommend (Lebowitz & Appelbaum, 2019).

Effects among patients

Self-stigma

There has been a recent increase in attention to how patients affected by mental health difficulties perceive their problem and how biological explanations of their own difficulties is associated with self-stigmatising attitudes (Carter, Read, Pyle, & Morrison, 2017; Larkings & Brown, 2018; Lebowitz, 2014). The few studies conducted have generally shown mixed results, suggesting that the relationship between self-stigma and biological explanations may differ depending on the disorder being considered, the measures used and potentially other variables (Lebowitz & Appelbaum, 2019).

Self-efficacy

As discussed earlier, biological explanations tend to reduce perceptions of blameworthiness, possibly because the occurrence of symptoms is deemed as outside of the control of the individual. One concern which emerges from this line of reasoning is that biological explanations may lead those affected by psychiatric disorders to perceive a lack of agency or self-efficacy over their own difficulties, thereby resulting in feelings of helplessness (Kong, Dunn, & Parker, 2017). Research conducted in this area has produced mixed results, with some

studies finding biological explanations result in reduced self-efficacy (Dar-Nimrod, Zuckerman, & Duberstein, 2013; Kemp, Lickel, & Deacon, 2014) and others reporting the contrary (Lebowitz et al., 2013; Lee et al., 2016). As with self-stigma, these mixed findings could be attributed to difference in the disorder being studied, the measures used or the information that biological explanations are compared against (Lebowitz & Appelbaum, 2019).

Prognostic expectations

As discussed earlier, prognostic expectations and beliefs about the efficacy of a certain treatment have important implications for treatment outcomes. Research which has investigated the relationship between biological explanations and prognostic beliefs among patients has reported worrying results, given the rise in popularity of such explanations. Recent studies have found that patients who were given a biological explanation of their difficulty were in fact more pessimistic about their prognosis (N. Farrell, Lee, & Deacon, 2015; Kemp et al., 2014; Lebowitz et al., 2013; Lebowitz, Pyun, & Ahn, 2014; Lebowitz & Ahn, 2018). In addition, biological explanations made participants more likely to view medication as an effective treatment for their difficulties (Carter, Read, Pyle, & Morrison, 2017; Lüllmann, Berendes, Rief, & Lincoln, 2011), and this can also come at the cost of faith in psychological therapies (Iselin & Addis, 2003; Kemp et al., 2014; Lebowitz & Appelbaum, 2017).

To summarise: Given the rise in biological explanations of psychopathology, an understanding of their implications in both social and clinical contexts is extremely relevant. To understand such implications, three theoretical explanations have been referred to, including attribution theory, essentialism and mechanistic dehumanisation (Lebowitz & Appelbaum, 2019). Based on the results of several meta analyses, there is strong evidence for a 'mixed blessings' model, which incorporates both beneficial and harmful effects (Haslam & Kvaale, 2015). Amongst the general public, biological explanations of a psychiatric disorder result in reduced blame toward

those affected on the one hand, but the perception that the disorder is relatively immutable and that symptomatic individuals are unpredictable and dangerous on the other hand. The desire for social distance reports mixed results. Amongst clinicians, biological explanations elicit less empathy and lead to a preference for medication over psychological treatment (Lebowitz & Ahn, 2014). Amongst patients, biological explanations tend to reduce perceptions of blameworthiness, whilst their influence on self-stigmatising attitudes and self-efficacy is mixed. Furthermore, prognostic pessimism tends to rise when a psychiatric disorder is explained in biological terms, with medication being viewed as a more effective treatment at the cost of faith in psychological therapy (Carter, Read, Pyle, & Morrison, 2017; Iselin & Addis, 2003)

Section 4 Neuroplasticity and argument for the biopsychosocial model

Neuroplasticity

The negative social and clinical consequences of biological conceptualisations represents a serious cause for concern, given that neuroscience provides an incomplete account of the aetiology of mental health difficulties (Schultz, 2018). Some neuroscientists have argued that neuroessentialistic conceptions of the mind disregard evidence which points to the influence of the environment in mental processes (Slaby & Gallagher, 2015). They posit that in order to understand phenomenological experience, we must appreciate how neural processes and environmental structures form a reciprocal causal relationship to allow higher cognitive performance (Schultz, 2018). Indeed, neuroessentialist beliefs about the biology of psychiatric disorders are actually inconsistent with current science (Lebowitz & Ahn, 2015). In a process known as neuroplasticity, the brain has a lifelong ability to change and reorganise in response to environmental pressures and experiences, which is important in the recovery from

psychiatric disorders (Lozano, 2011). The interaction between genes and risk for psychiatric disorders is also typically moderated by environments and experiences, and at times these elements can result in epigenetic changes in gene expression, thereby altering subsequent emotional development (Lau & Eley, 2010; Zhang & Meaney, 2010). This evidence clearly challenges the notion that psychiatric symptoms are relatively immutable, which are often evoked by biological conceptualisations of psychopathology, a misconception which should be addressed.

Functional magnetic resonance imaging

The introduction of functional magnetic resonance imaging (fMRI) and other brain imaging tools has allowed scientists to address important psychological questions by determining the neurobiological circuitry of emotional, social, behavioural and developmental processes (Schwartz, Lilienfeld, Meca, & Sauvigne, 2016). Over the past decade, an increased understanding of the principles of brain plasticity has resulted in cognitive training interventions which target cortical malleability (M. Fisher, Loewy, Hardy, Schlosser, & Vinogradov, 2013). These interventions have led to behavioural improvements in neuropsychiatric disorders, with some of the most promising results emerging from the field of schizophrenia (Vinogradov, Fisher, & De Villers-Sidani, 2011). In addition to cognitive training, there is mounting evidence that psychiatric and psychological treatments, including non-biomedical psychological therapies, can create neurobiological changes in patients (Linden, 2006).

A recent systematic review of the effects of psychological therapy on brain function found that therapy either leads to a correction of abnormal patterns of brain activity or the incorporation of additional brain areas which did not show altered activation prior to treatment, or a combination of both (Barsaglini, Sartori, Benetti, Pettersson-Yeo, & Mechelli, 2014).

Subsequent research has shown that talking therapies such as CBT for psychosis (CBTp) have been shown to bring about changes in neural activation and connectivity, promoting the processing of threatening stimuli in a less stressful way (Kumari et al., 2011; Mason, Peters, Williams, & Kumari, 2017).

One such study found that reorganisation at the neural level following psychological therapy could actually predict the recovery path of participants with psychosis up to 8 years after therapy ceased (Mason et al., 2017). Such findings imply that psychological therapy produces sustained change, even within a strictly biological approach to mental illness, and are a relevant illustration of the interaction between neuroplasticity and experience. Another recent systematic review investigated the impact of psychological therapies on the functional integration between brain regions. Of the 15 studies which met their inclusion criteria, the vast majority involved CBT. A key finding was that functional connectivity both predicts and is altered by CBT, with connections to the prefrontal cortex appearing to be especially important in symptom reduction (Mason, Peters, & Kumari, 2016). In principle, disseminating the results of the above findings could promote not only the viability of psychological therapy as a treatment option but also a more integrated causal model of mental illness, moving away from a mind-body dualism which currently exists (Kendler, 2005).

Neglected environmental factors

Without doubt, neuroscience has added a considerable amount of knowledge and data to the field of psychology. It has helped us to better understand how the brain develops and has helped identify the link between brain function and psychological processes. However, an eliminative reductionist perspective which attempts to explain behaviours, feelings and thoughts at the cellular and molecular level does not adequately explain the origins of psychiatric disorder and is at odds with much neuroscientific research that emphasises the importance of environmental

influences (Schwartz, Lilienfeld, Meca, & Sauvigne, 2016). Furthermore, the increased focus on neuroscience has overshadowed the socio-cultural-economic aspects of psychiatric disorder despite widespread evidence of their relevance.

Research has found that environmental factors such as wealth distribution, gender inequality and societal stigma can act as obstacles to physical and mental healthcare, resulting in inequity of access (Seedat, 2014). Socioeconomic factors have been found to be contributing factors to child and adult psychopathology, with one review finding that children from disadvantaged socioeconomic families were twice as likely to develop mental health issues as peers from more advantaged backgrounds (Reiss, 2013; Shonkoff, J. and Garner, 2012). Another related study has found that children from low socioeconomic households are at higher risk of developing psychosis or schizophrenia (Wicks, Hjern, Gunnell, Lewis, & Dalman, 2005). Regarding gender, issues such as restricted control over reproductive health, economic deprivation and cultural values were found to be mediating factors in the occurrence of postnatal depression in Goa, India (Patel, Rodrigues, & DeSouza, 2002). As previously discussed, there has been a disproportionate emphasis on the biological factors which predict schizophrenia, despite substantial evidence for the influence of environmental factors on its onset and maintenance (Calton et al., 2009; Jones & Fernyhough, 2007).

Given the empirical evidence for the importance of environmental factors in the explanation of mental illness, caution should be exercised when using the individualised discourse of biological explanatory models. In the first instance, there are many negative unintended consequences of using such a model, which have been examined in the previous section. Moreover, the benefits of biomedical medicine and genomic testing remain largely unavailable to disempowered cohorts such as children who grow up in socioeconomically disadvantaged families or immigrants with restricted access to public health services (Kong et

al., 2017). The discourse of individualised biomedical medicine is dangerous in that it might limit the focus of public health interventions to those who are privileged enough to access genomic testing, or to countries which have the necessary technology to conduct relevant analyses. By focusing energy and resources on biological factors, attention is diverted away from wider systemic issues such as socioeconomic inequality, gender, immigration and intergroup conflict (Kong et al., 2017).

Biopsychosocial model

In 1977, Engel (1977) criticised a dominant biomedical paradigm which was conspicuously comparable to the one we are living in today. He proposed a new medical model founded on a biopsychosocial approach, acknowledging that a range of perspectives are necessary in informing our understanding of psychiatric disorders. Such disorders can be studied at different levels of analysis (cognitive neuroscience, personality, molecular genetics, environment, neurobiology) but no level is more significant or superior to any other (Deacon, 2013). Rather, each level of analysis can be used for different objectives. For example, public health officials who are attempting to reduce alcohol dependence might target tractable environmental factors such as taxation or social norms while pharmaceutical researchers would focus their efforts on drug treatments that could target molecular genetic variants (Kendler, 2012).

Psychiatric disorders, which have been referred to as "high-order disturbances in multi-level mechanisms" (Kendler, 2012, pg 17), are too complex to be fully explained by any one area such as neurotransmitter dysregulation, childhood trauma, self-esteem or irrational thinking. The biopsychosocial model acknowledges such complexity, avoiding futile searches for simple explanations and minimising competition between professions over what the more fundamental level of analysis is (Deacon, 2013). Instead, the biopsychosocial approach promotes collaboration and dialogue across theoretically different healthcare professions to

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investigate and clarify how processes at one level of analysis can impact those at another (Caspi et al., 2003). In doing so, the substantial contributions of neuroscience can complement and enhance the findings in other areas of psychology (Schwartz, Lilienfeld, Meca, & Sauvigne, 2016).

To summarise: Biological conceptualisations of psychopathology often evoke neuroessentialist beliefs about psychiatric symptoms as being relatively immutable. Such beliefs are actually inconsistent with current science, which has shown that the brain can adapt in response to environmental experience in a process known as neuroplasticity (Lebowitz & Ahn, 2015). The recent introduction of fMRI technology has allowed neuroscientists to identify the neurobiological correlates of emotional, social and behavioural processes (Schwartz, Lilienfeld, Meca, & Sauvigne, 2016). Furthermore, research has shown that psychological treatments can cause neurobiological changes in patients, with CBTp bringing about changes in neural activation and connectivity which predict the recovery path of patients after therapy ceases (Mason et al., 2017). Although neuroscience has enhanced our understanding of how the brain develops, its current dominance has overshadowed the social, cultural and economic factors which contribute to mental illness. The recent focus on biological explanatory models diverts resources away from wider systemic issues and risks isolating those who do not have access to such technology (Deacon, 2013). A biopsychosocial model of psychiatric disorder has been proposed, with equal weight given to each level of analysis. Such a model allows for the integration of multiple explanatory perspectives, investigating and clarifying how processes at one level of analysis can impact those at another (Schwartz, Lilienfeld, Meca, & Sauvigne, 2016).

Section 5 Emphasising malleability and treatability

Challenging neuroessentialist beliefs

Considering the substantial negative effects that biological explanations of psychopathology can have on social and clinical attitudes, recent studies have explored how such effects can be mitigated. A large proportion of the negative effects of biological explanations result from the neuroessentialist thinking they evoke; that psychiatric disorders are relatively immutable and are caused by biological dysfunction in a deterministic manner (Lebowitz & Appelbaum, 2019). Thus, research aimed at overcoming such thinking has focused on the malleability of biological factors involved in such conditions as well as informing participants about effective treatments for psychiatric disorders. By emphasising the reciprocal relationship between biological and psychosocial factors, malleability-focused education may serve in part by challenging beliefs in mind-brain dualism (Lebowitz, 2014).

Emphasising treatability and malleability

One study found treatment information to be more effective in improving attitudes towards people with mental illness if it is paired with a biological versus psychosocial explanation of psychiatric disorder, possibly because the biological explanations evoked prognostic pessimism whilst the psychosocial explanations did not (Lebowitz & Ahn, 2012). Another recent study found that showing individuals with depression a psychoeducation video about neuroplasticity and epigenetics reduced their prognostic pessimism and increased their feelings of agency (Lebowitz et al., 2013). A subsequent study extended these findings by showing that such benefits persist 6 weeks after the intervention (Lebowitz & Ahn, 2015). Another study found a malleability-focused intervention produced more prognostic optimism and self-efficacy amongst a group of participants displaying eating disorder symptoms (N. Farrell et al., 2015).

A study with the general population found that providing treatment information for schizophrenia reduced perceptions of dangerousness, unpredictability and anxiety towards

those affected, with prognostic pessimism also reducing following CBT information (Schlier, Lange, Wiese, Wirth, & Lincoln, 2016a). A more recent study found that information about the malleability of genetic effects can counteract pessimistic beliefs evoked by genetic test results suggesting predisposition to depression (Lebowitz & Ahn, 2018). These results suggest that cultivating incremental beliefs which are based on potential for change and growth can be used as an interventive strategy in the treatment of psychological dysfunction (Howell, 2017). It highlights how clinicians should be aware of the prognostic pessimism and reduced self-efficacy which can be elicited by biological conceptualisations of their difficulties and how they should respond with an emphasis on the malleability of their condition (Lebowitz & Appelbaum, 2019).

Gaps in the knowledge

fMRI imaging

The studies described above used a variety of interventions to promote the malleability of psychiatric disorders, ranging from written descriptions of effective treatments to audio-visual presentations emphasising the malleability of neurochemistry. However, none of the studies used the results of recent neuroimaging studies which illustrate the malleability of neural connections described in section 4. To the best of our knowledge, this is not an intervention which has ever been used to counteract essentialist thinking which might be evoked by biological explanations of psychopathology. It is plausible that presenting the evidence of neuroplasticity following CBT could not only challenge essentialist assumptions, but also add credibility to psychological therapy as an intervention which addresses the biology of mental illness.

Psychosis

While a number of the above studies have examined the impact of an education intervention after participants are given biological explanations of depression, fewer studies have done so in the context of psychosis. This is particularly important because psychosis is a condition which has traditionally been conceptualised in biological terms (Calton et al., 2009) and just one in ten suitable patients actually gets access to psychological therapy (Schizophrenia Commission, 2012). Indeed, there is research to suggest that when a disorder is considered to have a biological aetiology, medication is expected to be more effective than psychological therapy in treating it (Ahn et al., 2009). As discussed, a previous study involving clinicians found that biological explanations elicited significantly higher medication effectiveness ratings than psychosocial explanations for a range of psychiatric disorders except for schizophrenia, which recorded equally high medication effectiveness ratings regardless of explanation type (Lebowitz & Ahn, 2014). One reason for this finding could be that as schizophrenia has traditionally been conceptualised in biological terms, clinicians were more heavily endorsing an intervention which they perceive to be targeting the biological phenomena underlying the symptoms (Lebowitz & Appelbaum, 2019). It is possible that emphasising how psychological therapy targets the biology of a condition which is conceptualised in biological terms might challenge treatment preferences that both the general public and clinicians may hold.

Pre-existing illness perception beliefs

As previously discussed, biological explanations can evoke neuroessentialist thinking patterns which view psychiatric disorders as emerging from biological dysfunction in a deterministic manner. It is therefore important to consider one's pre-existing illness perception beliefs when measuring the impact of biological explanations of psychopathology on one's attitudes. None of the malleability-focused studies measured prior beliefs about the malleability of mental illness, which could be modulating the effect of an education intervention. The Illness

Perception Questionnaire (IPQ) is a self-report assessment which was originally developed to measure patients' beliefs about their physical health conditions so that their responses to illness and adherence to clinical interventions could be better understood (Weinman, Petrie, Moss-Morris, & Horne, 1996). It draws from the Self-Regulation Model, which posits that an individual's beliefs about their condition can be represented across five dimensions: causal, identity, acute-chronic, cure-control and consequences (Leventhal & Diefenbach, 1991). The IPQ has sound psychometric properties and there is much evidence which supports the influence of illness perceptions upon recovery (Petrie, Jago, & Devcich, 2007; Stewart & Yuen, 2011). Since its inception, the IPQ has been adapted for a range of conditions, including those involving mental health. One such modification was designed to predict response to cognitive therapy for psychosis (Marcus et al., 2014b). Two reliable subscales were derived from the modified IPQ with the first subscale representing an expanded cure-control construct and the second assessing perceptions of problem duration (timeline). The modified IPQ has been adapted for the current study to measure one's pre-existing assumptions about the determinability of mental illness in addition to perceived effectiveness of psychological therapy in treating it.

Treatment appraisals

The malleability-focused studies measured the effectiveness of education interventions on changing attitude constructs such as blame, prognostic pessimism, desired social distance and self-efficacy regarding mental health difficulties. However, treatment appraisals were not included as variables of interest. The effectiveness of an education intervention in influencing one's treatment recommendation is of particular clinical significance because research has shown that biological explanations can not only influence the type of treatments clinicians recommend (Carter, Read, Pyle, Law, et al., 2017; Lebowitz & Ahn, 2014), but they can also

lead patients to view medication as a more effective treatment for their difficulties and reduce perceived effectiveness of psychological therapy (Iselin & Addis, 2003; Lüllmann et al., 2011)

Clinician participants

As previously discussed in section 3, there has been much research conducted on the stigmatising impact of biological explanations on the general public. However, research investigating their impact on clinician or patient attitudes has been relatively scant (Larkings & Brown, 2018). Furthermore, whilst the malleability-focused studies measured the effectiveness of an education intervention amongst the general population and patients themselves, none of the studies investigated the impact on clinician attitudes. Clinician attitudes are of particular importance because it has been found that clinicians can employ a mind-brain dichotomy when reasoning about clinical cases (Miresco & Kirmayer, 2006). It is proposed that presenting clinicians with recent neuroimaging evidence that CBTp leads to functional connectivity changes will not only add credibility to psychological therapy as a treatment option, but also challenge beliefs in a mind-brain dualism that the clinician may hold.

To summarise: Much of the negative impact that biological explanations can have on social and clinical attitudes stems from the essentialist thinking that they evoke. Research aimed at challenging such thinking has focused on providing information about the malleability of biological factors involved, in addition to effective treatments available (Lebowitz & Appelbaum, 2019). Such malleability-focused interventions have led to an improvement in attitudes toward those with mental illness, reduced prognostic pessimism and increased self-efficacy amongst those affected. Whilst these studies have shown encouraging results, there are a number of areas where the research could be developed. Firstly, the effectiveness of a malleability-focused intervention on clinician attitudes, with an emphasis on treatment recommendation, is an area not yet explored, yet of great importance, because clinicians are

not immune to the mind-brain dualism which exists amongst the general public (Miresco & Kirmayer, 2006). Secondly, many of the malleability-focused studies have used biological explanations of depression or eating disorders, but psychosis is an area which has not yet been explored. Third, presenting recent neuroimaging evidence which illustrates neuroplasticity following CBTp is an innovative way to implement an education intervention which introduces both the malleability of biological factors involved in psychosis and an effective treatment which is available for such a condition (Mason et al., 2017). Finally, relevant pre-existing beliefs which might modulate the effects of a malleability-focused intervention have not previously been investigated but will be included in the present study.

Section 6 Conclusion

There has been an increased emphasis on neuroscientific research which examines psychopathology at the neurobiological and genetic level (Haslam & Kvaale, 2015). This increased focus has aided our understanding of psychiatric disorders but has also been criticised as being reductionist, focusing on biological factors at the expense of psychosocial factors involved (e.g. Deacon, 2013). Furthermore, research has shown that biological explanations of psychopathology can have a number of unintended negative implications for attitudes amongst the general public, clinicians, and patients themselves (Lebowitz & Appelbaum, 2019). Whilst much is known about the importance of emphasising the malleability of mental health difficulties, typically this is targeting attitude constructs such as blame, desired social distance and prognostic pessimism. What is less known is whether evidence of neuroplasticity can be an effective way to address treatment assumptions that result from biological explanations. Recent advancements in neuroimaging technology have demonstrated that reorganisation can and does occur at the neural level following a course of psychological therapy, and indeed these neural changes predict the recovery course of people with psychosis over several years post-

therapy (Mason et al., 2017). The present study aims to examine whether presenting such evidence could impact public and clinician perceptions about the effectiveness of psychological therapy as a treatment option for psychosis, a condition which has traditionally been conceptualised in biological terms (Calton et al., 2009).

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Chapter 2: Empirical Paper

How evidence of neurobiological changes following psychological therapy impacts treatment recommendation

Abstract

Aim

Biological explanations of mental health difficulties have remained dominant in recent decades, particularly in the case of psychosis. Research has shown that these explanations can lead to reduced empathy and a bias towards offering pharmacological rather than psychological intervention. This study aimed to explore whether this bias can be reduced by presenting evidence that CBT for psychosis (CBTp) also brings about neurobiological changes (i.e. neuroplasticity). This was examined both in samples from the general population (Study 1) and mental health clinicians (Study 2).

Method

An experimental design was employed in which participants read a vignette of a person with psychosis. Participants were asked to report which treatment they would recommend (i.e. medication versus psychological therapy) and how effective they perceived each to be. These ratings were elicited before and after reading information about the effects of CBTp: either control information about its clinical effectiveness or evidence of CBTp-led neuroplasticity.

Results

In Study 1, the predominantly biological description of psychosis elicited lower therapy effectiveness and higher medication effectiveness ratings compared to the predominantly psychosocial description, replicating previous work. Therapy effectiveness ratings were higher and medication effectiveness ratings were lower after either type of CBTp effectiveness evidence was presented. In Study 2, evidence of CBTp-led neuroplasticity had a greater impact than the control information on treatment recommendation ratings amongst psychologists, but not psychiatrists.

Conclusions

We replicate a previous finding that when biological factors are emphasised in patient information, it biases people to choose medication rather than psychological therapy as a treatment option. When the general population are presented with any type of evidence that CBTp is an effective treatment, they are likely to rate psychological therapy as a more effective treatment method. The impact amongst clinicians is more varied, depending on their training background.

Introduction

Biological explanations of psychopathology

Biological explanations of psychopathology have become increasingly dominant in recent decades (Haslam & Kvaale, 2015). Funding has increasingly prioritised research that focuses on the neurobiology and genetics of psychiatric disorders, with the National Institute of Mental Health (NIMH, USA) recently launching the Research Domain Criteria (RDoC) framework for identifying biomarkers of psychiatric disorders (Hyman, 2007). This increased focus on biomarkers has been accompanied by greater interest in the development of psychotropic medication, with the field of mental health moving from one traditionally influenced by Freudianism and other psychodynamic conceptualisations to one in which biological explanations play a more important role (Rosenbloom, 2002). Between 1998 and 2007, data from nationally representative surveys in the United States suggests that the prevalence of pharmacological treatment in outpatient mental health care rose, while rates of psychological therapy declined, with an increasing proportion of patients receiving psychotropic medication without any psychological therapy (Olfson & Marcus, 2010). This shift toward biological conceptualisations of psychopathology is not just restricted to the field of neuroscience and psychiatry. Over the past decade, the media has portrayed neurobiological processes as the dominant source of psychiatric disorders, leading to a shift in how the public understand mental health difficulties (Racine et al., 2010; Schomerus et al., 2012).

There have undoubtedly been advances in the assessment, diagnosis and understanding of psychiatric disorders due to the increased focus on the biological level of analysis. The introduction of functional magnetic resonance imaging (fMRI) and other brain imaging tools has allowed scientists to address important psychological questions by identifying the neurobiological circuitry associated with human experience and behaviour (Schwartz,

Lilienfeld, Meca, & Sauvigne, 2016). To illustrate the point, combined anatomical and functional approaches have shown reduced brain connectivity in psychosis (Skudlarski et al., 2010). However, classifying psychiatric disorders as brain diseases has been criticised for being reductionist, with the danger that other levels of analysis are being neglected (Bentall, 2006; Kagan, 2013). Despite this, the increased emphasis on biological research is a trend which seems set to continue, reinforcing the degree to which clinicians will use biological explanations in their practice (Lebowitz & Appelbaum, 2019).

Implications for social and clinical attitudes toward patients

When psychopathology is explained in biological terms, studies have shown that there may be several unintended ramifications for how patients are perceived (Lebowitz & Appelbaum, 2019). Research exploring effects amongst the general public has shown biological explanations to elicit neuroessentialist beliefs, resulting in the perception that psychiatric disorders are relatively immutable (Bennett et al., 2008b; Kvaale, Haslam, et al., 2013; Lam et al., 2005; Magliano et al., 2016). On the one hand, biological explanations are associated with reduced perceptions of personal responsibility and blame (Deacon & Baird, 2009; Gangi et al., 2016; Lebowitz et al., 2016), but on the other hand they are linked to perceived dangerousness and unpredictability (Angermeyer et al., 2014; Bennett et al., 2008b; Jorm & Griffiths, 2008; Schnittker, 2008b).

Concerning effects among clinicians, biological explanations can impact empathy in a number of ways. Firstly, biological explanations of various psychiatric disorders have consistently elicited less empathy than psychosocial explanations amongst clinicians (Lebowitz & Ahn, 2014). Secondly, clinicians who describe psychiatric disorders in biological terms have been rated as less warm by members of the general public than when they describe such disorders in psychosocial terms (Lebowitz et al., 2015). These effects are concerning as

they can adversely impact the therapeutic alliance and result in poorer clinical outcomes (Horvath et al., 2011). Biological explanations are also known to bias treatment decision-making; specifically, they have been shown to increase confidence in medication and reduce trust in psychological therapy as a treatment option (Ahn, Proctor, & Flanagan, 2009; Carter, Read, Pyle, Law, & Morrison, 2017). Amongst clinicians, biological explanations of psychopathology have consistently elicited lower psychological therapy effectiveness ratings and higher medication effectiveness ratings than psychosocial explanations (with the exception of schizophrenia; Lebowitz & Ahn, 2014).

Biological explanations also impact patients' perception of their own mental health difficulty. Research examining associations between biological explanations of one's own mental health difficulty and perceived self-efficacy over their own condition or self-stigmatising attitudes has reported mixed results, suggesting that such effects depend on the mental health difficulty in question or the measures which are used (Lebowitz & Appelbaum, 2019). Regarding prognostic expectations, patients who were given a biological explanation of their difficulty were actually more pessimistic about their prognosis (Farrell, Lee, & Deacon, 2015; Kemp, Lickel, & Deacon, 2014; Lebowitz, 2014; Lebowitz, Ahn, & Nolen-Hoeksema, 2013) and were more likely to view medication as an effective treatment for their difficulties (Carter et al., 2018; Lüllmann et al., 2011); often at the cost of faith in psychological therapies (Iselin & Addis, 2003; Kemp et al., 2014; Lebowitz & Appelbaum, 2017).

Neuroplasticity of psychiatric disorders

The negative social and clinical implications of biological conceptualisations represents a serious cause for concern, given that mental health neuroscience still has a long way to go in explaining psychiatric disorders (Schultz, 2018). Furthermore, the belief that psychiatric disorders are immutable, which is often evoked by biological explanations, is actually

inconsistent with current science (Lebowitz & Ahn, 2015). In a process known as neuroplasticity, the brain has been shown to adapt and reorganise in response to environments and experiences well into adulthood and is important for the recovery from psychiatric disorders (Lozano, 2011). In a widely cited example of neuroplasticity, the hippocampal volume of London taxi drivers is significantly larger than that of controls (Maguire et al., 2000). Moreover, talking therapies such as CBT for psychosis (CBTp) have been shown to bring about changes in neural activation and connectivity between brain regions involved in top-down regulation of emotion and threat processing (Kumari et al., 2011; L. Mason et al., 2017). One such study found that reorganisation at the neural level following CBTp could actually predict the recovery path of participants with psychosis across multiple years (L. Mason et al., 2017). Such findings imply that psychological therapy produces sustained change, even within a biological framework, and are a relevant illustration of the interaction between neuroplasticity and learning. It is plausible that presenting such evidence might challenge the notion that biologically explained psychiatric disorders are somehow immutable and add credibility to psychological therapy as a treatment option.

Interventions that promote the malleability of psychiatric disorders

Many of the aforementioned negative effects of biological explanations derive from the neuroessentialist assumptions that they evoke, leading people to view mental illness as caused by immutable biological irregularities (Bennett et al., 2008b; Kvaale, Haslam, et al., 2013). Thus, studies aimed at challenging such thinking have focused on teaching people about effective treatments for psychiatric disorders as well as the malleability of biological mechanisms involved (Lebowitz & Appelbaum, 2019). Studies have shown that such malleability-focused interventions can not only decrease prognostic pessimism among people with symptoms of eating disorders (Farrell et al., 2015) and depression (Lebowitz et al., 2013);

& Ahn, 2018). Furthermore, providing members of the public with information about effective CBT treatment for schizophrenia has been shown to reduce anxiety towards those affected and also decrease prognostic pessimism (Schlier, Lange, Wiese, Wirth, & Lincoln, 2016b).

The current investigation

Based on the literature reviewed so far, it is proposed that conceptualising psychosis with a biological explanation will trigger essentialist ways of thinking about the disorder, resulting in diminished levels of empathy and a bias to not recommend psychological therapy as a treatment option. We focus on psychosis because this population has historically been most strongly conceptualised in biological terms (Calton et al., 2009) and evidence shows clinicians consider it to be biological (Ahn et al., 2009). It is also proposed that presenting recent empirical evidence of CBTp-led neuroplasticity could modify this bias by increasing the perceived credibility of psychological therapy for disorders that are strongly biologically conceptualised. It is possible that such evidence would have a greater impact on perceived treatment effectiveness when psychosis is explained in biological, rather than psychosocial terms, as it would challenge the treatment bias that can result from a biological explanation. Specifically, we wish to investigate whether evidence of CBTp-led neuroplasticity leads to a greater change in treatment recommendation and perceived effectiveness of psychological therapy and medication compared to generic CBTp effectiveness evidence.

We also predict that the above effects will be modulated by pre-existing beliefs about the malleability of mental illness, political orientation and dogmatic beliefs. Regarding pre-existing beliefs about mental health, previous research has shown that the credibility of psychological therapy was predicted by a patient's beliefs about their difficulties; specifically that the more controllability a patient perceives over their own illness, the more they will

engage in therapy (Freeman et al., 2013). Concerning political attitudes, previous research has shown greater agreement with scientific facts amongst more liberal individuals (Medlin, Sacco, & Brown, 2019). Other research has shown a general cognitive bias amongst those espousing radical beliefs, (Brandt, Evans, & Crawford, 2015; Greenberg & Jonas, 2003; Ortoleva & Snowberg, 2015; Toner, Leary, Asher, & Jongman-Sereno, 2013) with individuals reporting higher levels of dogmatism being more resistant to revising their decisions when presented with post-decision evidence (Rollwage, Dolan, & Fleming, 2018).

The current study tested the following hypotheses:

H1: Participants receiving a description of a patient with psychosis in which predominantly biological factors are emphasised will 1) endorse medication over psychological therapy (H1a), 2) rate psychological therapy as a less effective treatment (H1b), 3) feel less empathy for the patient described (H1c). This would replicate previous findings for different disorders by Lebowitz & Ahn (2014).

H2: Presenting evidence of CBTp-led neuroplasticity (relative to generic information about clinical effectiveness) will lead participants to 1) recommend psychological therapy over medication (H2a), 2) rate psychological therapy as a more effective treatment (H2b).

H3: Change in treatment effectiveness ratings will be modulated by pre-existing beliefs and attitudes. Specifically, participants who believe more strongly in the malleability of mental illness will rate psychological therapy as a more effective treatment (H3a), and those espousing stronger political attitudes will be less likely to change their therapy effectiveness ratings (H3b).

Method

Pilot study

The experiment was first piloted to test the methods and procedures and examine the feasibility of an approach that was to be used on a larger scale. The pilot was conducted online over two stages, with 3 participants competing the first phase and 55 participants completing the second phase following initial amendments to the design. All participants were recruited via SONA and Prolific participant recruitment platforms. Qualitative feedback was also elicited and used to inform alterations to the design to be used in the main experiments (see Results).

Participants

Two separate main experiments were conducted; the first recruited a self-selecting sample of the general public (n = 270) and the second recruited a self-selecting sample of mental health clinicians (n = 83). In Study 1, a sample of the general public completed an online experimental questionnaire. In Study 2, a sample of mental health clinicians was administered the same online experimental questionnaire. Study 1 recruited a sample of adults over the age of 18 via the online platforms (SONA and Prolific). Participants provided basic demographic data (see Table 1). Study 2 recruited a sample of trainee and qualified mental health professionals working in NHS trusts and other mental health organisations. The sample consisted of mental health professionals, including both medically trained mental health clinicians (i.e. psychiatrists, mental health nurses) and those with less biomedical training (e.g. psychologists and other health professionals; see Table 2). Information about the study was provided on NHS trust newsletters, social media groups and Twitter. If participants expressed an interest in taking part, they accessed a link to an online survey where they could read further information and take part in the research.

Ethical approval and informed consent

Ethical approval for the study was granted by the UCL Research Ethics Committee (Project ID 15721/001; Appendix A). Prior to completing the online experiment, participants were informed of the study objectives, their right to withdraw from the study and potential harms and benefits of taking part in a project information sheet (Appendix B) and provided informed consent online (Appendix C). Participants were given researcher contact details if they had any questions or concerns about any aspect of the study.

Design

Both studies employed an experimental design, with two independent variables: the first independent variable, type of vignette explanation, was a between-groups variable with two levels: a vignette of an individual with psychosis in which the information was given a predominantly biological (Appendix E) or psychosocial explanation (Appendix F). The vignettes were based on those from a previous study (Lebowitz & Ahn, 2014). We selected a subset of vignettes from this study; vignettes that were predominantly biological or psychosocial rather than purely biological or psychosocial because we sought to be as conservative as possible and closely mirror real world scenarios in which both kinds of information are typically present (Lebowitz & Ahn, 2014).

The second independent variable, CBTp treatment information, was a within-groups variable: fMRI information about CBTp-led neuroplasticity and generic information about CBTp being effective in reducing psychotic symptoms. The amount of treatment information (four key points in each) and presence of visual stimuli (i.e. one graphical image in each; see Figure 1) was carefully balanced between each and confirmed through piloting. The order in which treatment information was presented was counterbalanced, thus allowing a between-groups comparison on the initial information presented (see analyses). The dependent variables

were type of treatment and strength of recommendation, perceived effectiveness of psychological therapy, perceived effectiveness of medication (reported as supplementary material), and participant empathy (see Measures). The study also investigated possible modulating factors which helped to explain the degree of change in appraisals that occurred. These included pre-existing beliefs about mental illness, dogmatic beliefs and political orientation (see Measures).

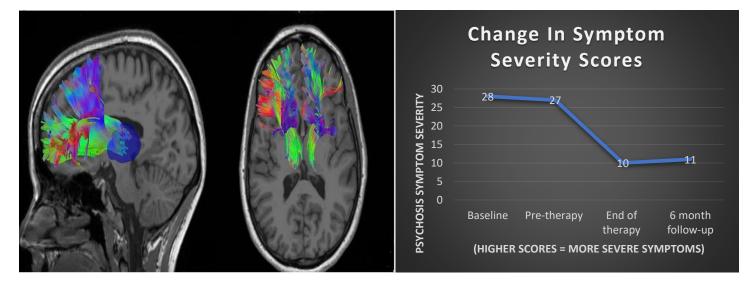


Figure 1. Treatment Information which was Counterbalanced and Presented to Participants.

A) CBTp-led neuroplasticity information: participants were presented with the fMRI scan and the following four points:

- Several recent studies have shown that cognitive behaviour therapy (CBT), a specific kind of
 psychological therapy, can bring about lasting changes in the brains of patients with
 psychosis.
- In one study, the brains of patients with psychosis were scanned before and after a course of CBT. The scans showed strengthened connections between several brain regions after CBT.
- Most important for improvement were connections between the amygdala (sometimes called the brain's "threat centre") and the frontal lobes (which are involved in thinking and reasoning). These brain changes were not seen in a control group of patients who did not receive CBT.
- In another study, the strengthening in these brain connections was key for sustained improvement in symptoms and recovery several years after patients completed therapy.

B) Generic effectiveness information: participants were presented with the pre/post symptom line graph from a clinical trial and the following four points:

- A number of research studies have shown cognitive behaviour therapy (CBT) is a helpful psychological therapy for psychosis.
- This means that many patients benefit from improvements in functioning and reduced distress from their psychotic symptoms.
- In addition to medication, CBT is one of several other treatments recommended by the National Institute for Health and Care Excellence (NICE, 2013).
- Some studies also show that CBT can continue to benefit patient's long-term recovery several years after therapy has been completed.

Measures

Perceived effectiveness of psychological therapy and medication

Participants gave slider ratings for how much they believed the patient's symptoms could improve with psychological therapy or medication on a previously validated 7-point scale, ranging from 0 (not effective at all) to 7 (completely; Lebowitz & Ahn, 2014) separately for each treatment type (Appendix J).

Treatment recommendation

A similar slider scale was used for treatment recommendation. Participants rated how strongly they would recommend medication or psychological therapy using a single linear construct ranging from -10 (strongly recommend medication) to 10 (strongly recommend psychological therapy). To control for any potentially leading effects of positive and values, the number values were not visible to participants as they completed these slider ratings (Appendix J).

Illness perception questionnaire

Pre-existing beliefs about the malleability of mental illness were measured using a 14-item previously validated modified version of the Illness Perception Scale for Psychosis, divided into three subscales (IPQ; Marcus et al, 2014). The 'Cure/Control' subscale incorporates items measuring hopefulness, self-efficacy and therapy fit, with the 'Timeline' and 'Causes' subscales assessing perceived duration and causes of the mental health problem respectively.

This scale has been shown to have acceptable internal consistency and good test-retest reliability (Appendix K).

Political attitudes

Dogmatic beliefs were measured by administering a 20-item previously validated questionnaire with good internal consistency and reliability (Altemeyer, 2001; Appendix M). Political orientation was measured by administering a previously validated questionnaire measuring how conservative one's world views are (Appendix L). Overall political attitude was measured on a sliding scale from 0 (liberal) to 100 (conservative). Number values were not visible to participants as they completed the measure. This simple question has a loading of .95 on the political orientation factor identified in a previous study on radical beliefs (Rollwage et al., 2018).

Empathy

Following Lebowitz & Ahn (2014), participant empathy was measured using an abbreviated version of a well-validated method that has been extensively used in empathy research (Appendix N). After reading the vignette, participants were presented with the word "compassionate" and rated how much this adjective describes their feelings toward the patient described on a seven-point scale, from 1= "not at all" to 7= "very much" (Lebowitz & Ahn, 2014). "Compassionate" was used because it had an average loading of .85 on the empathy factor from previous studies (Batson, 2011; Batson et al, 1997). Participants were also asked to rate how much empathy they feel for the patient on a ten-point scale, from 0= "no empathy at all" to 10= "the most empathy I could feel". This empathy rating was found to be have a strong positive association with the "compassionate" rating. [$r_s(268) = .50, p < .001$].

Procedure

Study 1

Participants who expressed an interest by clicking a link were then provided with basic information about the study explaining what they were required to do and asked for their consent to take part. Those who consented were given further information about the research, after which they were prompted to fill out demographic information and measures which captured their political orientation, dogmatic and mental illness perception beliefs.

Once the initial measures were completed, they were presented with a vignette describing a patient with a diagnosis of psychosis (Appendix D), paired with either a predominantly biological or predominantly psychosocial explanation. After reading the vignette, they were prompted to fill out more measures which captured their response to each vignette in terms of empathy, treatment recommendation, perceived effectiveness of psychological therapy and medication.

Participants were then presented with information about treatment for psychosis: either CBTp-led neuroplasticity associated with symptom improvement or a pre/post symptom line graph from a trial which shows that CBTp is effective in reducing symptoms of distress. The generic CBTp information was presented to control for demand characteristics; the expectation that participants should increase therapy effectiveness ratings after being presented with CBTp treatment information. After being presented with CBTp treatment information, participants completed the measures which captured treatment recommendation and perceived effectiveness of psychological therapy and medication for a second time. Participants were then presented with the other type of treatment information (CBTp-led neuroplasticity or generic CBTp information), after which they completed the measures which captured treatment recommendation and perceived effectiveness of psychological therapy and medication for a third time.

Participants were then debriefed, thanked, and automatically remunerated if using the online platform (Appendix O).

Study 2

For the study involving clinicians, we used a similar study design as in Study 1 but only used the vignette with the biological explanation in order to have sufficient power to detect the hypothesised effects. To reduce the number of ratings participants had to perform, participants read the vignette presented at the same time as the first treatment information. Participants gave their initial ratings and were then presented with the other type of treatment information, thereby only completing two sets of ratings. The between-groups comparison of interest was comparing post-manipulation treatment ratings across the groups that received CBTp-led neuroplasticity relative to generic treatment information at time point 1 (T1). The withingroups comparison of interest was whether the change in rating at T2 was larger when the CBTp-led neuroplasticity information was presented second compared to when it was presented first.

Statistical plan

Missing data

The online experiment was designed to prevent participants from skipping questionnaire items in order to maximise item responsiveness and reduce the occurrence of missing data. Therefore, there was no missing data. A total of 270 participants completed Study 1. Only 23 exited the study before completion (9% attrition rate). A total of 83 participants completed Study 2, with 37 exiting before completion (31% attrition rate). A listwise deletion procedure (complete case analysis) was used whereby only completed cases were included in the analysis (Study 1: n = 270; Study 2: n = 83) in order to minimise possible errors induced by missingness and to

maximise comparability across analyses (Field, 2013). The exception was thirty participants who completed Study 2 but did not complete the final dogmatic beliefs measure and three participants who did not complete the IPQ measure. A pairwise deletion procedure was used in these cases in order to retain valuable data.

Distribution of data

Assumptions of normality were tested for the main study variables. Observation of the frequency distribution histogram revealed that several variables appeared to be normally distributed including illness perception (IPQ), dogmatic beliefs, empathy ratings and medication effectiveness ratings. Across the whole sample, the Kolmogorov-Smirnov (KS) tests for normality (Field, 2013) indicated that all study variables were non-normal. However, research suggests that KS tests are not appropriate for larger samples ($n \ge 25$; Field, 2013). Non-parametric tests were used for cases where parametric assumptions were not met.

Data analyses

To test H1 (effect of biological and psychosocial explanations on treatment ratings) and H2 (the impact of treatment information on treatment ratings) we analysed Study 1 data using a MANOVA with factors vignette type: (biological vs psychosocial), treatment information (CBT-led neuroplasticity vs generic), and rating time point (prior to treatment information, after initial treatment information, after second set of information). The dependent variables were 1) treatment recommendation, 2) perceived therapy effectiveness and 3) perceived medication effectiveness ratings (the latter are reported as supplementary material). Follow-up Mann Whitney *U*-tests were used to test for mean differences between groups. To test the effect of biological versus psychosocial explanations on empathy ratings we ran an independent *t*-test

to investigate whether there was any significant difference in explanation type on DV empathy ratings.

We analysed Study 2 data using a similar MANOVA with factors treatment information (fMRI vs generic) and rating time point (after initial treatment information, after second set of information) and the same DVs.

To test H3a and H3b (modulating influence of illness perception beliefs and political attitudes) we ran multivariate analyses of covariance (MANCOVAs). The personality and attitude variables were tested as covariates organised into two separate groups: 1) Illness Perception (IPQ scores) 2) Political Attitudes (including political orientation and tendency towards dogmatic beliefs). As H3b involves two political attitude measures, we applied Bonferroni correction for the two tests being run.

All data were analysed with IBM SPSS Statistics for Windows, version 25 (IBM Corp., Armonk, N.Y., USA).

Results

Pilot study

Qualitative feedback was elicited from a total of 58 participants and subsequent amendments to the study design were made. On the treatment recommendation slider scale, participants noted that medication scores were negative whilst psychological treatment scores were positive. The numerical values on the slider scale were subsequently removed from sight to control for any potentially leading effects of positive and negative values. A few participants reported that they struggled to keep patient vignette and treatment information in working memory before reporting their ratings. To address this, study directions and subsequent questionnaires were condensed into fewer pages so that participants were required to keep less information in working memory. Participants also expressed concern that there was no option for choosing a combination of medication and psychological treatment as a treatment option on the slider scale. We included this single linear construct because we wanted to capture each participant's treatment preference and safeguard against those who might rate both therapy and mediation highly on the subsequent effectiveness scales.

Participant Demographics

Study 1 recruited a self-selecting sample of the general public (see Table 1) whilst Study 2 recruited a self-selecting sample of mental health clinicians (see Table 2). The demographic information which participants provided is reported below.

Table 1. General Population Demographics

itudy 1 (n = 270)	
Age group	18-24 years = 185
	25-34 = 50
	35-44 = 25
	45-54 = 8
	55-64 = 2
Gender	Male = 69
	Female = 198
	Rather not say = 2
	Other = 1
Level of education	Secondary school = 49
	Diploma = 52
	Degree = 112
	Postgrad = 56
Experience with mental health difficulties	None = 21
	A person close to me experiences/experienced = 6
	I myself experience/experienced = 103
	I have an informal knowledge = 38
	I have studied an academic course = 33
	I am a professional with clinical training = 2
Have they taken medication to help with a mental health difficulty?	Yes = 47
	No = 159
	Not asked = 64

0 - 3 = 117

Familiarity with psychiatric medication

(0-10); higher scores = greater familiarity) 4-6=53

7 - 10 = 36

Not asked = 64

Have they received CBT to help with a

mental health difficulty?

Yes = 46

No = 160

Not asked = 64

Familiarity with CBT 0-3=95

(0-10); higher scores = greater familiarity) 4-6=43

7 - 10 = 68

Not asked = 64

Table 2. Clinician Demographics

Age group	18-24 years = 2
	25-34 = 50
	35-44 = 22
	45-54 = 6
	55-64 = 3
Gender	Male = 29
	Female = 53
	Rather not say = 1
Profession	Psychology = 34
	Nursing mental health = 3
	Nursing medical = 1
	Psychiatry = 41
	Other health professional = 2
	Other profession = 2
Familiarity with psychosis	0 – 3 = 3
(0-10; higher scores = greater familiarity)	4 – 6 = 9
	7 – 10 = 66
Familiarity with psychiatric medication	0 – 3 = 9
(0-10; higher scores = greater familiarity)	4 – 6 = 11
	7 – 10 = 58

Study 1: general population

1. Treatment recommendation:

There was a main effect of time [F (1.42, 378.28) = 186.50, p < .001], explanation type [F (1, 266) = 28.61, p < .001], an interaction between time and explanation type [F (1.42, 378.28) = 22.57, p < .001] but no main effect of treatment information type [F (1, 266) = .004, p = .95] nor interaction between explanation type and treatment information type [F (1, 266) = .10, p = .75; see Supplementary Table 1]. Regarding the main effect of time, post hoc Wilcoxon tests confirmed that participants significantly increased their recommendation for therapy from T1 to T2 [Z= -11.02, p < .001], and again from T2 to T3 [Z = -5.73, p < .001; see Figure 2]. Concerning the time x explanation type interaction, post hoc U-tests confirmed that compared to biological explanations, psychosocial explanations made participants significantly more likely to choose therapy over medication at T1 (U = 4890, p < .001) and led to a stronger preference for therapy at T2 (U = 7084, p = .01) and T3 (U = 7190, p = .01; see Figure 3).

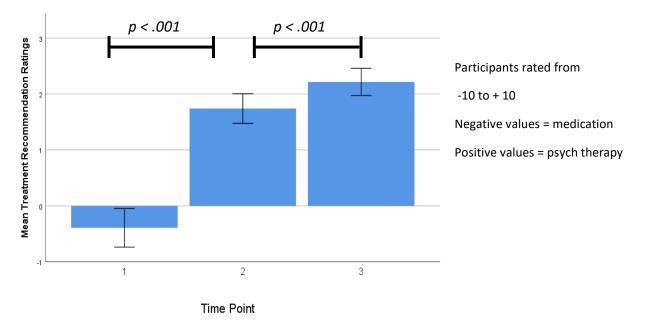


Figure 2. *Treatment Recommendation Ratings over the Three Time Points*. Participants were more likely to recommend therapy over medication at T2 (T1 vs T2) and showed a stronger preference for therapy at T3 (T2 vs T3).

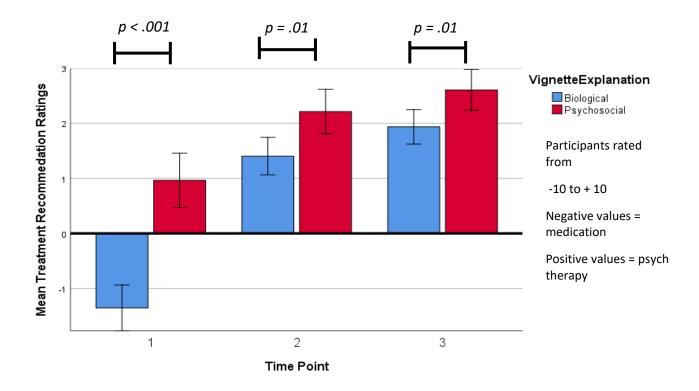


Figure 3. Treatment Recommendation Ratings by the Explanation Type Participants Received (Biological versus Psychosocial). Compared to biological explanations, psychosocial explanations elicited a preference for therapy over medication at T1 and a stronger preference for therapy at T2 and T3.

1. Effects of individual differences on treatment recommendation

There were no main effects of IPQ (p = .17), political orientation (p = .40), dogmatic beliefs (p = .33) nor interactions (p = .67) for any treatment recommendation ratings (see Supplementary Tables 2 & 3).

2. Therapy effectiveness:

As with treatment recommendation, there was a main effect of time [F(1.42, 376.46) = 146.92, p < .001], explanation type [F(1, 266) = 17.26, p < .001], an interaction between time and explanation type [F(1.42, 376.46) = 6.64, p = .001] and no effect of treatment information type

[F (1, 266) = .07, p = .79] nor interaction between explanation type and treatment information type [F (1, 266) = .06, p = .81; see Supplementary Table 1]. As with treatment recommendation, post hoc tests confirmed that participants significantly increased their therapy effectiveness rating from T1 to T2 (Z = -9.61, p < .001), and again from T2 to T3 (Z = -7.36, p < .001). With respect to the time x explanation interaction, post-hoc U-tests confirmed that participants perceived psychological therapy to be significantly less effective when symptoms were explained in biological rather than psychosocial terms at T1 (U = 5949, p < .001); T2 (U = 7811, p = .05) and T3 (U = 7256, p = .01; see Figure 4). This pattern of results mirrors that of treatment recommendation, above.

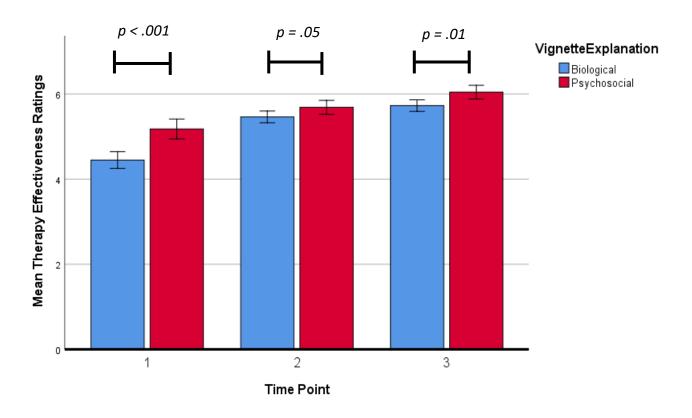


Figure 4. Therapy Effectiveness Ratings by the Explanation Type Participants Received (Biological versus Psychosocial). Ratings were higher for psychosocial condition than biological condition at T1, T2 and T3.

2. Effects of individual differences on therapy effectiveness ratings

i) Illness perception beliefs

As predicted, there was a main effect of IPQ for perceived therapy effectiveness at T1 (p = .04) and first change in therapy effectiveness rating (p = .02), but not for subsequent ratings (p = .37) and there were no interactions (p = .34). A Spearman correlation test confirmed the significant association was driven by a positive correlation [$r_s(270) = .148$, p = .02; see Figure 5], meaning those who believe more strongly in the malleability of mental illness were more likely to rate therapy as an effective treatment at baseline, independent of whether they received CBTp-led neuroplasticity or generic treatment effectiveness information.

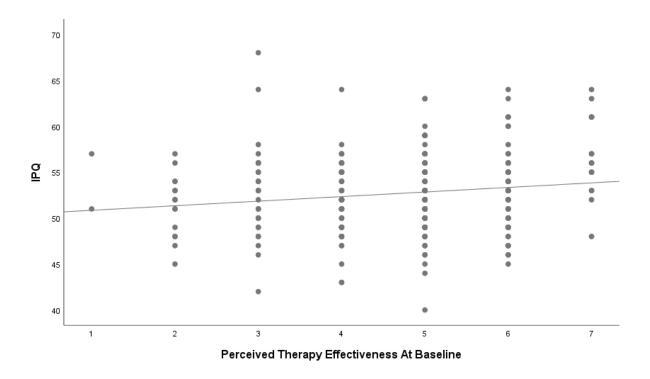


Figure 5. Association between IPQ and Therapy Effectiveness Ratings at Baseline, Prior to Receiving any Treatment Information. A Spearman test confirmed a positive correlation, with those believing more strongly in the malleability of mental illness rating therapy as a more effective treatment (p = .02).

There was an unexpected negative association between IPQ and first change in therapy effectiveness ratings [$r_s(270) = -.193$, p = .001], suggesting that those who believe more strongly in the malleability of mental illness were less likely to make increment in therapy effectiveness ratings after reading CBTp treatment effectiveness information. However, this was no longer significant when controlling for baseline therapy ratings in a partial correlation [r(270) = -.102, p = .09], suggesting the smaller increment can be attributed to high baseline ratings.

ii) Political attitudes

There was no significant main effect of political orientation for any therapy effectiveness ratings (p = .46). As expected, there was a main effect of dogmatic beliefs for first change in therapy effectiveness (p = .05), but this did not survive Bonferroni correction for the two comparisons (corrected p = .10). There were no interactions (p = .89). Follow-up tests confirmed the significant association was driven by a negative correlation [$r_s(270) = -.162$, p = .01; see Figure 6], meaning those who report higher levels of dogmatic beliefs made less increment in therapy effectiveness rating after reading CBTp treatment effectiveness information. This association remained after controlling for perceived therapy effectiveness at T1 in a partial correlation [r(270) = -.149, p = .02], suggesting that even when accounting for baseline differences, more dogmatic individuals are resistant to information aimed at increasing perceived treatment effectiveness.

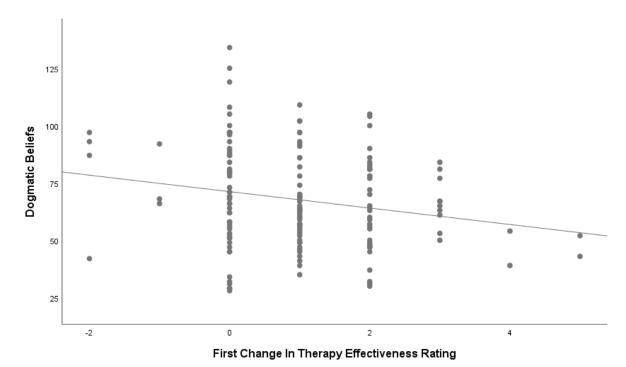


Figure 6. Association between Dogmatic Beliefs and First Change in Therapy Effectiveness Ratings. A negative correlation was found, with those espousing stronger dogmatic beliefs making less increment in therapy effectiveness ratings (p = .01).

3. Empathy ratings

As expected, the level of compassion for the patient was strongly positively correlated with empathy ratings $[r_s(268) = .50, p < .001]$. However, there was no difference in empathy ratings between participants who received the biological vs psychosocial vignette [t (268) = 1.16, p = .25]; see Figure 7; Supplementary Table 4].

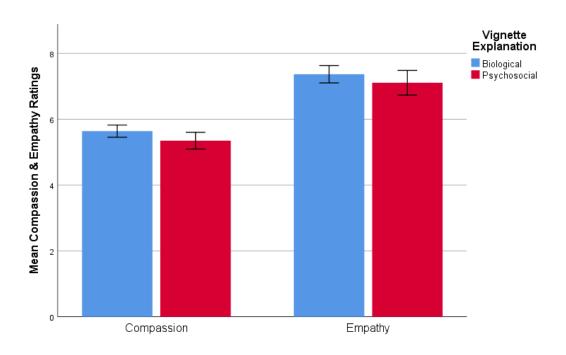


Figure 7. Mean Compassion and Empathy Ratings by the Explanation Type Participants Received (Biological versus Psychosocial). No differences were found.

Study 2: mental health clinicians

Note that in this design participants only gave two ratings: the first after reading the patient vignette and receiving the first type of treatment information (CBTp-led neuroplasticity or generic treatment information) and a second rating after receiving the other treatment information (CBTp-led neuroplasticity or generic treatment information).

1. Treatment recommendation:

There was no main effect of time [F(1, 79) = 2.43, p = .12], treatment information type [F(1, 79) = .06, p = .81] nor interaction between time and treatment information type [F(1, 79) = .12, p = .73] on treatment recommendation ratings (see Figure 8; Supplementary Table 5). A post hoc *U*-test showed that overall, those who received CBTp-led neuroplasticity compared to generic treatment information were not more likely to have a stronger preference for therapy as a treatment option at T1 (U = 803, p = .59).

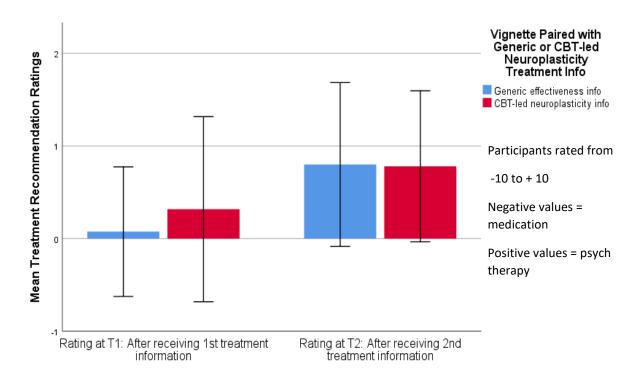


Figure 8. Clinician Treatment Recommendation Ratings Following First and Second Set of Treatment Information. No differences were found.

To examine whether these effects were modulated by professional background, we ran an exploratory ANOVA with an additional variable which distinguished psychiatrists (n = 41) from psychologists (n = 34). There was a main effect of profession [F(1, 69) = 16.58, p < .001] and a trending interaction between time, profession and information type [F(1, 69) = 3.58, p = .06]. Post hoc U-tests showed that psychologists who received CBTp-led neuroplasticity compared to generic treatment information had a significantly stronger preference for therapy as a treatment option (U = 84, p = .04; see Figure 9a), but the same effect was not found amongst psychiatrists (U = 161, p = .20; see Figure 9b).

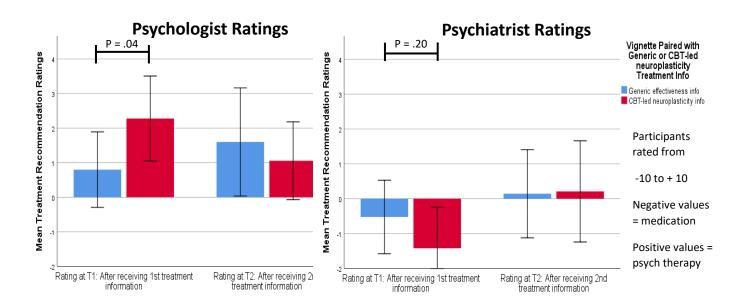


Figure 9. Clinician Treatment Recommendation Ratings Split by Profession

- A) Psychologists: CBTp-led neuroplasticity information led to a stronger preference for therapy at time point 1 (p = .04).
- B) Psychiatrists: No differences were found (p = .20).
- 1. Effects of individual differences on treatment recommendation
 - i) Illness perception beliefs

There was a main effect of IPQ for treatment recommendation rating at T1 (p = .05) but there were no interactions (p = .22). Follow-up tests confirmed the significant effect was driven by a trend association, but this did not reach significance [$r_s(80) = .213$, p = .06; see Figure 10]. Those who believe more strongly in the malleability of mental illness were more likely to recommend therapy as a treatment option after reading CBTp effectiveness information.

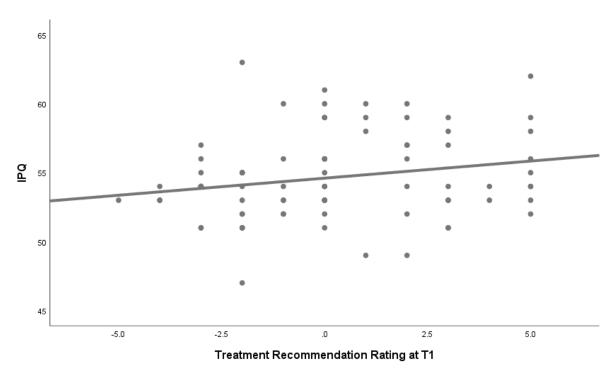


Figure 10. Association between IPQ and Treatment Recommendation Rating at Time Point 1. There was a trend positive association, with those believing more strongly in the malleability of mental illness choosing therapy as a treatment option (p = .06).

ii) Political attitudes

There were no significant main effects of political orientation (p = .15), dogmatic beliefs (p = .68) nor interactions (p = .21) for any treatment recommendation ratings (see Supplementary Table 7).

2. Therapy effectiveness ratings

As for treatment recommendation, there was no main effect of time [F(1, 79) = 1.99, p = .16], information type [F(1, 79) = .12, p = .73], nor interaction between time and treatment information type [F(1, 79) = .05, p = .83] on therapy effectiveness ratings (see Figure 11; Supplementary Table 5). A post hoc U-test showed that those who received CBTp-led neuroplasticity compared to generic treatment information did not record higher therapy effectiveness ratings at T1 (U = 789, p = .49).

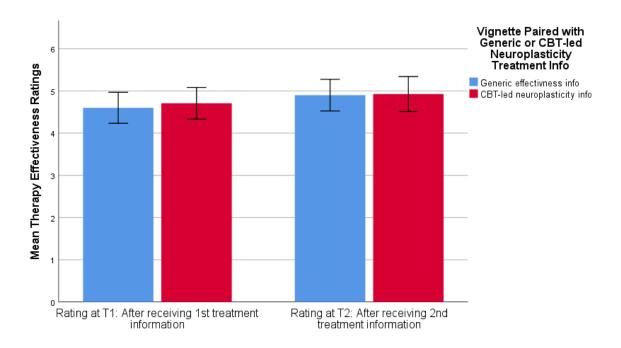


Figure 11. Clinician Therapy Effectiveness Ratings Following First and Second Set of Treatment Information. No differences were found.

2. Effects of individual differences on therapy effectiveness ratings

i) Illness perception beliefs

Amongst clinicians, we found the same effects as before regarding the main effect of IPQ for perceived therapy effectiveness at T1 (p = .004; see Figure 12a), with no interactions (p = .20). As in Study 1, there was a moderate positive association between IPQ and perceived therapy effectiveness at T1 [$r_s(80) = .296$, p = .01], with clinicians who believe more strongly in the malleability of mental illness being more likely to rate therapy as an effective treatment after

reading CBTp effectiveness information. This effect was significant for those who received CBTp-led neuroplasticity information [$r_s(39) = .343 p = .03$] but did not reach significance for those who received generic information [$r_s(41) = .286 p = .07$].

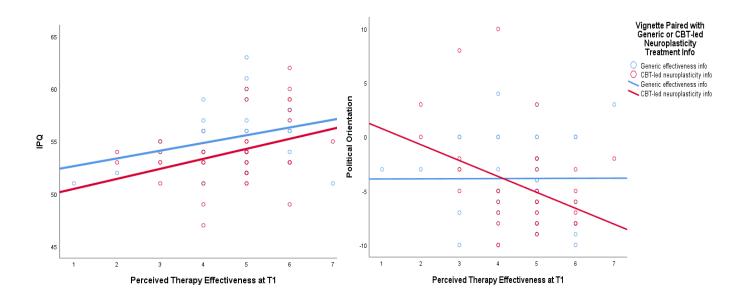


Figure 12a (left side). Association between IPQ and Perceived Therapy Effectiveness at Time Point 1. There was a significant positive association in the CBTp-led neuroplasticity (p = .03), but not in the generic information condition (p = .07).

Figure 12b. (right side). Association between Political Orientation and Perceived Therapy Effectiveness at Time Point 1. There was a negative trend association in the CBTp-led neuroplasticity condition (p = .06), but not in the generic information condition (p = .86).

ii) Political attitudes

There was a main effect of political orientation for perceived therapy effectiveness at T1 (p = .05; see Figure 12b), but this did not survive Bonferroni correction for the two comparisons (corrected p = .10). Follow-up tests confirmed the significant effect was driven by a non-significant negative trend association between political orientation and perceived therapy

effectiveness at T1 after receiving CBTp-led neuroplasticity information [$r_s(42)$ =-.307, p = .06; Bonferroni corrected p = .12]; but not after receiving generic treatment information [$r_s(39)$ = .03, p = .86]. Those who self-report as more politically liberal were more likely to give higher therapy effectiveness ratings, but only after receiving CBTp-led neuroplasticity information.

There was no main effect of dogmatic beliefs (p = .46) nor interactions (p = .37) for any therapy effectiveness ratings.

Discussion

The present study aimed to test whether a bias toward medication elicited by a predominantly biological (compared to psychosocial) description of psychosis could be reduced by presenting evidence of CBTp-led neuroplasticity (relative to control information). This was tested in two experiments.

Hypothesis 1: A predominantly biological description of psychosis, compared to predominantly psychosocial description, will lead participants to: H1a) prefer medication over psychological therapy, H1b) rate psychological therapy as a less effective treatment, and will elicit less empathy H1c)

The first hypothesis was confirmed. In Study 1, a predominantly biological description of psychosis elicited a preference for medication over therapy and significantly lower psychological treatment effectiveness ratings compared to participants who received a predominantly psychosocial description. This replicates findings from a previous study with clinicians (Lebowitz & Ahn, 2014). However, whilst the previous study did not find any significant difference regarding medication effectiveness ratings for schizophrenia, we additionally found that medication effectiveness ratings were significantly higher in the biological condition (see Supplementary material).

Study 2 was designed to only use the predominantly biological vignette because a priori power analysis indicated that when using both vignettes, we needed to have a sample size of 171 participants to have 90% power for detecting a medium effect size (Cohen's d= 0.5). We anticipated a limited sample size of clinicians and thus chose to use the biological vignette, where the largest effect was predicted a priori. Whilst the clinicians were only presented with biological explanations of psychosis, they rated medication as significantly more effective than

psychological therapy overall. This finding is consistent with previous research conducted with clinicians (Lebowitz & Ahn, 2014) and with patients as well (Iselin & Addis, 2003; Kemp et al., 2014). This result was found even though clinicians were presented with evidence about the effectiveness of CBTp before recording their ratings. Previous research has shown that the more a psychiatric disorder is considered to have a biological aetiology, the more effective medication is expected to be in treating it (Ahn et al., 2009). As there is evidence that mental health clinicians consider schizophrenia to be biological (Ahn et al., 2009), one possibility is that their pre-existing beliefs about its aetiology over-rode any impact the therapy effectiveness intervention may have had on their beliefs about best treatment.

Our finding adds further support to the notion that biological conceptualisations of mental illness can reduce confidence in non-biological treatments such as therapy (Carter et al., 2018; Lebowitz & Appelbaum, 2017). Researchers have proposed that such descriptions compel both members of the public and clinicians to choose medication, which has a biological target, rather than therapy, which is perceived as targeting the mind, as the most effective treatment method (Lebowitz & Appelbaum, 2017).

Hypothesis 1c) was not confirmed; results indicated that a predominantly biological description of psychosis did not elicit less empathy than a predominantly psychosocial description. This finding contrasts with previous evidence (Lebowitz & Ahn, 2014) which showed that biological descriptions evoked comparatively less empathy amongst clinicians. This difference in finding could be attributed to the measures used, with the previous study using a composite measure of six adjectives rather than a single rating to arrive at an empathy score (Lebowitz & Ahn, 2014). We therefore cannot draw any strong conclusions from our failure to replicate the finding from the previous study.

Hypothesis 2: Presenting evidence of CBTp-led neuroplasticity (relative to generic information about clinical effectiveness) will lead participants to: H2a) recommend psychological therapy over medication, H2b) rate psychological therapy as a more effective treatment.

The second hypothesis was not confirmed amongst the general public at the group level (see Hypothesis 3 for individual difference effects). Although there was a significant shift in ratings post intervention, the CBTp-led neuroplasticity information did not have a greater impact than the generic information. A shift in appraisals was found regardless of whether CBTp-led neuroplasticity or generic treatment information was presented after each vignette. This suggests that members of the general public are equally persuaded by any information of CBTp effectiveness, regardless of whether it is couched biologically. The finding could be attributed to the generic treatment information being more compelling than the evidence base actually suggests. The information presented implied a large effect size (see Figure 1) whereas meta analyses suggests effect sizes for CBTp are weak and variable (Jauhar et al., 2014; Laws, Darlington, Kondel, McKenna, & Jauhar, 2018). The finding could also be attributed to the fact that the vignette explanation was predominantly rather than purely biological as we sought to mirror real world scenarios in which both kinds of information are typically present (Lebowitz & Ahn, 2014; see Appendix E). The shift in perceived treatment effectiveness which we found mirrors the shift in appraisals found in previous malleability-focused studies, in constructs such as the desire for social distance (Lebowitz & Ahn, 2012), prognostic pessimism (Lebowitz et al., 2013) and perceived self-efficacy (Lebowitz & Ahn, 2015, 2018), and further strengthens the case for interventions which cultivate incremental beliefs as a mechanism for change (Howell, 2017). Although the hypothesised effect was not found, results suggest that the general public's perceptions of the credibility of psychological therapy are amenable to change when presented with empirical evidence of treatment effectiveness.

Similarly, with clinicians in Study 2, at the group level there was also no significant difference in therapy or medication effectiveness ratings, regardless of whether CBTp-led neuroplasticity or generic treatment information was paired with the vignette they read. However, exploratory analyses revealed that psychologists made a significantly stronger preference for therapy after reading CBTp-led neuroplasticity information compared to generic treatment information. There was no significant difference in such ratings amongst psychiatrists. This effect suggests that even when a condition such as psychosis is described in biological terms, an intervention which targets the biological phenomena underlying the symptoms becomes more credible amongst psychologists (Lebowitz & Appelbaum, 2019). Compared to psychologists, psychiatrists were significantly more likely to show a preference for medication, rate medication as more effective and therapy as a less effective treatment method. This effect could be explained by the fact that psychiatrists' beliefs about the efficacy of therapy for particular conditions such as psychosis are more firmly entrenched (Kingdom, Sharma, & Hart, 2004; Magliano et al., 2013) and therefore their appraisals are less responsive to influence, regardless of whether the psychological treatment information presented targets the biology of the condition described.

Hypothesis 3: Change in treatment effectiveness ratings will be modulated by pre-existing beliefs and attitudes.

H3a) Participants who believe more strongly in the malleability of mental illness will rate psychological therapy as a more effective treatment.

Hypothesis 3a was confirmed. Study 1 participants who believe more strongly in the malleability of mental illness (as measured by IPQ; Marcus et al., 2014) were more likely to rate therapy as an effective treatment at baseline, independent of whether they received any treatment information. This result lends some support to previous research which has shown

that perceived control and optimism about change increase the likelihood of engaging in therapy (Freeman et al., 2013; Marcus et al., 2014a). Amongst clinicians, a similar effect was found, but specifically for CBTp-led neuroplasticity rather than generic treatment information. This suggests that for participants who believe more strongly in the malleability of mental illness, psychological therapy was perceived as more effective when it was presented as targeting the neurobiology of the mental illness described, as hypothesised.

H3b) Following the presentation of CBTp evidence, those espousing stronger political attitudes will be less likely to change their therapy effectiveness ratings

Hypothesis 3b was confirmed. Study 1 participants who report higher levels of dogmatic beliefs made smaller increments to their therapy effectiveness ratings after reading both kinds of treatment effectiveness information. This supports previous research which showed more dogmatic individuals are less likely to integrate disconfirmatory evidence after making a decision, thereby restricting scope for the recognition and reversal of incorrect choices (Rollwage et al., 2018). We propose that more dogmatic individuals were more reluctant to change their initial therapy ratings, despite being presented with empirical evidence of its effectiveness. This may have important implications for disseminating information about the helpfulness of psychological therapies. Clinicians who self-report as more politically liberal were more likely to give higher therapy effectiveness ratings, specifically if receiving CBTp-led neuroplasticity information. This lends some support to H2b and also suggests that there is an important difference between more liberal and conservative people regarding the treatment information presented, with it having an effect on perceived helpfulness of therapy amongst the former but not the latter. Indeed, previous research has found that individual differences in political ideology influence acceptance of factual and nonfactual information, with more

politically liberal individuals reporting greater agreement with scientific facts (Medlin et al., 2019).

Clinical Implications

Biological and psychosocial explanations of psychopathology have a real impact in terms of how useful certain members of the general public view different forms of treatment. If biological explanations of psychiatric disorders are associated with positive attitudes about medication (Angermeyer et al., 2017), this will impact public discourse on the perceived helpfulness of psychiatric medication, which in turn has the potential to influence the support network of a patient who is considering treatment options (Mojtabai, 2009). Clinicians may not be aware of their own potential biases toward a particular causal model, as this could implicitly influence their treatment preferences (Carter, Read, Pyle, Law, et al., 2017). Furthermore, the way in which they present the symptoms of mental health conditions can not only influence the type of treatment they recommend themselves, but also the type of treatment a patient perceives as helpful. Extrapolating from our results, this can have implications for treatment adherence and a patient's willingness to try new treatments in clinical practice (Marsh & Romano, 2016). Patients should receive care from a truly multidisciplinary team (including both psychiatrists and psychologists) so that they can be made aware of the efficacy of different forms of treatment, both medical and psychological, and make informed decisions based on the latest evidence. Brief interventions which target perceptions of the helpfulness of psychological treatment and the possibility of change may increase active engagement in CBTp, thereby increasing the likelihood of a successful outcome. Such interventions could be made readily available online or readily used in relevant contexts such as GP surgeries or early intervention in psychosis settings.

Advances in fMRI technology have shown us that psychological therapy can lead to sustained change at the neurobiological level. When this information is presented to clinicians, its impact is modulated by their training background. Interestingly, amongst those with psychological training, such evidence enhances their preference for psychological therapy as a treatment option, despite the prevailing dominant preference for medication to treat psychosis in the field of mental health (Calton et al., 2009; Carter, Read, Pyle, Law, et al., 2017). Amongst those with biomedical training, the same evidence does not impact their treatment recommendations. Even if such clinicians were convinced by the evidence of CBTp-led neuroplasticity, qualitative feedback suggests that there are factors involved which would prevent them from recommending psychological therapy as a treatment option. Such factors include how motivated the patient is to engage in therapy, the perceived need for medication to stabilise a patient's psychotic symptoms before they are able to meaningfully engage in therapy and the length of wait list times for therapy. Indeed, a previous study has shown that a high proportion of clinicians reported not being able to offer CBTp due to limitations in resources (Carter, Read, Pyle, Law, et al., 2017), which is reflective of previous findings critical of the lack of psychological interventions in mental health services throughout the United Kingdom (Berry & Haddock, 2008; Goldhamer & Marano, 2012). Such obstacles are a reminder of the systemic factors which influence treatment provision, apart from the chosen preference of a patient or clinician.

Lastly, our analyses of modulating factors showed that a CBTp-led neuroplasticity treatment information intervention interacts with one's pre-existing illness perception beliefs and even political attitudes in influencing any change in treatment appraisal which might occur. Such beliefs are an important consideration when designing and implementing interventions aimed at inducing incremental belief patterns as a mechanism for change (Howell, 2017). At

relevant services such as early intervention in psychosis settings, highlighting the degree to which patients have control over their symptoms can influence self-efficacy, thereby facilitating their engagement with treatment (Freeman et al., 2013). On clinician training courses, an emphasis on the scientist-practitioner model (Peterson & Park, 2005), which urges clinicians to allow empirical research to influence their applied practice, is something that could attenuate the negative impact of one's dogmatic beliefs when encountering disconfirmatory evidence.

Limitations

There are a number of study limitations which should be considered when interpreting the current results. Beginning with the study design, our treatment information intervention consisted of a fMRI image (CBTp-led neuroplasticity condition) or line graph (generic condition) accompanied by a brief paragraph describing the effectiveness of CBTp. Previous studies have used an 5-8 minute audio-visual presentation to inform participants about the malleability of a given psychiatric disorder (Farrell, Lee, & Deacon, 2015; Lebowitz et al., 2013; Lebowitz & Ahn, 2015, 2018). This might partly explain why the hypothesised effects of type of treatment information interacting with vignette explanation were not found at the group level. In addition, previous studies have used a manipulation check to ensure that participants have properly understood information presented (Kemp et al., 2014; Lebowitz & Appelbaum, 2017). This could have been included in the current study to check understanding of vignette information or CBTp-led neuroplasticity treatment information to establish whether participants sufficiently held relevant information in mind when recording the main variables of interest. However, the vignette and treatment information underwent many iterations to ensure they were as comprehensible as possible and the amount of information presented was relatively short compared to that in the audio-visual presentations. Pilot feedback suggested that the information was clear and so we are relatively confident that participants understood the presented material.

Sampling

Online samples like those recruited on Prolific tend to be far more diverse than typical samples used in psychology research (Palan & Schitter, 2018) and are also less likely to have strong prior expectations and biases about psychological therapies. However, we acknowledge that recruiting participants via online platforms has the potential to introduce sampling bias in that users are self-selecting and a truly age and sociodemographic representative sample is not achieved, which has implications for the generalisability of findings. We aimed to get as representative a sample as possible and so used multiple online platforms rather than restricting to student platforms.

Regarding the Study 2 sample, clinician recruitment was self-selecting, which introduces potential sampling bias. Furthermore, from our attrition rate data (31%), we can deduce that at least some of the clinicians were limited for time, which could have potentially influenced the results. Indeed, much research suggests that clinicians are notoriously difficult to recruit, possibly due to the limited free time they have to take part in studies (Hysong et al., 2013). Future research could replicate this experiment in a more controlled environment, where participants commit adequate time to process information without distractions that could confound results. However, we achieved a sample of clinicians in their natural environment and thus demand characteristics would have been lower than if the study was conducted in a laboratory setting.

Effort/Consistency Checks

Outliers which could have impacted the results were excluded from the dataset. We consider that Study 1 data may be more prone to participant effort because it involved financial incentives for taking part whereas Study 2 involved clinicians with more intrinsic motivation as there was no financial incentive. Inspection of times across both studies indicated that completion times were relatively uniform. In addition, the large sample size in Study 1 (n = 270) is likely to reduce the impact of any outliers due to variations in effort.

Measures

Other methodological limitations include some of the measures used to capture personality and attitudes. The self-report political orientation scale was chosen because it has a loading of .95 on the political orientation factor from a previous study on radical beliefs (Rollwage et al., 2018). However, this measure assumes that participants have a knowledge of what politically liberal or conservative means. Indeed, a frequency distribution of political orientation data shows a non-normal distribution, with a higher than expected number of participants choosing "0" (neither liberal nor conservative) on the scale. This could have been because some participants did not understand the implications of being liberal or conservative, and so answered '0' because they were unsure.

Some of the clinician feedback expressed dissatisfaction at recording treatment recommendation as a single linear construct, with a few responses stating that they would prefer to have had the opportunity to offer a combination of both or begin treatment with medication and then follow up with a therapeutic intervention. We included this single linear construct because we wanted to capture each participant's treatment preference and safeguard against those who would rate both therapy and mediation highly on the subsequent effectiveness scales. We recognise that a single slider response to denote recommendation of either medication or psychological therapy may not reflect clinical practice. However, research indicates that of

patients who could benefit from psychological therapy, only about one in ten are actually offered it (Schizophrenia Commission, 2012), suggesting that for those with psychosis, treatment options may indeed be more polarised (medication or psychological therapy). Other clinician feedback stated that they would have liked to offer therapy but know that there is currently a year long wait for such an intervention. To address this, we could have stated that the therapy option is readily accessible, but also wanted to make the study as ecologically valid as possible.

As previously stated, the presented generic treatment information line graph was more compelling than the evidence base actually suggests. This presents methodological issues in the form of unduly biased responses and ethical issues in the form of participants being left with the impression that CBTp is more effective than it actually is. To mitigate this, the study should have included a debrief advising that the effectiveness of CBTp is more equivocal than as depicted in the line graph they saw, thereby contextualising the state of the evidence base and highlighting that participants should not make recommendations based on this graph.

Some of the ratings collected such as treatment recommendation and treatment effectiveness involved ordinal rather than continuous data. We acknowledge that we could have used larger ranges on the scales to mitigate this . However, the decision was to replicate the design of a previous study (Lebowitz & Ahn, 2014) and we reasoned that a narrow scale was more intuitive for participants. Non-parametric tests were used in our analyses of the data where the assumption of normal distribution was violated.

Directions for future research

There are numerous directions for future research regarding the impact of biological explanations of psychopathology. Firstly, more research is required that directly examines the

association between type of psychopathology explanation and clinicians' treatment preference, and the moderation of this relationship by the training they receive. Due to study time limitations, clinicians were not presented with psychosocial explanations nor did they give baseline ratings before being presented with treatment information. Future research should elicit baseline ratings so that it can conclude whether presented CBTp-led neuroplasticity (relative to generic) treatment information reduces or increases any treatment bias that might emerge from biological (relative to psychosocial) explanations, and whether any effects are modulated by type of training background.

Additional experimental intervention research is needed to test whether the presentation of treatment effectiveness information has any impact on the perceived helpfulness of psychological therapy amongst patients themselves, most likely the most clinically meaningful of all. There is a relative lack of empirical evidence which has investigated this cohort (Lebowitz & Appelbaum, 2019) and so future research could not only investigate the impact of clinical effectiveness information on their treatment preference, but also measure whether the cultivation of an incremental view of a psychiatric disorder is conducive to increased willingness to engage in treatment. Lastly, future research should collect objective measures to supplement subjective measures of variables of interest, which are vulnerable to contamination due to social desirability response bias (Krumpal, 2013). An attempt was made to collect an objective measure in the present study but had insufficient power for analysis (see Critical Appraisal).

In conclusion, this study builds on previous research which suggests that biological explanations of psychopathology have clear implications for the perceived helpfulness of different forms of treatment. Clinicians need to be aware that the way in which they conceptualise a patient's mental health difficulties can not only implicitly influence their own

treatment recommendation, but also impact the type of treatment a patient may choose. When the public is presented with CBTp effectiveness evidence, it can reduce a bias toward medication elicited by biological explanations, while the effect amongst clinicians is more varied, depending on their training background. Biological explanations have undoubtedly improved our understanding of psychopathology and it is unlikely that their dominance will abate anytime soon (Lebowitz & Appelbaum, 2019). However, this research highlights the possible unintended implications for treatment decisions which such explanations evoke. We propose that biological research should further acknowledge the brain's ability to adapt and reorganise in response to environments and experiences (Lozano, 2011), thereby giving more consideration to the influence of psychological and environmental factors in the manifestation of and recovery from psychiatric disorders (Deacon, 2013). As ever, treatment decisions should be based on clinical guidelines and the latest empirical evidence to ensure a consistent and effective approach to mental health service provision.

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Supplementary Materials

Results

Medication effectiveness ratings

Study 1: general population

As with treatment recommendation and therapy effectiveness ratings, there was a main effect of time $[F\ (1.62,431.85)=37.34,p<.001]$, explanation type $[F\ (1,266)=4.18,p=.04]$, an interaction between time and explanation type $[F\ (1.62,431.85)=6.92,p=.002]$ and no effect of treatment information type $[F\ (1,266)=1.80,p=.18]$ nor interaction between explanation type and treatment information type $[F\ (1,266)=.07,p=.79]$, see Supplementary Table 1]. Regarding the main effect of time, post hoc t-tests confirmed that participants significantly decreased their medication effectiveness ratings from T1 to T2 $[t\ (269)=5.47,p<.001]$, and again from T2 to T3 $[t\ (269)=4.97,p<.001]$. Concerning the interaction effect, post-hoc t-tests revealed that participants perceived medication to be significantly more effective when symptoms were explained in biological rather than psychosocial terms at T1 $[t\ (268)=3.77,p<.001]$, but not at T2 $[t\ (268)=1.25,p=.21]$ or T3 $[t\ (268)=.73,p=.46]$; see Supplementary Figure 1].

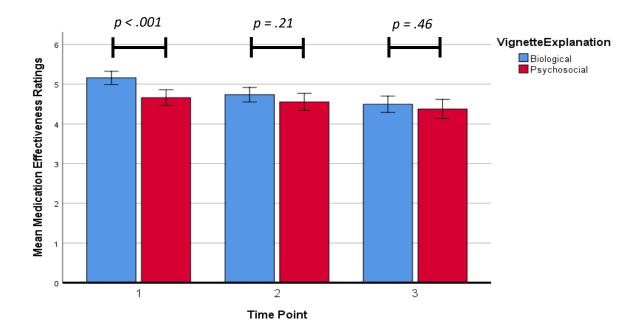


Figure 1. *Medication Effectiveness Ratings by Type of Explanation Participants Received (Biological versus Psychosocial)*. Ratings were higher for biological than psychosocial condition at T1, but not T2 or T3.

1. Effects of individual differences on medication effectiveness ratings

There were no main effects of IPQ (p = .91), political orientation (p = .36), dogmatic beliefs (p = .11) nor interactions (p = .28) for any medication effectiveness ratings.

Study 2: mental health clinicians

Medication effectiveness ratings

A repeated measures MANOVA revealed that there was no main effect of time (F(1, 79) = .24, p = .62), information type (F(1, 79) = .21, p = .65), nor interaction between time and explanation type (F(1, 79) = .24, p = .62) on medication effectiveness ratings (see Supplementary Figure 2).

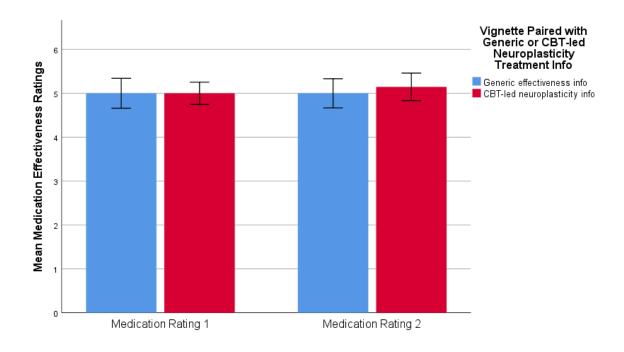


Figure 2. Clinician Medication Effectiveness Ratings Following First and Second Set of Treatment Information. No differences were found.

2. Effects of individual differences on medication effectiveness ratings

There were no main effects of IPQ (p = .88), political orientation (p = .35), dogmatic beliefs (p = .86) nor interactions (p = .39) for any medication effectiveness ratings.

Table 1. Study 1 Repeated Measures Analysis of Variance

Effect	Dependent Variable	Mean Square	df	F	р
Time	Treatment Rec	633.47	1.42	186.50	< .001
	Therapy	112.86	1.42	146.92	< .001
	Medication	18.23	1.62	37.34	< .001
Explanation	Treatment Rec	315.56	1	28.61	< .001
	Therapy	35.23	1	17.26	< .001
	Medication	14.35	1	4.18	.04
Treatment	Treatment Rec	.05	1	.004	.95
Information	Therapy	.14	1	.07	.79
	Medication	6.18	1	1.80	.18
Time x Explanation	Treatment Rec	76.66	1.42	22.57	< .001
	Therapy	6.64	1.42	6.64	.001
	Medication	3.38	1.62	6.92	.002
Explanation x	Treatment Rec	1.09	1	.10	.75
Treatment Information	Therapy	.12	1	.06	.81
information	Medication	.24	1	.07	.79
Time x	Treatment Rec	.81	1.42	.24	.71
Explanation x	Therapy	.07	1.42	.09	.85
Treatment Information	Medication	.97	1.62	1.98	.15

Table 2. Study 1 Repeated Measures Analysis of Variance with IPQ as Covariate

Effect	Dependent Variable	Mean Square	df	F	р
Time	Treatment Rec	7.98	1.42	2.34	.12
	Therapy	5.51	1.42	7.27	.003
	Medication	.09	1.62	.19	.79
Explanation	Treatment Rec	2.45	1	.22	.64
	Therapy	.49	1	.24	.62
	Medication	.02	1	.01	.94
Treatment Information	Treatment Rec	4.08	1	.37	.54
	Therapy	1.05	1	.52	.47
	Medication	.00	1	.00	.99
IPQ	Treatment Rec	21.19	1	1.92	.17
	Therapy	3.58	1	1.76	.19
	Medication	.49	1	.01	.91
Time x	Treatment Rec	.38	1.42	.11	.83
Explanation	Therapy	.76	1.42	1.004	.34
	Medication	.55	1.63	1.14	.31
Time x	Treatment Rec	.98	1.42	.29	.67
Explanation x Treatment	Therapy	.04	1.42	.06	.89
Information X IPQ	Medication	1.01	1.63	2.08	.14

Table 3. Study 1 Repeated Measures Analysis of Variance with Political Orientation and Dogmatic Beliefs as Covariates

Effect	Dependent Variable	Mean Square	df	F	р	
Time	Treatment Rec	49.31	1.44	14.94	< .001	
	Therapy	12.98	1.42	17.56	< .001	
	Medication	2.84	1.61	5.83	.01	
Explanation	Treatment Rec	99.89	1	9.34	.01	
	Therapy	.59	1	.29	.60	
	Medication	14.51	1	4.21	.04	
Treatment	Treatment Rec	.53	1	.05	.83	
Information	Therapy	1.04	1	.51	.48	
	Medication	5.56	1	1.61	.21	
Dell'ite el	Total or al Rec	7.50	4	74	40	
Political Orientation	Treatment Rec	7.56	1	.71	.40	
	Therapy	1.11	1	.54	.46	
	Medication	2.96	1	.86	.36	
Dogmatic	Treatment Rec	10.19	1	.95	.33	
Beliefs	Therapy	.05	1	.03	.87	
	Medication	8.94	1	2.60	.11	
Time x	Treatment Rec	6.90	1.44	2.09	.14	
Explanation	Therapy	2.98	1.42	4.02	.03	
	Medication	1.35	1.61	2.77	.08	

Time x Explanation x Treatment	Treatment Rec	.11	1.44	.03	.93
	Therapy	.05	1.42	.06	.89
Information x Political Orientation	Medication	.16	1.61	.34	.67
Time x	Treatment Rec	.87	1.44	.26	.69
Explanation x Treatment	Therapy	.04	1.42	.06	.89
Information x Dogmatic Beliefs	Medication	.61	1.61	1.25	.28

Table 4. Study 1 Empathy t-Test Comparing Biological and Psychosocial Conditions.

	Biolo	Biological (n = 158)		Psychosocial (n = 112)		p	Cohen's d
	M	SD	M	SD	_		
Empathy Rating	7.37	1.67	7.11	2.00	1.16	.25	0.14

Table 5. Study 2 Repeated Measures Analysis of Variance

Effect	Dependent Variable	Mean Square	df	F	р	
Time	Treatment Rec	14.30	1	2.43	.12	
	Therapy	2.73	1	1.99	.16	
	Medication	.22	1	.24	.62	
Treatment Information	Treatment Rec	.50	1	.06	.81	
	Therapy	.18	1	.12	.73	
	Medication	.22	1	.21	.65	
Time x	Treatment Rec	.69	1	.12	.73	
Treatment Information	Therapy	.07	1	.05	.83	
	Medication	.22	1	.24	.62	

Table 6. Study 2 Repeated Measures Analysis of Variance with IPQ as Covariate

Effect	Dependent Variable	Mean Square	df	F	р	
Time	Treatment Rec	14.51	1	2.55	.12	_
	Therapy	7.00	1	5.14	.03	
	Medication	.25	1	.28	.60	
Treatment Information	Treatment Rec	14.93	1	1.71	.20	
	Therapy	1.80	1	1.18	.28	
	Medication	8.03	1	.00	.99	
IPQ	Treatment Rec	14.56	1	1.67	.20	
	Therapy	4.75	1	3.10	.08	
	Medication	.02	1	.02	.88	
Time x	Treatment Rec	.29	1	.05	.82	
Treatment Information	Therapy	.98	1	.72	.40	
	Medication	.08	1	.09	.77	
Time x	Treatment Rec	.40	1	.07	.79	
Treatment Information x	Therapy	.89	1	.65	.42	
IPQ	Medication	.08	1	.09	.76	

Table 7. Study 2 Repeated Measures Analysis of Variance with Political Orientation and Dogmatic Beliefs as Covariates

Effect	Dependent Variable	Mean Square	df	F	p
Time	Treatment Rec	2.02	1	.43	.52
	Therapy	.30	1	.21	.65
	Medication	.62	1	.56	.46
Treatment Information	Treatment Rec	4.89	1	.72	.40
	Therapy	.16	1	.12	.73
	Medication	.19	1	.31	.58
Political Orientation	Treatment Rec	14.52	1	2.15	.15
	Therapy	2.23	1	1.65	.21
	Medication	.56	1	.90	.35
Dogmatic	Treatment Rec	1.17	1	.17	.68
Beliefs	Therapy	.74	1	.55	.46
	Medication	.02	1	.03	.86
Time x	Treatment Rec	8.10	1	1.73	.20
Treatment	Therapy	.91	1	.65	.42
Information	Medication	3.01	1	.00	.99
Time x	Treatment Rec	.38	1	.08	.78
Treatment	Therapy	.11	1	.08	.78
Information x Political Orientation	Medication	.84	1	.76	.78

Time x	Treatment Rec	7.45	1	1.59	.21
Treatment Information x	Therapy	1.17	1	.84	.37
Dogmatic Beliefs	Medication	.10	1	.09	.76

Chapter 3: Critical Appraisal

Introduction

This appraisal provides a critical reflection on the current study. The appraisal will briefly describe the reasons why I was drawn to this particular study, outlining my previous experience in attribution research. Reflections will then be made on methodological issues which arose whilst conducting the study, highlighting issues such as the study design, attempts at collecting a behavioural measure, recruitment of eligible clinicians and wider contextual factors which impacted data collection. Finally, reflections are offered on how the research has impacted my development as a researcher and clinician.

Background

I chose to conduct research on the impact of biological explanations of psychopathology for several reasons. Firstly, despite advances in our biomedical understanding of psychopathology, the prevalence of mental illness is rising, not falling (Deacon, 2013). Although the increase in prevalence could be attributed to improved diagnostic methods and the reduction of stigma around mental health in wider society, I still question why the dominant paradigm is not having more success in mental health outcomes or leading to meaningful improvements in clinical practice (Deacon, 2013; Insel, 2009). Secondly, the increasing prevalence of medication to treat mental health difficulties is something that has been well documented (Bastiampillai, Allison, Harford, Perry, & Wong, 2019) despite relatively small effect sizes in some cases (Linde et al., 2015), and the evidence that a combined approach typically outperforms medication or psychological therapy alone (Huhn et al., 2014; Moss et al., 2016). Third, I have always had an interest in attributions and their implications for human behaviour (Newcomb & Heider, 1958). I first developed an interest in attribution theory (Schmidt & Weiner, 1988) whilst studying for my B.Sc. in psychology. I was involved in a project about self-serving attributional

bias (Greenberg, Pyszczynski, Burling, & Tibbs, 1992); specifically, gender differences in the reasons people attribute their depression to. Subsequently, my master's dissertation investigated how one's implicit theory of intelligence has implications for the types of reasons they attribute their failures to (Hong, Dweck, Chiu, Lin, & Wan, 1999). This attribution pattern then has implications for how one responds after a setback. Following on from this line of research, I was drawn to the idea that the different reasons people attribute to the occurrence of psychiatric disorders have a subsequent impact on their attitudes and how they then respond (Lebowitz & Appelbaum, 2019).

Methodological Issues

Study design

We decided on an online experiment to test our hypothesis because internet-based questionnaires have become a standard tool in the field of psychology (Bartneck, Duenser, Moltchanova, & Zawieska, 2015) and recruitment of participants via Prolific offered us the opportunity to recruit a sample which is more diverse than typical samples used in psychology research (Palan & Schitter, 2018). In Study 2, we observed a high attrition rate in the earlier stages of recruitment. Evidence suggests a positive association between experiment length and attrition rate (Hoerger, 2010) and it is possible that participants did not complete the experiment because the questionnaire battery was perceived as too time consuming. In order to address this, we moved the questionnaire measures to the end of the study, after participants had completed the experiment, and subsequently found the study completion rate increased. Researchers must consider whether the benefit of testing multiple hypotheses with various questionnaires outweighs the potential for participant attrition and where possible, use shorter questionnaires which may assist study completion rates.

A priori power analysis indicated that we needed to recruit 170 participants for Study 1 in order to detect a medium-sized effect. The within-subjects element of the study was something that my supervisor and I discussed and hesitated with at first as it meant that each participant had to complete a set of ratings three times. We were aware of findings which support the paradoxical entrenching of viewpoints rather than changing opinions in the direction which reading material suggests (Bail et al., 2018). If recruitment of a sufficient number of participants were not an issue, we would have made the study design exclusively between subjects, with each participant only having to read a single vignette and treatment information description. Despite early concerns, the ratings did in fact move in the hypothesised direction and a significant main effect of time was found for each of the dependent variables in Study 1.

Once the study design was confirmed, the vignettes and treatment information pages went through a series of iterations to make them as comprehensible and acceptable as possible. Although we were using vignettes from a previously validated study (Lebowitz & Ahn, 2014c), we made adjustments to them in order to enhance ecological validity and make them acceptable to clinicians who would have a working knowledge of psychosis. Moreover, the language used for the treatment information pages had to be carefully considered in order to reflect NICE guidelines (National Institute of Health and Clinical Excellence, 2014) and control information had to be as closely matched to the CBT-led neuroplasticity information as possible to control for any potential demand characteristics. We subsequently carried out a series of pilot studies on both members of the general public and clinicians in order to elicit feedback that would inform further iterations. Throughout this process, I learned about the importance of both supervision consultation and pilot testing to gauge comprehensibility and acceptability of information presented.

Participant recruitment

From an early stage in the project, it was decided that we would conduct two studies, with the first recruiting a sample from the general public and the second recruiting a sample of mental health clinicians. We decided that recruiting from the general population was preferable to recruiting a sample of university undergraduate students because we reasoned that compared to psychology students, the general population would be naive to the treatment information intervention presented in the study. Regarding Study 2, we knew that it would be difficult to recruit the required number of clinicians for a medium-sized effect. We were also mindful that clinicians would be more limited for time and not be able to complete the three sets of ratings required in Study 1 (Hysong et al., 2013). In order to address this issue, we altered the design of Study 2 so that only two sets of ratings were required. However, the drawback to this design was that we did not collect baseline ratings before any treatment information was presented. It was therefore difficult to explore the modulating impact of pre-existing beliefs on change scores, as there was no change in treatment appraisal from baseline to after being presented with treatment information.

COVID-19 pandemic and impact on data collection

From the outset, we recognised that clinicians are a notoriously difficult sample to recruit and began constructing a plan on how to disseminate the study within relevant institutions and NHS trusts across London. The Royal College of General Practitioners and Royal College of Psychiatrists initially gave us permission to advertise the study in their respective newsletters, but unfortunately this did not materialise. In February, The Royal College of General Practitioners stated that they were not promoting any external studies until a set of terms and conditions were decided by the board. The Royal College of Psychiatrists were due to publish the study at the end of March but unfortunately the outbreak of COVID-19 prevented this from

going ahead. Once the World Health Organisation declared COVID-19 a pandemic, we ceased recruitment of participants to avoid potential contamination of data in the context of a crisis. The lack of publication in the newsletters, coupled with the premature cessation of online data collection, meant that we did not recruit as many clinicians in Study 2 as we could have, which then impacted the power of our findings.

Omitted behavioural measure

Much research has suggested that there is an over-reliance on self-report measures in psychological research (Moshagen, Hilbig, Erdfelder, & Moritz, 2014) Studies have found that such measures are vulnerable to possible contamination in the form of social desirability bias (Krumpal, 2013). This bias has been found to affect the measurement of personality variables (Mick, 1996), attitudes (R. J. Fisher, 1993) and self-reported behaviours (Mensch & Kandel, 1988). Indeed, researchers have proposed that greater resources are needed to go beyond psychological self-reports to observe actual behaviour (Corrigan et al., 2012). In order to balance our reliance on self-reported data we chose to include a behavioural measure in the form of a simple visual perception task. The simple task required participants to complete a series of perceptual discrimination judgements as to which of two flickering patches contained a greater density of dots, followed by confidence ratings in their choices. The task has been used in previous studies to measure one's metacognitive sensitivity, which quantifies participants' ability to discriminate correct from incorrect decisions (Rollwage et al., 2018).

We recruited a total of 69 participants to do the metacognition task, which took a total of 21 minutes to complete, and then used a Matlab program to convert raw scores into a score which would indicate the level of insight into the accuracy of their choices. We had hoped that the data collected using this behavioural measure would supplement the subjective data collected on political attitudes and dogmatic beliefs. Specifically, we wanted to investigate

whether those espousing radical beliefs hold an unjustified certainty in the accuracy of their choices and are less likely to update their decisions when confronted with disconfirmatory evidence (Rollwage et al., 2018). Preliminary analyses showed those who have better insight into the accuracy of their decisions in the task were more likely to resist treatment appraisal change. However, due to budget restrictions, we could not pay more participants to complete the behavioural measure and thus had insufficient power to make conclusive findings regarding associations with radical beliefs.

Clinician feedback

A number of weaknesses to the study have been previously discussed in the empirical paper. Qualitative feedback from clinicians in Study 2 pointed out a number of additional design issues which they said made it difficult to record some of their ratings. Firstly, a few clinicians stated that there wasn't enough information in the patient formulation to make treatment-related decisions. They stated that they would not usually base treatment decisions on so little patient information. We had considered providing more patient information but the vignettes used had been validated in a previous study (Lebowitz & Ahn, 2014c) and we did not want to overload participant working memory as they moved through the study.

Another feedback point related to the treatment options which were made available in the study. Some clinicians stated that the dichotomy offered between psychological therapy and medication was neither helpful nor realistic, yet the study offered treatment options as a binary choice. As previously stated in chapter 2, we recorded treatment recommendation as a single linear construct because we wanted to protect again those who would rate medication and therapy effectiveness ratings as equally high. However, a further feedback point was made about CBTp being the only treatment described in the study. Indeed, one participant stated that CBT for psychosis was the standard treatment offered (National Institute of Health and Clinical

Excellence, 2014) despite meta analyses which suggest that effect sizes are weak and variable (Jauhar et al., 2014). They reported that they would advise family therapy for the patient described in the vignette. We acknowledge that describing other forms of psychological therapy would have enhanced the ecological validity of the study. However, the treatment information intervention pertained to neurobiological changes following CBTp and we did not want to overload participant working memory by describing other forms of psychological therapy.

Impact on researcher

Conducting this piece of research has influenced me in a number of ways that will have implications for my future practice as a clinician. In the first instance, conducting the literature review has highlighted the evolution of a dominant paradigm in the field of mental health, from an early emphasis on Freudianism and other psychodynamic conceptualisations to the current emphasis on biological explanations. This dominant paradigm has had a wide-ranging impact on discourse both at the societal level and in the clinical context, and has implications for how useful we perceive a particular form of treatment to be, even when evidence suggests that it might not be as effective as considered (Deacon, 2013). Conducting the literature review has also taught me that clinicians are not immune to a mind-body dualism (Miresco & Kirmayer, 2006) and results from the study do indeed suggest that the more a disorder is considered to have a biological aetiology, the more effective medication is perceived to be in treating it.

Secondly, the differences in treatment recommendation between psychologist and psychiatrist participants has highlighted how mental health clinicians with different training backgrounds may hold biases toward a particular causal model, which could in turn implicitly influence their treatment preferences (Carter, Read, Pyle, Law, et al., 2017). The process has alerted me to blind spots that different mental health professionals may hold and is something

I will consider when consulting in future multidisciplinary team meetings. Indeed, no part of the biopsychosocial model should be neglected in favour of another when accounting for the origin of a mental health difficulty (Deacon, 2013). The study results indicate that the way in which we present symptoms of mental health difficulties will not only influence the type of treatment we recommend as clinicians, but also the type of treatment a patient might perceive as helpful. This important point is something that I will strive to remember in my own future clinical practice, when I hope to be considering clinical guidelines as well as the latest empirical evidence when collaboratively forming a treatment plan.

Qualitative feedback gathered from clinicians in Study 2 has informed me of how much faith clinicians may put in psychological therapy and also highlighted the systemic factors which might dissuade a psychiatrist or general practitioner from offering it as a treatment option. Indeed, a number of participants cited wait time lists, the degree of family involvement or even patient motivation as factors that they would have to consider before offering a psychological intervention.

Finally, working on this project has furthered my experience in every stage of the research process; from the early conceptualisation of an idea, to considerations of study design, to statistical analysis and eventual write up. Throughout this process, I have learned that each stage of the research is an iterative process, best facilitated by an experienced supervisor who can explore ideas and scaffold the learning process. I used each consultation to reflect on issues as they arose and enhance my research skills by responding to feedback and applying the knowledge gained. The skills I have learned have allowed me to become a more competent practitioner and will allow me to become involved in future projects which can contribute to evidence-based practice.

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Appendix A: UCL Ethical Approval Letter

UCL RESEARCH ETHICS COMMITTEE

OFFICE FOR THE VICE PROVOST RESEARCH



12th July 2019

Dr Liam Mason

Research Department of Clinical, Educational and Health Psychology

UCL

Cc: Liam McAuliffe

Dear Dr Mason

Notification of Ethics Approval with Provisos

<u>Project ID/Title: 15721/001: Causal explanations of mental illness and perceived credibility of psychological therapy</u>

Further to your satisfactory responses to my comments, I am pleased to confirm in my capacity as Chair of the UCL Research Ethics Committee (REC) that I have ethically approved your study until 12th July 2020.

Ethical approval is subject to the following conditions:

Notification of Amendments to the Research

You must seek Chair's approval for proposed amendments (to include extensions to the duration of the project) to the research for which this approval has been given. Each research project is reviewed separately and if there are significant changes to the research protocol you should seek confirmation of continued ethical approval by completing an 'Amendment Approval Request Form' http://ethics.grad.ucl.ac.uk/responsibilities.php

Adverse Event Reporting – Serious and Non-Serious

It is your responsibility to report to the Committee any unanticipated problems or adverse events involving risks to participants or others. The Ethics Committee should be notified of all serious adverse events via the Ethics Committee Administrator (ethics@ucl.ac.uk) immediately the incident occurs. Where the adverse incident is unexpected and serious, the Joint Chairs will decide whether the study should be terminated pending the opinion of an independent expert. For non-serious adverse events the Joint Chairs of the Ethics Committee should again be notified via the Ethics Committee Administrator within ten days of the incident occurring and provide a full written report that should include any amendments to the participant information sheet and study protocol. The Joint Chairs will confirm that the incident is non-serious and report to the Committee at the next meeting. The final view of the Committee will be communicated to you.

Final Report

At the end of the data collection element of your research we ask that you submit a very brief report (1-2 paragraphs will suffice) which includes in particular issues relating to the ethical implications of the research i.e. issues obtaining consent, participants withdrawing from the research, confidentiality, protection of participants from physical and mental harm etc.

Office of the Vice Provost Research, 2 Taviton Street

University College London

Tel: +44 (0)20 7679 8717 Email: ethics@ucl.ac.uk

http://ethics.grad.ucl.ac.uk/

In addition, please:

ensure that you follow all relevant guidance as laid out in UCL's Code of Conduct for Research: https://www.ucl.ac.uk/srs/file/579

note that you are required to adhere to all research data/records management and storage procedures agreed as part of your application. This will be expected even after completion of the study.

With best wishes for the research.

Yours sincerely



Professor Lynn Ang

Joint Chair, UCL Research Ethics Committee

Appendix B: Participant Information Sheet

Please save or print this information sheet if you would like to keep a copy. Alternatively, you could contact the research team to request a copy.

Title of Project: The impact of different explanations of mental health difficulties

UCL Research Ethics Committee Approval ID Number: 15721/001

Department: Research Department of Clinical Educational and Health Psychology

Name and Contact Details of the Researcher: Liam Mc Auliffe ucjulmm@ucl.ac.uk

Name and Contact Details of the Principal Investigator: Dr. Liam Mason, I.mason@ucl.ac.uk

We are inviting you to take part in a research project. We want to find out about the impact of explaining mental health difficulties in different ways; specifically attitudes toward different kinds of available treatments.

Before you decide whether to take part it is important that you understand why the research is being done and what this study will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Contact details are provided above.

What is the purpose of this study?

The study aims to investigate the ways in which we explain a mental health difficulty can affect our impression of a person who is affected. We are also interested in how people make use of evidence about mental health interventions.

Why have I been invited?

You have been invited to participate in this study as you are a clinician with a good understanding of English.

Do I have to take part?

No. You are under no obligation to take part in this study.

What will I be asked to do?

Your participation will involve answering a few questions about yourself such as age, gender and a questionnaire about your beliefs and attitudes. You will then be presented with a paragraph describing a person with mental health difficulties and asked to complete a few more questions which measure your reaction to that information. You will then be presented with more information about psychological treatment and be asked to complete a final few questions. The study will last approximately **5 MINUTES.**

If you decide to take part, you are still free to withdraw at any time during the process and without giving a reason.

What are the benefits of participating in this study?

If you take part, you will be contributing to a project which is designed to help researchers understand the implications for how we describe mental illness, thereby hopefully benefiting those affected in future.

In addition, you will have the opportunity to enter a prize draw for a £100 gift voucher or to donate this money to a charity of your choice.

What are the risks of participating in this study?

We do not envisage any risks of taking part in the study. Details will be provided in the debrief for obtaining more information should you find any of the issues in this study distressing. You can also contact the researchers (details below) for further information.

What if I no longer want to take part in this study?

You can stop taking part in this study at any time and without giving a reason. However, if you have completed the entire study, research data that we have already collected cannot be withdrawn or recalled as it is a fully anonymous study.

How will my information be used?

To help future research and make the best use of the research data you have given us (such as answers to questionnaires) we may keep your research data indefinitely.

The data we collect may be shared as follows:

- In research publications, your research data will usually be reported as part of an average of the group of people being studied, so you cannot be identified as an individual. If any of your individual data are reported, they will be published anonymously with your personal details completely removed.
- We may share your research data in public research databases, but your data will always be anonymised. This means that a code will be used instead of your name (or other personal details), and protections applied that minimize the risk of deliberate or accidental reidentification of you as an individual.
- Personal data is any information that might identify you as an individual. Your personal data will be kept securely and will only be kept as long as it is necessary for the research. It will be deleted if it is no longer required.
- Your contact details are part of your personal data. Contact details will be held securely and will never be shared except with the researchers conducting this research and other authorised persons working with the study team.
- We may share your research data with other accredited researchers, and this may include personal data necessary for the research, such as your date of birth so that your age is known for certain analyses.
- The legal basis used to process your personal data is known as the provision of public task. This means that the research you are taking part in is deemed to be in the public interest. We will follow UCL and legal guidelines to safeguard your data.

Who is the Sponsor for this Study?

University College London (UCL) is the sponsor for this study based in the United Kingdom. We will be using information from you in order to undertake this study and UCL will act as the data controller for this study. This means that we are responsible for looking after your information and using it properly.

How will my information be used on research databases?

When you agree to take part in a research study, anonymised data that does not contain any personally identifiable information may be made openly available. Your information will only be used by organisations and researchers to conduct research in accordance with the UK Policy Framework for Health and Social Care Research.

Who is organising the funding of this study?

The study is funded by UCL's Research Department of Clinical, Educational and Health Psychology. The student researcher will be liaising with UCL to organise funding for the study

Who has reviewed the study?

This study has been reviewed by the UCL ethics committee on 12/07/19

Local Data Protection Privacy Notice

The controller for this project will be University College London (UCL). The UCL Data Protection Officer provides oversight of UCL activities involving the processing of personal data and can be contacted at **data-protection@ucl.ac.uk**. This 'local' privacy notice sets out the information that applies to this particular study. Further information on how UCL uses participant information can be found in our 'general' privacy notice:

For participants in health and care research studies, click <u>here</u>

What if there is a problem?

If you wish to complain or have any concerns about any aspect of the way the information has been gathered whilst participating in the research, then please talk to the researcher or the chief investigator (contact details below) about your complaint. If you then feel that the complaint has not been resolved satisfactorily, please contact the chair of the UCL Research Ethics Committee (ethics@ucl.ac.uk).

If you are concerned about how your personal data are being processed, please contact UCL data protection officer via protection@ucl.ac.uk. If you are not satisfied with the response you receive, you may wish to contact the Information Commissioner's Office (ICO). Contact details, and details of your rights, are available on the ICO website at: <u>Link</u>

Who do I contact for further information?

If you would like any further information about this study, please contact us by email: Liam Mc Auliffe: ucjulmm@ucl.ac.uk Liam Mason: l.mason@ucl.ac.uk If you would like a copy of this information sheet, please request via email.

Thank you for reading this information sheet and for considering taking part in this research study.

Appendix C: Consent Form

Project Title: The impact of different explanations of mental health difficulties

Please complete this Consent Form after you have read the Information Sheet and had the opportunity to speak to the researcher if you wish to. If you need any further information to help you decide whether or not to take part, then please speak to the researcher before completing this form.
I confirm that I understand that by ticking each box below I am consenting to this element of the study. I understand that it will be assumed that unticked/initialled boxes means that I DO NOT consent to that part of the study. I understand that by not giving consent for any one element that I may be deemed ineligible for the study.
I confirm that I have read and understand the Information Sheet (previous web page). I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.
I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason. However, if I have completed the entire study, research data that has already been collected cannot be withdrawn or recalled as it is a fully anonymous study.
I consent to the processing of my personal data for the purposes explained to me in the information sheet. I understand that my information will be handled in accordance with all applicable data protection legislation and ethical standards in research.
I understand that it will not be possible for others to identify me in any publications.
I understand that by agreeing to take part in this study, anonymised data that does not contain any personally identifiable information may be shared with others for future research, shared in public databases and in scientific reports.
I understand the potential benefits and risks of participating and who to contact if I wish to lodge a complaint.
I voluntarily agree to take part in the above study.
PLEASE PRESS NEXT TO ACCEPT THESE TERMS

Appendix D: Patient Vignette

Nicholas is an 18-year-old student who is seeking treatment at the urging of his family. His parents became worried when he began talking about mysterious "spies" who he believes are controlling his thoughts.

More recently, he has been claiming that he can hear conversations between the "spies" using special powers that allow him to pick up radio signals through his ears. His family has also noticed that he has become more withdrawn socially and does not seem to enjoy anything he does anymore.

His voice has even taken on a dull, monotonous quality. Nicholas' teachers report that his grades have been slipping drastically this year, as he seems to have extreme difficulty concentrating or even making sense when he speaks.

Appendix E: Predominantly Biological Explanation

According to his medical records, Nicholas experienced a hypoxic birth, which means he did not receive enough oxygen to his brain during birth.

Genetic testing revealed that Nicholas carries a variation in the gene known as neuregulin 1 (NRG1); this finding is common in people who experience hallucinations and delusions.

The atmosphere in Nicholas' home was highly emotional and extremely stressful. Nicholas constantly felt anxious and stressed, wondering what the next family conflict would entail.

In Nicholas' medical records, a neurologist noted that there is evidence of dysfunction in his neurotransmitter systems, including dopamine abnormalities.

Recent tests revealed reduced temporal lobe volume, an area of the brain which plays an important role in processing emotions and language.

The tests also found increased neuronal packing density, compared with most healthy people, particularly in Nicolas' dorsolateral prefrontal cortex (a brain area highly involved in attention and inhibition)

Appendix F: Predominantly Psychosocial Explanation

Nicholas grew up in a poor, inner-city neighbourhood.

He and his family live in a very small crowded apartment in a poorly maintained building.

As a child, he was often physically and verbally abused by his parents.

His father was an alcoholic and regularly beat Nicholas during his frequent drunken episodes.

Nicholas's mother constantly criticised him and pointed out what she perceived to be his many flaws as a son.

Genetic testing revealed that Nicholas carries a variation in the gene known as neuregulin 1 (NRG1); this finding is common in patients who experience hallucinations and delusions.

In general, the atmosphere in the home was highly emotional and extremely stressful. Nicholas constantly felt anxious and stressed, wondering what the next family conflict would entail.

Appendix G: Treatment Information Introduction

You are now going to read about a psychological intervention which is available to help somebody with symptoms like Nicholas'. Keeping the vignette in mind, please read the below information carefully and then answer the questions which follow.

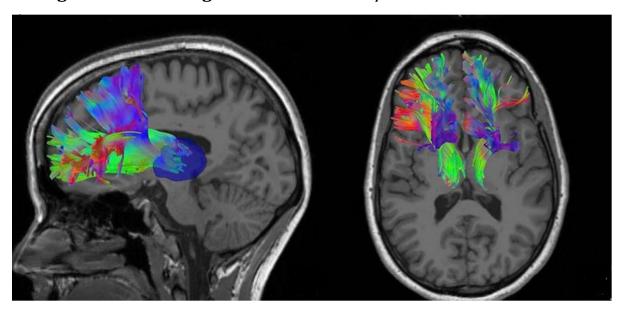
Cognitive behaviour therapy (CBT) is a talking therapy that involves patients changing the way they think about and respond to their thoughts and experiences.

For patients experiencing symptoms of psychosis (like Nicholas), CBT involves learning to think differently about their unusual experiences, such as distressing beliefs that others are out to get them.

For example, CBT would involve helping patients to recognise delusions (ideas that are not true) and hallucinations (hearing or seeing things that no one else hears or sees) when they are occurring and manage the stressful situations in which they occur. The goal is to make them less distressing and impairing in day to day life.

Appendix H: Treatment Information - CBTp-led Neuroplasticity

Brain scan showing connectivity changes. Specific pathways that strengthened following CBT shown in blue/red.



Several recent studies have shown that cognitive behaviour therapy (CBT), a specific kind of psychological therapy, can bring about lasting changes in the brains of patients with psychosis.

In one study, the brains of patients with psychosis were scanned before and after a course of CBT. The scans showed strengthened connections between several brain regions after CBT.

Most important for improvement were connections between the amygdala (sometimes called the brain's "threat centre") and the frontal lobes (which are involved in thinking and reasoning). These brain changes were not seen in a control group of patients who did not receive CBT.

In another study, the strengthening in these brain connections was key for sustained improvement in symptoms and recovery several years after patients completed therapy.

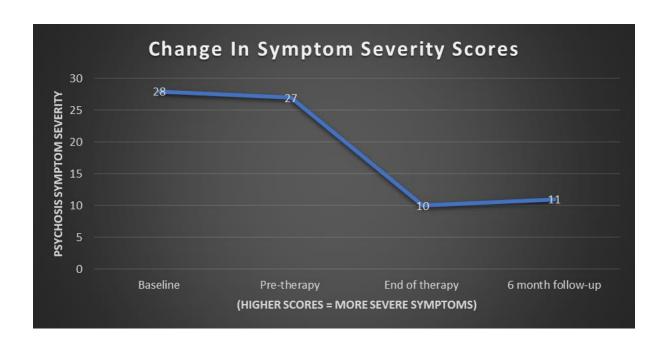
Appendix I: Treatment Information – Generic Effectiveness Information

Results from [a single] Randomised Controlled Trial using CBT for Psychosis

A number of research studies have shown that CBT is a helpful psychological therapy for psychosis. This means that many patients benefit from improvements in functioning and reduced distress from their psychotic symptoms.

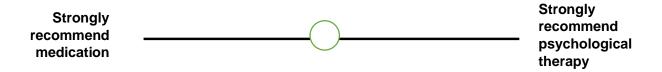
In addition to medication, CBT is one of several other treatments recommended by the National Institute for Health and Care Excellence (NICE, 2013).

Some studies also show that CBT can continue to benefit patient's longterm recovery several years after therapy has been completed.



Appendix J: Treatment Recommendation, Psychological Therapy and Medication Effectiveness Ratings

Considering the above information, what would you recommend; medication or psychological therapy, to treat Nicholas' difficulties?



To what extent do you believe Nicholas' symptoms could improve with psychological therapy?

Not								-
at all	1	2	3	4	5	6	7	Completely

To what extent do you believe Nicholas' symptoms could improve with medication?

Not								1
at all	1	2	3	4	5	6	7	Completely

Appendix K: Illness Perception Questionnaire (Modified)

Please read each statement and decide how much you agree or disagree with that statement.

that statemen	nt.			
1. A person's	state of mind plays	a major part in ca	using their men	tal health problems
Strongly disagree	Disagree	Undecided	Agree	Strongly agree
2. A person's	mental health prob	lems will last a sh	ort time.	
Strongly disagree	Disagree	Undecided	Agree	Strongly agree
3. There is ver	ry little that can be	done to improve a	person's menta	l health problems.
Strongly disagree	Disagree	Undecided	Agree	Strongly agree
4. What a pers	son does can deter	mine whether their	mental health p	roblems get better or
Strongly disagree	Disagree	Undecided	Agree	Strongly agree
5. Talking the	rapy will be effectiv	ve in improving a p	erson's mental l	nealth problems.
Strongly disagree	Disagree	Undecided	Agree	Strongly agree
6. A person's	mental health prob	lems can improve.		
Strongly disagree	Disagree	Undecided	Agree	Strongly agree
7. Something problems	about a person's p	ersonality plays a	role in causing t	heir mental health
Strongly disagree	Disagree	Undecided	Agree	Strongly agree
8. A person's	mental health prob	lems will improve	in time.	
Strongly disagree	Disagree	Undecided	Agree	Strongly agree

Strongly disagree	Disagree	Undecided	Agree	Strongly agree
l0. Changing nealth probler		hinks or the way th	ey do things car	n improve their men
Strongly disagree	Disagree	Undecided	Agree	Strongly agree
1. Recovery thance.	rom a person's me	ental health probler	ns is largely dep	pendent on fate or
011	Disagree	Undecided	Agree	Strongly agree
Strongly disagree	Disagree			
disagree 12. A person's Strongly		blems will last for a	a long time. Agree	Strongly agree
disagree 12. A person's Strongly disagree	b mental health pro		Agree	
disagree 12. A person's Strongly disagree 13. There is a Strongly disagree	Disagree lot a person can de	o to improve their n	Agree	oblems.

Appendix L: Political Orientation

How would you describe your political orientation?



Appendix M: Dogmatic Beliefs

Please read each statement and decide how much you agree or disagree with that statement.

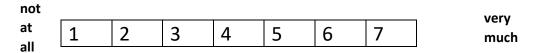
1. Anyone who	is ho	onest	ly and	d truly	/ seel	king t	he tru	ıth wi	ll end ı	up believing what I believe.
Disagree										Agree
completely	1	2	3	4	5	6	7	8	9	Completely
2. There are so many things we have not discovered yet, nobody should be certain his beliefs are absolutely right.										
Disagree										Agree
completely	1	2	3	4	5	6	7	8	9	Completely
3. The things I	belie	ve in	are s	o con	plete	ely tru	e, I co	ould r	never o	loubt them.
Disagree							1	1		Agree
completely	1	2	3	4	5	6	7	8	9	Completely
4. I have never	disc	overe	d a s	ystem	of be	eliefs	that e	explai	ins eve	erything to my satisfaction.
Disagree			_	_	_	_		_		Agree
completely	1	2	3	4	5	6	7	8	9	completely
5. It is best to	be op	en to	all po	ossibi	lities	and r	eady	to re-	-evalua	ate all your beliefs.
Disagree			_	_	_	_		_		Agree
completely	1	2	3	4	5	6	7	8	9	completely
6. My opinions	are r	ight a	and w	ill sta	nd th	e test	of tir	ne.		
Disagree			_	_	_	_		_		Agree
completely	1	2	3	4	5	6	7	8	9	completely
7. Flexibility is	a rea	l virtu	ue in 1	thinki	ng, s	ince y	ou m	ay we	ell be v	vrong.
Disagree			_	_	_	_		_		Agree
completely	1	2	3	4	5	6	7	8	9	completely
8. My opinions	and	belief	s fit p	erfec	tly to	gethe	er to n	nake	a cryst	al-clear "picture" of things
Disagree		_			_	_	_ 1	_		Agree
completely	1	2	3	4	5	6	7	8	9	completely
9. There are no discoveries or facts that could possibly make me change my mind about the things that matter most in my life.										
Disagree										Agree
completely	1	2	3	4	5	6	7	8	9	completely
10. I am a long	way	from	reach	ning c	onclu	isions	s abo	ut the	centra	al issues in life.

Disagree		Ι.	12	T 4	T =	1.6	—	T 0		Agree
completely	1	2	3	4	5	6	7	8	9	completely
11. The person who is absolutely certain she has the truth will probably never find it.								probably never find it.		
Disagree			_	1	1	1		1		Agree
completely	1	2	3	4	5	6	7	8	9	completely
12. I am abso	lutely	certa	in tha	at my	ideas	s abo	ut the	fund	lamenta	al issues in life are correct.
Disagree			T _	1 -	T_	1 -	1_	1 _	T - 1	Agree
completely	1	2	3	4	5	6	7	8	9	completely
13. The peopl	e who	disa	gree v	with r	ne m	ay we	ell tur	n out	to be r	ight.
Disagree		1_	-	1 -		1 _		1_	1-	Agree
completely	1	2	3	4	5	6	7	8	9	completely
14. I am so su could convinc		_			e imp	ortar	t thir	ngs in	life, th	ere is no evidence that
Disagree				1	1					Agree
completely	1	2	3	4	5	6	7	8	9	completely
15. If you are reach the wro	-				the I	most	impo	rtant	things	in life, you will probably
Disagree			,	1	1	1	1	1		Agree
completely	1	2	3	4	5	6	7	8	9	completely
16. Twenty ye probably have			ow, s	ome d	of my	opin	ions a	about	the im	portant things in life will
Disagree				1	1	1		1		Agree
completely	1	2	3	4	5	6	7	8	9	completely
17. "Flexibility	in th	inkin	g" is	anoth	er na	ame fo	or "wi	ishy-v	washy"	
Disagree				1	1	1		1		Agree
completely	1	2	3	4	5	6	7	8	9	completely
18. No one knows the essential truths about the central issues in life.										
Disagree		T _		1 -	T _	1 -	T	1 _		Agree
completely	1	2	3	4	5	6	7	8	9	completely
19. Someday I will probably realise my present ideas about the BIG issues are wrong										
Disagree				1	1	1		1		Agree
completely	1	2	3	4	5	6	7	8	9	1 . 1
20. People who disagree with me are just plain wrong and often evil as well.								I.		completely
20. People wh	o dis	agree	1	1	re jus	st plai	n wro	-1		
20. People who	o dis	agree	1	1	re jus	st plai	n wro	-1		

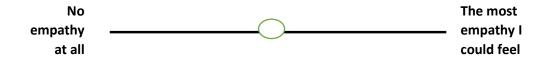
Appendix N: Empathy Ratings

Please indicate how much each adjective describes your feelings whilst reading Nicholas' story.

Compassionate



Please indicate how much empathy you feel for Nicholas on the scale below:



Appendix O: Participant Debrief Sheet

Please save or print this information sheet if you would like to keep a copy. Alternatively, you could contact the research team to request a copy.

PLEASE CLICK 'NEXT' TO SUBMIT YOUR ANSWERS...

Study: The Impact of Different Explanations of Mental Health Difficulties

UCL Research Ethics Committee Approval ID Number: CEHP/15721/001

Department: Research Department of Clinical Educational and Health Psychology

Name and Contact Details of the Researcher: Liam Mc Auliffe ucjulmm@ucl.ac.uk

Name and Contact Details of the Principal Investigators: Dr. Liam Mason, I.mason@ucl.ac.uk

Thank you for your participation in our study.

The aim of this study was to investigate the impact of causal explanations of mental illness. Previous studies have shown that a description of mental illness in biological (vs psychosocial) terms has many implications. When participants read a description of a patient containing a predominantly biological explanation of their difficulties, they feel less empathy for the person and are biased towards offering medication rather than psychological therapy to treat them.

We would like to find out if people are given information that psychological therapies, like medication, also lead to neurobiological changes, they might be more likely to recommend psychological therapy to treat a patient. If so, this has important implications for how mental illness is described.

If you have any further questions, or you feel you've been adversely affected by taking part in the study, please feel free to contact the research team using the contact information below:

Principal investigator: Dr. Liam Mason, I.mason@ucl.ac.uk

Researcher: Liam Mc Auliffe, liam.auliffe.15@ucl.ac.uk

All data is anonymous, however, if you no longer wish to submit your answers please slide the circle across to 'withdraw my answers' and click 'next'. If you are happy to submit your answers, please keep the circle where it is and click 'next'.

Happy to	Please
submit	withdraw
my	 my
answers	answers