Beliefs about the causes of depression

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Thesis submitted in partial fulfilment of the requirements of Staffordshire and Keele Universities for the jointly awarded degree of Doctorate in Clinical Psychology

April 2016

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Acknowledgements

I would like to thank my thesis supervisor, Dr Helen Scott, for all your effort and perseverance in this research process.

I would like to thank my cohort for all your support and friendship over the past three years. Lisa and Abbye, your whatsapps have cheered me along during these last weeks and I am so glad that we had each other.

To my family, thank you for your encouragement and help over the course of this Doctorate. Especially for helping me with my car, without which I never would have made it anywhere.

But thanks go especially to Mike. Not only for bringing me food and letting me use your computer for months on end, but for your endless patience, kindness and faith in me. I couldn't have done it without you.

Candidate Declaration Form

CANDIDATE DECLARATION

Title of degree programme	Doctorate in Clinical Psychology
Candidate name	**Stephanie Davies**
Registration number	**10030776**
Initial date of registration	**September 2013**

Declaration and signature of candidate
I confirm that the thesis submitted is the outcome of work that I have undertaken during my programme of study, and except where explicitly stated, it is all my own work.
I confirm that the decision to submit this thesis is my own.
I confirm that except where explicitly stated, the work has not been submitted for another academic award.
I confirm that the work has been conducted ethically and that I have maintained the anonymity of research participants at all times within the thesis.
Signed: Date:

Abstract

This thesis consists of three papers: a literature review, an empirical paper and a reflective account. The literature review was conducted to examine the associations between causal beliefs in depression and preferences for and attitudes towards different interventions. Ten papers were included which all used clinical samples. Papers were critiqued for quality, and findings suggest that belief in the biological model is associated with a preference for and more favourable attitudes towards biological interventions such as medication. The association between causal beliefs and preferences for different psychological interventions is less clear, but findings suggest that people prefer therapeutic modalities that 'match' their causal beliefs.

The empirical paper investigated predictors of self-stigma and prognostic pessimism in 184 people experiencing depression. It was hypothesised that depression severity, self-efficacy and biological causal beliefs would be significant predictors in a regression analysis. However, only depression severity and self-efficacy predicted self-stigma, and there were no significant predictors found of prognostic pessimism. The data for prognostic pessimism violated normality. Due to this violation and because the sample was mainly White British women, generalisability of findings is limited.

The last paper contains the authors reflections on the research process, with research decisions outlined and critiqued, including topic choice, participants, data collection and measure choice. It focuses not only on the difficulties but also the positive aspects of the research process. Reflections are considered in relation to the authors epistemological position, and to the more over-arching issues that arise when conducting research in clinical psychology.

Word count: 19221

Paper 1: Literature Review

How do causal beliefs in depression affect preferences for and attitudes towards different interventions?

For 'Psychotherapy Research" (no word count)

8848 words (not including figures or tables as per journal stipulations)

10520 words (including tables and figures as per award requirements)

Contents

Abstract

Objective

The aim of this literature review was to examine and summarise research investigating the relationship between causal beliefs in depression and attitudes and preferences towards interventions in people experiencing depression

Method

A systematic approach was used to identify relevant research from the following databases in September 2015; PSYCHInfo, CINAHL, MEDLine, AMED, Web of Science, PSYCHArticles and Cochrane Library.

Results

Ten relevant papers were identified and are included in this review, all of which used quantitative methods. All of the papers investigated preferences, perceptions or attitudes towards interventions[1]

Conclusion

The papers examined suggest there is evidence for a positive relationship between endorsing biological causal beliefs and preference for medication. Evidence suggests that people prefer psychological interventions that 'match' their causal beliefs. More research is needed on causal beliefs and preferences for different therapeutic modalities in more naturalistic settings. Future experimental research could investigate the relationship between information giving about causes of depression and intervention preferences.

Clinically, the research suggests clinicians should address causal beliefs in assessment sessions with participants in order to match facets of therapy to these beliefs. For psychologists, ethical guidelines suggest they have a role in challenging biological explanations for depression in clinical settings.

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[1] The word 'intervention' rather than 'treatment' is mainly used in this review as it is considered a more encompassing term for medical and psychological management of depression. 'Causal beliefs' and 'causal attributions' are used interchangeably but refer to the same concept.

Keywords: Depression, causal beliefs, etiological beliefs, psychotherapy, treatment, intervention.

Introduction

Depression

Depression is a very common mental health problem which is estimated by the World Health Organisation to affect 350 million people worldwide (World Health Organisation [WHO], 2012). In the UK, about one in 20 adults experiences an episode of depression each year (National Institute for Health and Care Excellence [NICE] 2015). People who experience depression typically have persistent low mood and loss of interest in activities as well as a variety of other emotional, cognitive, physical and behavioural symptoms (NICE, 2015) such as fatigue, reduced activity and feeling negative about the future.

Depression is not only associated with an increased risk of suicide (Evans et al., 2004) but also with increased risk of other causes of mortality, such as heart disease (Cuijpers & Smit, 2002). Severe depression can affect all areas of life, including educational attainment, occupational productivity, as well as impacting negatively on family and social life (Cuijpers et al., 2012; Kessler, 2012). Depression not only disrupts an individual's quality of life, it is also the largest cause of 'disease burden' for a non-fatal health problem worldwide (WHO, 2008). 'Disease burden' is the impact of a health problem as measured by financial cost, mortality and morbidity (WHO, 2008).

Although classification systems such as the DSM-V (American Psychiatric Association [APA], 2013) and the ICD-10 (WHO, 2012) distinguish between levels of severity of depression, these systems are not in agreement on the definition of 'clinically significant depression'. Classification systems may differ due to the difficulties in quantifying personal experiences and separating out a 'disorder' from the person and their surrounding as a whole. Due to the diversity in the presentations and experiences of depression, it may be helpful to consider it as a multidimensional problem that occurs on a continuum of severity (Lewinsohn, Solomon, Seeley & Zeiss, 2000), with

greater severity of depression being associated with greater risk of death and greater impact on functioning (Kessing, 2007; Lewinsohn et al., 2000).

The length of time someone might experience depression for is also variable; a global study found that 50% of people still had a diagnosis of depression a year after initial onset (Simon, Goldberg, Von Korff & Ustun, 2002) and at least 10% had persistent depression (Kessler et al., 2003), meaning that their depression was an on-going problem. Half of all people diagnosed with 'major depression' will go on to have another episode (DSM-IV (APA, 2013)), and prior episodes of depression are one of the best predictors of future episodes (Burcusa & Iacono, 2007). Depression, therefore, can be a recurrent or chronic problem for many people.

Historical and current perspectives on the causes of depression

Theoretical models of depression have attempted to link the origins of depression with a conceptual understanding of what depression actually is. For example, if depressive symptoms are thought to be caused by a chemical imbalance within the brain, depression would be thought of as a physical illness like diabetes or epilepsy (Schnittker, 2008). Considering current causal explanations is important for understanding how depression is conceptualised and treated.

The biopsychosocial model (Engel, 1978) of mental health problems has been rising in prominence in the West since the 1980s and is the dominant ideology of mainstream psychiatry today (Ghaemi, 2009). It encourages clinicians to take a holistic view of the person's distress and consider three aspects in the management of their depression: biological, psychological and social. This biopsychosocial model was developed in response to the purely biomedical view of illness and health that developed during the evolution of medical science in the early twentieth century (Hatala, 2012). It was deemed to be more useful to clinicians and clients as it would be more representative of the complex reality of being a human (Ghaemi, 2009). The model conceptualises these three areas as separate parts of a whole that interact and influence each other, and are linked to 'mind-body' dualism; the idea that the physical being and the mind are separate but

linked. It was hoped that this model would lead to further consideration of the impacts of contexts, personal histories and relationships on mental health (Engel, 1977).

Despite Engels' (1977) assertion that all three aspects need to be considered equally, the lack of evidence supporting one area over another can lead to prioritisation dependent on personal viewpoints (Ghaemi, 2009). The model also originated as a framework for clinicians to understand the different dimensions of an 'illness', a terminology which in itself suggests a medical perspective (Engel, 1977). Indeed, the model gave rise to the 'vulnerability-stress' idea; that although stress plays a role in the development of a mental health problem, a primary causal factor is an underlying biological (be it genetic or chemical) vulnerability that makes the person more susceptible to its development (Zubin & Spring, 1977). Currently, whether there is any evidence for this vulnerability is still debated (Hindmarch, 2002; Moncrieff, 2007). The British Psychological Society (BPS) advises its clinicians that assuming this underlying vulnerability is a leading cause of a mental health problems not only undermines the personal meaning of events but also assumes that these problems cannot be legitimate responses to stress (BPS, 2011). However, the BPS does support the consideration of biological factors when thinking about a client's problems (BPS, 2011).

Research into the biological causes of mental health problems is a large area of study. In October 2015, it was announced that University College Los Angeles would be spending $525 million on research into depression, primarily investigating its genetic, molecular and chemical causes (Sullivan, 2015). Also, biological models of depression, especially the chemical imbalance theory, have been highly promoted in the US (Leo & Lacasse, 2008) and became the dominant construction of depression in media articles about depression in the 2000s (Clarke & Gawley, 2009). By 2006, a survey found that over 67% of the US population endorsed the chemical imbalance theory of depression, whilst over half endorsed a genetic cause (Pescosolido et al., 2010).

The US and New Zealand are the only Western countries where companies that develop and sell prescription drugs are allowed to directly market their products to consumers, and they heavily rely on the chemical imbalance theory of depression to promote their products (Grow, Park & Han, 2006). Time trend analyses conducted in Australia and Germany have also reflected an increase in the endorsement of the biological model (Angermeyer, Holzinger, & Matschinger, 2009; Jorm, 2005), despite this lack of direct advertising from drug companies. A 2008 cross-sectional study (Budd, James & Hughes, 2008) undertaken with the general public in the UK found that a 'chemical imbalance in the brain' was rated only behind the bereavement of an immediate family member for perceived importance in the development of depression.

Studies from outside the US (Switzerland, Turkey, UK, and Ireland), show that although people may be increasingly endorsing a biological viewpoint, they still endorse psychosocial causes of depression (such as stress) to a higher degree (Çirakoğlu, Kökdemir & Demirutku, 2003; Furnham & Kuyken, 1991; Lauber, Falcato, Nordt, & Rossler, 2003; McKeon & Carrick, 1991). A systematic review of the research conducted in 2014 (Hagmayer & Engelmann, 2014) found that in western countries, 'stress due to environmental factors' was considered to be the most endorsed cause of depression, followed by psychological causes, then biological causes.

An alternative way of conceptualising depression is the 'psychosocial' viewpoint; that the presentation of depression is indistinguishable from normal reactions to the realities of life (Conrad, 2008). For example, the DSM-V (APA, 2013) removed the 'bereavement clause' from the diagnosis of depression which was in the previous editions. This clause prevented people who had suffered a loss from being classified as 'clinically depressed'. However, its removal from the DSM- 5 (APA, 2013) means that grief reactions can now be classified as depressive disorder, treatable with medication. It can be argued that classifying human response to loss as a 'disorder' suggests that it is abnormal, rather than a normal and necessary process. Critics of the biological model would also argue that the high rates of depression reflect the medicalisation of the understandable distress

caused by the social, political and economic problems in much of the world (Kleinman, 1987). For example, the higher rates of suicide during the recent recession (Reeves, McKee & Stuckler, 2014) suggest that factors such as unemployment, poverty and cuts to benefits play a significant role in these tragedies. Researchers who focus on social inequalities as a cause of depression may have trouble making their case heard in a climate where the importance of genetics are amplified by the media (Conrad, 2001). Researchers also reason that the large variety of presentations or comorbidities with other mental health problems indicates that it cannot be thought of as a classifiable disorder because it is so rarely the same from one person to another (Bentall, 2010; Brown, Campbell, Lehman, Grisham & Mancill, 2001).

Current interventions for depression

Interventions for depression reflect the changing views on the causes of depression. As the prevalence of biological causal beliefs has increased, prescriptions for antidepressants have also increased. In England, prescribing rates of anti-depressants have increased by 165% since 1998 (The Health Foundation, 2014), and most clinical guidelines now recommend intervention with medication. The APA recommends either antidepressant medication alone or a combination of antidepressants and psychotherapy for all types of depression, but only 'depression focused psychotherapy' if the depression is 'mild or moderate' (APA, 2010). This indicates that more severe depression is seen as treatable only with biological interventions.

In the UK, recommendations take a stepped care approach, suggesting self-help or low intensity Cognitive Behavioural Therapy for mild depression, with increasing intensity of psychological intervention as the chronicity or severity of depression increases (NICE, 2010). In contrast to the US, anti-depressants are only recommended for cases of moderate to severe depression (NICE, 2010). Both UK and US guidelines base their definitions and thresholds for severity on the DSM-V (APA, 2013) criteria. These different recommendations might therefore be based more on professional or societal beliefs about causation rather than evidence.

Despite recommendations for psychotherapeutic interventions as well as medication, many people with depression only receive medication (The Kings Fund, 2008). An estimate made by the Kings Fund (2008) was that of those people seen by NHS services in England diagnosed with moderate to severe depression, 30% were receiving only medication, 27% were receiving medication and therapy, and only 8% were receiving just psychological therapy. This is despite evidence showing that people consistently prefer psychological interventions over pharmacological ones (Churchill et al., 2000; Deacon & Abramowitz, 2005; Riedel-Heller, Matschinger & Angermeyer, 2005). In the US, the over-reliance on medication and the lack of emphasis on psychotherapy by psychiatrists has been raised as a concern by the president of the APA (APA, 2005). The efficacy of antidepressants over placebos is a disputed topic in the psychiatry community (Kirsch, 2014; Moncrieff, 2008), with questions being raised about their effectiveness in helping people recover from depression. In addition to this, the rates of adherence to medication remain very low (Hunot et al., 2007), suggesting that a significant number of people have difficulty engaging with an entirely bio-medical approach to treating their depression.

Cognitive Behavioural Therapy (CBT) has good evidence for efficacy in treating depression and is recommended by NICE guidelines (NICE, 2010). Other types of psychotherapy are also deemed appropriate for use with people experiencing depression, including more psycho-dynamically, behaviourally or interpersonally orientated therapies (National Collaborating Centre for Mental Health, 2010). A recent meta-analysis (Barth et al., 2013) of 198 randomised controlled trials found 'robust' evidence (moderate to large effect sizes between groups) for the efficacy of CBT, interpersonal therapy and problem solving therapy compared to waitlist controls in reducing symptoms of depression. They also found significant effects compared to waitlist controls for psychodynamic therapy, behavioural activation and social skills therapy in the management of depression.

Causal beliefs and their relationship to psychotherapy for depression

It is important to look at the association between causal beliefs and attitudes towards psychotherapy for a number of reasons. Client preference is a part of best practice standards in deciding intervention (APA, 2000; BPS, 2008) so potential influences on preferences are important to investigate. Research has also shown that having positive expectations for therapy are vital for its success (Constantino et al., 2011; Greenberg, Constantino & Bruce, 2006). So if causal beliefs influence people's preferences, belief in the chemical imbalance or other biological theories of depression may reduce people's preference for psychotherapy, thus reducing its use or potential effectiveness. Client preference in terms of intervention can influence important factors such as therapeutic alliance, participation in therapy and the success of the therapy (Iacoveilo et al., 2007; Thornett, 2001: van Schaik et al., 2004). Swift and Callahan (2009), carried out a meta-analysis of the research on client preferences and outcomes, and found that clients who received their preferred therapeutic intervention (each study offered different types of interventions) had a 58% chance of showing greater improvement than clients who did not receive their preference. They were also 50% less likely to drop out if the intervention they received matched their preference. Congruency between causal beliefs and intervention or therapy modality offered could, therefore, improve outcomes.

Beliefs also influence public sentiment about causes and intervention for depression, which provides the context within which decisions are made about intervention (Strecher & Rosenstock, 1997). Mental health professionals need to be able to contribute effectively to this public discussion about appropriate intervention for depression in order to increase opportunity for people to receive appropriate psychological therapies; understanding the relationship between causal beliefs and beliefs about interventions for depression can help inform their messages.

Endorsement of biological causal beliefs has been shown to have beneficial effects; one study found that it can reduce the stigma felt by those with depression (Schreiber & Hartrick, 2002). Other research has found negative implications for people living with depression. Lebowitz, Ahn and Nolen- Hoeksema (2013) found that endorsement of biological causal beliefs

was significantly associated with the belief that depression would have a longer duration, which may be associated with poorer therapy outcomes. There is concern that the increased endorsement of biological causes may increase a sense of fatalism and reduce belief in the possibility of change, thus affecting therapeutic outcomes (Nelkin & Lindee, 1995). Again, this may have important implications for people when choosing intervention options, and may even impact upon engagement in therapy.

The aim of this literature review is to combine and summarise research investigating the relationship between causal attributions in depression with preferences for and attitudes towards interventions. Although there are many different interventions for depression, this research will focus on psychological therapy and medication, as they are the recommended interventions and those used by mental health services in the UK (NICE, 2010). As preferences and beliefs impact on intervention outcomes, they are an important area to investigate (Swift and Callahan, 2009).

Method

Search Strategy

There were two aims of this literature review;

•   To investigate how causal beliefs in depression affect participant's preferences for different psychological or medical interventions;

•   To investigate how causal beliefs in depression affect participant's attitudes towards different psychological or medical interventions.

As medical interventions are not delivered by psychologists, papers that investigate only medical interventions are not considered relevant and are not included in this review.

The following search terms were defined using a PICOC structure based on the following five elements: (1) participants; (2) intervention; (3) comparison; (4) outcome; and (5) context (Petticrew & Roberts, 2006): 'Depression AND (caus* OR etiolo* OR aetiolo*) AND belie* OR model* OR

theor* OR attribut* OR perception*) AND (therap* OR intervention* OR treatment*)'. Advice was sought from an academic librarian in order to finalise the search terms and optimise search methods.

The following databases were searched during September 2015; PSYCHInfo, CINAHL, MEDLine, AMED, Web of Science, PSYCHArticles and Cochrane Library. Search terms were used for PSYCHInfo, CINAHL, MEDLine and AMED and PSYCHArticles. Terms were limited to being found in the title and abstract of the articles. This was due to the very large numbers of non-relevant papers found when searching the entirety of papers using these terms. In order to address publication bias, no date range was specified and non-peer reviewed articles were included.

In the Web of Science search these terms were limited to the title only as there is no option to search both the title and abstract, and more general 'Topic' searching yielded over 16,000 papers.

The Cochrane Library was searched using different terms after no results were found using the above terms. A simpler search using the terms 'Depression AND causal beliefs' was used instead.

This electronic search was then supplemented by hand searching relevant journals and reference and citation checking of selected papers. Review papers were excluded as they are not primary data sources, but were searched for relevant papers.

Study selection

Inclusion criteria

- Papers investigating the relationship between causal beliefs of depression and intervention preferences or attitudes towards intervention, when psychological therapies were included as an intervention
- Participants over the age of 18
- Written in English or translated into English
- Clinical samples of people with depression

Exclusion criteria

- Participants had a co-morbidity, such as a physical or mental health problem

- Papers that focused on post-natal depression

- Papers that investigated causal beliefs and interventions for depression other than psychological therapy or medical interventions, e.g. exercise

- Papers that only investigated causal beliefs and medical interventions

- Review papers were excluded as they are not primary data sources

- Papers had to specifically mention therapy/counselling/seeing a psychologist or use of medication/anti-depressants rather than vaguer professional roles or type of intervention e.g. Mental Health Professional, as it was not clear what type of intervention this role would deliver.

- Papers that investigated causal beliefs and intervention preferences, but did not address the relationship between the two variables.

- Papers that investigated general causal attributions such as 'external' or 'internal'.

The titles and abstracts of all results found with the search terms were reviewed for relevancy. If it was not clear from the abstract whether the paper met the relevant criteria, then the full text of the paper was read before exclusion. Papers with relevant titles and abstracts were then fully read and exclusion criteria applied (see Figure 1).

This search strategy yielded ten studies for review.

Figure 1. Literature Search process flow chart

Cochrane Library N=8	PSYCHInfo N= 2910	PSYCHArticles N= 87	Medline N= 3322	CINAHL N= 374	AMED N= 6	Web of Science N=14

Titles reviewed
N= 6721

Titles excluded for irrelevancy to topic
N= 6666

Abstracts or full articles reviewed   N= 55

Titles excluded N= 45

N= 27 excluded as used a non-clinical sample
N=8 excluded as participants had a co-morbid physical health problem
N=3 qualitative papers excluded for not linking causal beliefs and treatment attitudes or preferences
N=1 excluded for using participants with post-natal depression
N=1 excluded for looking at effects of treatment on causal beliefs
N=1 excluded as treatment option "seeing a Mental Health Professional" too vague
N= 4 removed as duplicates

Titles kept for review
N=10

Titles included N=0
Reviewing citations and references of relevant studies and review papers
Hand searching relevant journals

Selected titles for review
N=10

Results

Study characteristics

Ten papers are included in this review, all of which use quantitative methods. All were from Western countries, six from the US, two from the UK, one from Canada and one from the Netherlands. Studies were conducted in a variety of settings. Five studies looked at causal beliefs and preferences for different types of intervention, medical or psychological. Four of these were observational studies; Dunlop et al. (2012), Houle et al. (2013), Khalsa, McCarthy, Sharpless, Barrett and Barber, (2011) and Schweizer et al. (2010). Two of these studies used participants already involved in Randomised Controlled Trials (Dunlop et al., 2012 and Khalsa, et al., 2011). One study (Steidtmann et al., 2012) investigated intervention preference and outcomes using an RCT design.

Five studies investigated causal beliefs and attitudes towards psychological and medical interventions. Four of these were observational; Budd, James and Hughes (2008), Gaudino, Nowlan, Hughes and Miller (2014), Iselin and Addis (2003), and Meyer and Garcia-Roberts (2007). Kemp, Lickel and Deacon (2014) used an experimental design.

All papers used clinical samples but how 'depression' was defined and measured differed. Over half of the studies used a validated measure, Reasons for Depression Questionnaire (RFD) (Addis, Truax & Jacobson, 1995) as a measurement of causal beliefs. The papers ranged in date from 1996 to 2014. Table 1 is a data extraction table including the studies characteristics, findings, and strengths and weaknesses and quality score based on checklist criteria.

Critical Appraisal

Several sources were drawn upon in order to thoroughly review the methodological quality of each paper: The Critical Appraisal Skills Programme (CASP) checklist which is adapted from guidance by Guyatt, Sackett and Cook (1994), Young and Solomon's (2009) critical appraisal tool, the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) checklist for observational studies (Von Elm et al., 2007) and Downs and Black (1989) quality checklist.

The strengths and limitations of the studies have been summarised below using structure based on these appraisal tools. In addition, an eighteen item checklist was developed using the above tools in order to give each paper a score for quality (see Appendix 1 & 2). Table 1 shows a data extraction table including the studies characteristics, findings, and strengths and weaknesses and quality score based on checklist criteria. Studies achieving 75% or greater were considered high quality and 50%-74% as moderate quality (Crellin, Orrell, McDermott & Charlesworth, 2014).

The most pertinent strengths and weaknesses of the studies reviewed have been summarised below.

Table 1

Summary of Studies Reviewed, including Descriptive and Evaluative Information

Authors and country	Number and demographics of participants	Definition of clinical sample	Study focus	Study type/ method	Findings relevant to review	Main strengths of study	Main limitations of study	Criteria met by critical appraisal tool (%)
Budd, James and Hughes (2008) UK	N=173 62% female 38% male Mean age 45 years	Participants were all members of 'Depression Alliance', a self-help charity. No data collected on diagnosis of depression or current depressive symptoms. However nearly all had at some point received an intervention for depression.	Objective 1. To obtain a more complex and robust factor structure of lay theories of depression Objective 2. To explore the relationship between causal beliefs in depression and perceptions of helpfulness of different interventions received	Observational-cross-sectional Factor analysis Correlations	The belief that depression is caused by 'imbalance in brain biochemistry' was significantly positively correlated with the rated helpfulness of past or current medication taking	Causal belief measure carefully constructed using previous measures, literature and experienced clinician's advice. Causal belief measure had a clear two factor structure and scale had high alpha co-efficient, indicating reliability.	No knowledge of depression symptomology or diagnosis in sample-may lack generalisability to populations with depression Sample had exposure to CBT self-help materials, may not be representative of population with depression Participants asked about past interventions as well as present; possible recall bias Measure did not ask participants what they think caused their own depression- lacks ecological validity	81%

Dunlop et al. (2011) USA	N= 78 57% female,43% male 71% Age:18-60 Mean age 41 Caucasian, 22% Black, 8% Other	Adults with a current diagnosis of Major Depressive Disorder in the DSM-IV and Hamilton Depression Rating Scale of 18 or over at screening	Main objective: to assess whether intervention preferences or causal beliefs are related to outcomes in an RCT. The RCT compared CBT and medication	Observational (cross-sectional) Nominal and logistic regressions	Participants (pts) who endorsed 'out of the blue' cause less likely to prefer CBT. Pts who endorsed 'pessimism' less likely to prefer medication. Pts who endorsed 'emotional illness' more likely to prefer medication. No association between 'brain substances' or 'stress' and intervention preference	Sample accurately defined in terms of depression, multiple measures used. Sample size large enough to find moderate effect Thorough exclusion criteria- clearly defined sample, easier to generalise	Scale assessed a limited number of beliefs, no data on reliability or validity Clients willing to be randomized might not have strong preferences, so results may not be generalizable to population with depression Part of the design may have influenced preferences- participants told that people are equally like to benefit from CBT or medication	77%
Gaudiano, Nowlan, Hughes and Miller (2013) USA	N= 52 58.5% female, 41.5% male Age: >18 92.2% white, 3.8% Hispanic	Psychiatric inpatients with a diagnosis of depressive disorder according to DSM-IV	To examine potential gender differences in hospitalised patients' perceived causes for their depression and their relationship with intervention beliefs and preferences	Observational, cross-sectional Chi-square tests Correlational analysis Moderation analysis	Biological causal beliefs were associated with more positive beliefs about medication use	Used validated measures Good gender balance in sample, generalisable Power calculation indicated sample large enough to detect moderate effect size Naturalistic setting, ecological validity	Not all causal beliefs entered into analysis, limiting knowledge gained Due to sample characteristics, results may not be generalisable to population with depression Intervention did not depend on participants' preferences	90%
Houle et al.	N=88	Participants having a first	Examined	Observational-	Participants	Power calculation	Sample differed	78%

(2013) Canada	46% female, 54% male Age: >18 77% Canadian, 23% other	episode depression as diagnosed by a physician within the previous 8 weeks, having received a prescription for medication or psychotherapy, and a PHq9 score =>10	associations between intervention preferences, participants characteristics and illness representations of depression.	cross-sectional Quantitative measures T-tests and regression analysis	who preferred psychotherapy more strongly endorsed 'social' reasons for depression than participants who preferred medication.	conducted First study to use participants with 'first-episode depression'	from population as high proportion had a University degree- less generalisability. Effect sizes not reported Data on strength of preference not collected No data on length of time depressed- possible confounding variables Causal belief measure used normed on samples with physical illness (IPQ-R, Moss-Morris et al., 2002)	
Iselin and Addis (2003) USA	N=72. 36 clinical and 36 non-clinical participants. In clinical sample: 50% female and 50% male Mean age= 42	50% of participants were students without depression. Other 50% were participants recruited from an outpatient clinic. They had diverse mental health problems. 18/36 of this clinical sample had a diagnosis of depression. Diagnostic criteria was not listed	Effects of etiological information about depression on intervention preferences. Whether effects are similar in consumers on mental health services and non-consumers.	Observational-cross-sectional Repeated measures ANOVA Regression analysis	Participants considered interventions more helpful when cause of depression and focus of intervention were matching e.g. medical cause and medical intervention	Systematic exploration of etiological information impacts intervention helpfulness	Not all participants in the clinical sample had a diagnosis depression- lacks generalisability No validity/reliability information of measures used Vignette study; participants not	75%

							asked about own depression, which means less ecological validity. Analogue study rather than naturalistic; lacks generalisability to real clinical settings Small sample and no power calculation given	
Kemp et al. (2014) USA	N=73 64.4% female,35.6% male Mean age of 20 94.5% Caucasian	Undergraduate students who stated that they had a past or current depressive episode that lasted more than two weeks. No data on depression symptomology or diagnosis collected.	Experiment to examine the impact of chemical imbalance test feedback on perceptions of stigma, prognosis, negative mood regulation expectancies and intervention credibility and expectancy	Experimental design Independents samples Between subjects ANOVA	Participants in experimental condition (informed their depression had a biological basis) rate medication as more effective and credible than psychotherapy. No difference in ratings in control condition (participants informed that their depression was not the results of a chemical imbalance)	First study on chemical imbalance causal attribution to use experimental design Experimental design-determine cause and effect relationship between causal beliefs and intervention preferences Experimental design-greater control over extraneous variables	Experimental manipulation lacks 'real world' approximation Criteria for inclusion in clinical sample was basic-used only 'depressed mood screening item'-sample not accurately defined Participants were not all currently experiencing depression Average age of participants younger than population (20)-lacks generalisability to population No details of	61%

							randomisation procedure given	
Khalsa et al. (2011) USA	N=156 59% female, 41% male Age:18-70 52% identified as an Ethnic Minority, 45% of which were African American	Participants were taking part in an RCT comparing medication with supportive-expressive psychotherapy. Participants had a diagnosis of Major Depressive Disorder according to the DSM-IV	To investigate the relationships between participant's beliefs about the causes of their depression, intervention preferences, and demographic variables.	Observational, cross-sectional Independent sample T-tests Regression analysis	Participants who preferred psychotherapy endorsed childhood issues as a reason for their depression to a higher degree than those who preferred medication. No relationship was found between endorsing biological reasons and wanting medication as a preferred intervention	Sample had mixed ethnicity (52% BME) so more generalizable to population Sample accurately defined in terms of depression, multiple measures taken	Used shortened and modified version of the RFD which was un-validated Effect sizes not reported No power calculation Did not measure strength of preference and also 'forced choice' as no 'no preference' option	78%
Meyer and Garcia-Roberts (2007) UK	N=97 63% female, 37% male Age:19-80 mean age 39.21 66% White British	Outpatients who were receiving psychological help for depression in Primary Care	To investigate whether particular causal beliefs are systematically associated with motivations to engage with reason-matching interventions. Whether congruence between reasons and interventions would predict higher levels of	Observational, cross-sectional Factor analysis Correlational analysis	Participants who endorsed 'childhood issues' or 'biological' causes of depression were more motivated to engage in an intervention that targeted these issues. In general, there was	Used the RFD to measure causal beliefs, a validated measure Used participants receiving therapy; sample was ecologically valid as naturalistic setting.	Developed new measure which had not previously been validated Possible demand characteristics Measure did not reflect 'real-world' therapeutic modalities Statistical analysis unclear	65%

			intervention motivation		increased motivation for interventions that matched participants reasons for depression		
Schweizer et al. (2010) Holland	N=221 57% female, 43% male Mean age 42 ethnicity not given	Participants who were seeking intervention for depression at a community mental health centre Diagnosis of Major Depressive Disorder or dysthymia as determined by DSM-IV	To investigate how illness attributions in depression might influence intervention assignment in a naturalistic setting	Observational, cross-sectional MANOVA analysis Correlational analysis	'Intra-individual' causal beliefs associated with choice of pure or combined CBT (CBT with medication). Endorsing biological attributions associated with choosing purely medication. No relationship between causal attributions and choosing Interpersonal Therapy (IPT)	Used the RFD (Addis et al., 1995) to measure causal beliefs, a validated measure Ecologically valid study as participant's preferences determined real intervention Large sample size	As naturalistic, extraneous variables not controlled for, intervention choice was not only dependent on participant preferences	87%
Steidtmann et al. (2012) USA	N= 785 56% Women, 44% male Age: 18-75 Mean age 44 60% White, 25% Black	'Chronic depression' as defined by DSM criteria and duration over 2 years. Scores of >20 on the HAM-D	Main aim: To investigate the relationship between intervention preference, attrition rates and outcomes in an RCT. Secondary aim: to examine relationships between patient	RCT Observation, cross-sectional MANOVA analysis Correlational analysis Independent sample T-tests	Participants who preferred medication more likely to endorse 'chemical imbalance' explanation for depression. Those who preferred combined intervention	Large sample size Sample very clearly defined RCT, 'gold standard' in research Appropriate method of randomisation and blinding	Scale assessed a limited number of beliefs Sample 'highly educated', not representative of population Possible confounding variables not assessed, such as	83%

			beliefs about depression aetiology and intervention preference		more likely to endorse 'stress' as a cause of their depression		psychotherapy experiences	

Research into causal beliefs in depression and preferences for interventions

The sample used by Khalsa et al. (2011) consisted of 156 participants who were involved in a Randomised Controlled Trial (RCT) comparing the efficacy of medication and 'supportive-expressive psychotherapy'. The researchers used a shortened version of the Reasons for Depression Questionnaire (RFD) (Addis et al., 1995) in order to measure participant's causal beliefs. T-tests were then used to investigate differences in causal beliefs based on intervention preference. Participants who preferred psychotherapy endorsed childhood issues as a reason for their depression to a greater degree than those who preferred medication. No relationship was found between endorsing biological reasons and preferring medication as an intervention.

Dunlop et al. (2012) also used participants involved in an RCT. 79 participants were asked what their preferred intervention was (CBT, medication or no preference) and to rate the strength of this preference as 'mild, moderate or strong'. The researchers used regression analyses to predict the relationships between beliefs and preferences. Causal beliefs were measured using five questions drawn from the Patient Attitudes and Beliefs Scale (Elkin et al., 1989). It was found that those who endorsed an unknown cause ('out of the blue') for their depression were less likely to prefer CBT than participants who held other causal beliefs. Those who endorsed 'pessimism' were less likely to prefer medication and those who endorsed 'emotional illness' were more likely to prefer medication. These findings were not expected by the researchers, who had hypothesised that those participants who endorsed biological causal beliefs would be more like to prefer medication. They speculated that other beliefs which were not measured, for example, about harmfulness of medication, may have had a larger impact on preferences.

Steidtmann et al. (2012) collected baseline data on intervention preferences and causal beliefs from participants involved in an RCT

investigating the efficacy of 'augmenting medication with psychotherapy'. The participants' level of depressive symptoms was classified as 'chronic', meaning that they had experienced depression either continually or intermittently for at least two years (DSM-IV criteria were used for diagnosis). The researchers asked participants at baseline which intervention they would prefer: medication only, combined psychotherapy and medication, or no preference. The researchers measured causal beliefs using the same five questions as Dunlop et al. (2012), which were drawn from the Patient Attitudes and Beliefs Scale (Elkin et al., 1989). The sample in this study was large, consisting of 785 adults. MANOVA analysis suggested a significant effect of 'intervention preference' on causal beliefs. Post hoc comparisons suggested that participants with a baseline preference for medication were significantly more likely to endorse an 'imbalance of brain substances' cause to their depression than participants who preferred a combination of interventions or had no preference. Participants who had a preference for a combination of interventions were significantly more likely to endorse 'stressful events' as a cause for their depression than those who preferred medication only or who had no preference.

Houle et al. (2013) used a sample of participants with 'first episode depression' in order to investigate the associations between intervention preferences, patient characteristics and representations of depression. In order to measure causal beliefs, they used a section of the 'Revised Illness Perceptions Questionnaire (IPQ-R)' (Moss-Morris et al., 2002). Factor analysis revealed three separate subscales, 'psychological attributions', such as personality or attitude; 'physical attributions', such as hereditary or medical illness and 'social attributions', such as family issues or loss of a significant relationship. Choice of interventions was antidepressants or psychotherapy. T-tests showed that those who preferred psychotherapy as an intervention more strongly endorsed social attributions for their depression than participants who preferred anti-depressants. However, when entered into a regression analysis, causal attributions did not predict intervention preference.

In a study conducted in the Netherlands, Schweizer et al. (2010) used a sample of 221 adults seeking intervention for depression at an outpatient centre to investigate whether causal beliefs had any effect on the therapy modality chosen (interpersonal or cognitive behavioural or medication). Participants received verbal and written descriptions of different therapeutic modalities and intervention type from their therapist, following which a joint choice between therapist and participant was made as to which therapy they would have, with the decision being led by participant choice. They then collected data on participant's causal beliefs using the the RFD (Addis et al., 1995). They found that there were significant differences in intervention choice according to beliefs; individuals endorsing 'intra-individual' attributions (that is, items that could be labelled as individual problems such as lack of achievement, existential crises, character traits and physical problems) were significantly more likely to choose pure or combined CBT (CBT with medication). There was a significant relationship between endorsing biological attributions and preferring to receive medication without psychotherapy. There was no relationship between causal attributions and choosing Interpersonal Therapy (IPT). The researchers argued that as participants in the study averaged a 10-month history of feeling depressed, IPT may have been less attractive to them as interpersonal problems that triggered their depression might have been felt to be in the past. Therefore, even if they endorsed interpersonal reasons for depression, they may not have chosen IPT as an intervention.

Research into causal beliefs and attitudes towards different interventions

Iselin and Addis (2003) primarily investigated the differences between clinical and non-clinical samples in terms of causal beliefs of depression and rated 'helpfulness' of interventions. Therefore, the sample was a mix of 36 students and 36 'mental health clients'. Participants were given vignettes of characters with depression and a 'reason' for their depression. The 'reason' was either a biological or psychological cause. They were then asked to rate on a Likert Scale how helpful different interventions would be for the character in the vignette. In analysis, interventions were divided into

'psychological' (talking therapies) or 'medical' (medication). They hypothesised that psychological interventions would get higher helpfulness ratings when paired with a vignette with a psychological description of the cause, and medical interventions would get higher helpfulness ratings when paired with a biological description of the cause. The results of an ANOVA supported this hypothesis, and planned comparisons revealed that matching causes and interventions resulted in significantly higher helpfulness ratings (at a 0.01 level). The researchers conclude that the results point to the importance of causal information on perceived intervention helpfulness.

In an experimental, independent samples design, Kemp et al. (2014) used a novel experiment to test the effects of a manipulated causal belief on perceived credibility and effectiveness of interventions for depression. Research participants (73 undergraduate students) who either had experienced in the past or were experiencing a current episode of depression were randomised to control or experimental conditions. All participants were administered a fake 'Rapid Depression Test', which was described to them as a test of neurotransmitter levels that determined whether or not depressive episodes were caused by a chemical imbalance within the brain. In the experimental condition, participants were given a 'positive result' and told that their depression was caused by an imbalance of serotonin within the brain and were presented with a bar graph depicting this. In the control condition, they were told that their depression was not caused by low serotonin. The credibility of this test was checked as part of the experiment. Participants then completed a rating scale measuring how much they endorsed different causal beliefs and how credible and helpful they believed CBT or SSRIs would be in reducing depressive symptoms (Credibility and Expectancy Questionnaire (CEQ), Devilly & Borkovec, 2000).

An ANOVA revealed a significant condition intervention interaction; participants in the 'positive results' condition rated psychopharmacology as more credible and more likely to be effective than psychotherapy, whilst participants in the control condition rated each intervention as equally credible and effective. The researchers conclude that holding the chemical imbalance theory as a belief may interfere with response to psychotherapy.

Gaudino et al. (2014) examined data from 52 participants who were inpatients hospitalised voluntarily for depression. Their study focused primarily on gender differences in perceived causes of depression; however, they also investigated causal beliefs, beliefs about medication, psychotherapy and 'intervention acceptability'. 'Intervention acceptability' was adapted from an un-published questionnaire and defined by asking participants how likely they would be to try or continue with different interventions. Causal beliefs were measured using the RFD (Addis et al., 1995). However, as this study focused on gender as a moderator of any relationships, and there were only gender differences found on the endorsement of biological and physical causal beliefs, these were the only ones entered into analysis with the other variables. The researchers found that higher endorsement of biological causal beliefs was correlated with a greater belief in the necessity of medication. There was no relationship found between causal beliefs and acceptability of, or other beliefs about, psychotherapy.

Budd et al. (2008) hypothesised that there would be a correlation between causal beliefs and the rated 'helpfulness' of current or past interventions. 164 members of a Welsh self-help organisation for people with depression were asked what they thought caused 'people to become depressed' (rather than asking participants about their own depression). The authors developed their own questionnaire for this purpose with 77 items measuring six possible causal domains. Participants were also asked to state intervention that they were receiving or had received for their depression, and to rate how helpful they found it on a Likert Scale. Interventions included antidepressant medication, counselling, CBT, Electro-Convulsive Therapy and Psychodynamic therapy. The belief that depression is caused by 'imbalance in brain biochemistry' was significantly positively correlated with the rated helpfulness of past or current medication use, although this was a weak correlation. No other significant correlations between causal beliefs and rated helpfulness of current or past interventions was found.

Meyer and Garcia-Roberts (2007) utilised a sample of 97 participants with depression who were currently receiving some form of psychological

therapy in GP surgeries. The goal of the study was to ascertain whether clients would be more motivated to engage in intervention if it matched their beliefs regarding the cause for their own depression. As there is no previously validated measure for reason-matching interventions, they developed their own questionnaire: the 'Motivations for Interventions' (MFI) measure. This measure was developed so that the items 'matched' the causal beliefs items on the Reasons for Depression Scale (RFD, Addis et al., 1995). For example, a' Characterological' item on the RFD, 'I am depressed because that's the type of person I am' would be matched with a 'Characterological' intervention on the MFI, 'I would like the kind of therapy that somehow changes the very core of my personality'. Participants had to rate on a Likert Scale, how much they agreed with statements on the MFI. The researchers also added a subscale to the MFI which did not correspond with any items of the RFD; 'Cognitive Reasons', expecting that clients already engaged in a CBT based approach would be more likely to hold beliefs reflecting cognitive reasons for their depression.

The researchers summarised the results using a 'congruence coefficient' (a statistic of similarity between causal beliefs and motivations for matching interventions). They found strong congruence between 'childhood' reasons for depression and motivation for psychological interventions that focus on childhood issues. They also found a strong congruence between 'biological' reasons for depression and biologically based interventions. They separated the RFD items into two factors: 'autonomous' and 'interpersonal' and found that they correlated at a 0.01 level with congruent interventions. The researchers conclude that participants were more motivated to engage in interventions that matched their causal beliefs.

Review of the methodology

Sample

All samples came from Western populations, limiting the scope of the research to these areas of the world. Non-Western countries have been shown to have different causal beliefs and intervention preferences (Hagmayer & Engelmann, 2014). The reviewed studies also mostly used

White populations, with a larger proportion of participants being female. However, this gender bias does reflect the difference in reporting of depression in the general population worldwide (Kuehner, 2003), it does mean that male views are overlooked as a result. With regard to ethnicity, in the US, African-Americans (4%) are significantly more likely to report major depression in surveys than White Americans (3.1%) (CDC, 2010). However, they are less likely to seek help, which may explain the lack of ethnic representation across the US studies. Khalsa et al. (2011) did have a more racially mixed sample, with 52% being of BME background. Since they also found differences in causal beliefs according to ethnicity, it is important not to generalise the results of this review to Black and Ethnic Minority populations (Khalsa et al., 2011).

The reviewed studies differed in regard to the type of 'clinical' sample utilised. Iselin and Addis (2003) and Kemp et al. (2014) had samples which were classed as 'clinical' but were not all currently experiencing depression. This strictly limits the transferability of the findings of these studies. In contrast, participants in the study by Gaudino et al. (2013) were so severely depressed that they were hospitalised, meaning that caution needs to be applied to transferring these findings to less severely depressed populations. Budd et al. (2008) did not ask participants if they were currently depressed or measured current symptoms of depression. However, 97% of the sample were currently receiving some kind of intervention or support for depression, indicating that they were currently depressed. These differences between samples make it more difficult to make generalisations to populations or meaningful comparisons across studies.

The remaining seven studies either used DSM-IV criteria to only accept participants with a diagnosis of depression or measured level of symptoms as defined by validated depression symptom measures. This increased specificity would hopefully reduce within-sample variation and enables easier comparison of the results of these studies.

When thinking about the definition of 'clinical' samples, it is important to remember that depression is a condition which can vary considerably in

symptom presentation, length and severity, so it would be very difficult for all the studies to measure identical populations (Kanter, Bush, Weeks & Landes, 2008). Even when the studies did use DSM diagnoses as their basis for inclusion, they had similar but slightly different exclusion criteria (e.g. substance misuse). Due to the complicated nature of mental health problems, excluding any co-morbidities is almost impossible. Furthermore, some authors (Lilienfeld, Waldman & Israel, 1994) have argued that even the use of the term 'co-morbidity' inaccurately assumes that the depression is a distinct disorder that can be separated out from other issues. Along those lines, the use of DSM criteria to define samples can be criticised due to its inherent reliance on a bio-medical, rather than biopsychosocial, model of mental health that assumes that human experiences can be categorised (Pilgrim, 2002). Therefore, the use of the DSM criteria as exclusion and inclusion criteria may not ensure that the participants in those studies make a more homogeneous sample than in the studies without this criterion.

In terms of sample size, eight out of the ten studies did not report a power calculation (Budd et al., 2008; Dunlop et al., 2011; Kemp et al., 2014; Khalsa et al., 201; Iselin & Addis, 1996; Meyer & Garcia-Roberts 2007; Schweizer et al., 2010; Steidtmann et al., 2012). This makes it difficult to determine whether the sample size was adequate for the purpose of the study; especially for studies that used smaller sample sizes such as Dunlop et al. (2011) who used data from 45 participants. Those studies that did report this calculation (Gaudino et al., 2014; Houle et al., 2013) reported moderately strong effect sizes, indicating that the clinical significance of their findings is 'moderate' (Hojat & Xu, 2004)

Measurement of causal beliefs

There was variability in the measurement of causal beliefs across studies. The majority of the studies used the RFD (Addis et al., 1995) to measure causal beliefs. This measure is normed and validated on both non-clinical and clinical samples (Addis et al., 1995) in the US and in the UK (Thwaites, Dagnan, Huey & Addis, 2004). It consists of 48 items which are divided into nine subscales. Data on the validity of the scale suggests that

the scales are distinct from current depression symptoms (Addis et al., 1995). As this measure has been carefully evaluated, it lends more validity to the findings of these studies and more meaningful comparisons can be made across the studies that used them.

Some studies did not use the full version of the RFD or amended the questionnaire in different ways. Khalsa et al. (2011) used a version of the RFD from a previous study (Leykin, DeRubeis, Shelton & Amsterdam, 2007) which was shortened to thirteen questions and an additional un-validated question; 'I was born to be this way' was added. Although it makes sense when using a clinical sample to try to reduce the demands on participants, deleting sub-scales means that the measure had fewer options. It may not have truly reflected the range of beliefs endorsed by people with depression, especially as they did not then norm this new version, possibly affecting the integrity of the scale. Indeed, Leykin et al. (2007) found a low Cronbach alpha for their 'characterological' subscale and needed to use a lower cut off than recommended for their factor loadings, raising questions as to the validity of the sub-scale. Both of these researchers (Leykin et al., 2007; Khalsa et al., 2011), despite using the same scale with a similar sample, found a different number of factors, which indicates that this scale lacked internal reliability, making it difficult to make inferences from the results. Houle et al. (2013) used a section of the IPQ-R (Moss-Morris, 2002). This measure was normed on samples with physical illnesses, such as Diabetes. Some of the items measuring 'physical' causal beliefs therefore lack validity for a sample with depression, for example 'smoking', 'diet or eating habits' or 'poor medical care in my past'.

Budd et al. (2008) and Iselin and Addis (2003) used bespoke measures to ascertain causal beliefs and these varied in quality. Budd et al. (2008) developed their own questionnaire for this purpose with questions derived from previous research on causal beliefs, as well as information about possible causes proposed by Cognitive Behavioural and Interpersonal therapies. They found a clear two factor structure to their measure 'Stress' and 'Depressogenic beliefs'. The 'Stress' factor contained items assessing trauma, social, economic and interpersonal problems. 'Depressogenic

beliefs' covered negative thought processes and beliefs. This is consistent with the sample that they used; most had had CBT intervention previously and may have been exposed to CBT based self-help literature from the self-help organisation itself (CBT focuses heavily on thought processes such as beliefs as causes of depression). Research has shown that previous intervention can affect causal beliefs (Leykin et al., 2007). This factor structure may not have been found with a different sample.

The use of bespoke measures makes it difficult to compare findings because of the lack of measure validation, and also because similar items can be conceptualised in different ways. For example, similar items on the 'stress' factor on the measure by Budd et al. (2008) were conceptualised by Iselin and Addis (2003) on their measure as 'psychological' factors. The research showed that these factors were associated with increased preference for or rated helpfulness of psychotherapeutic interventions, which is helpful in terms of differentiating between medication and psychotherapy. However, they are too broad to give any data about which type of therapy might be preferred.

Dunlop et al. (2012) and Stietmann et al. (2012) measured causal beliefs using a bespoke scale, drawn from the 'Patient Attitudes and Beliefs Scale' which pertains to participant beliefs about the causes of their depression (Elkin et al., 1989). This scale only presents five options to participants and may not fully reflect the range of beliefs today given the date it was developed. This scale is quite different from others used in research making it hard not only to categorise responses (for example, it is difficult to know which equivalent causal beliefs on the other measures listed could be equated with the 'out of the blue' item on this measure) or make meaningful comparisons to the other studies in this review. Although Dunlop et al. (2012) and Stietmann et al. (2012) used this same measure with similar samples, the results were not replicated, suggesting the measure may lack reliability.

Measurement of preferences and attitudes towards interventions

In order to rate preferences, perceived helpfulness, efficacy or credibility of interventions, most studies used unique measures using Likert

Scales or multiple choice questions. Likert scales are often used in research as they are easy to construct and straightforward for participants to complete. However, it can be difficult to discern the psychometric properties of these unique measures.

Meyer and Garcia-Roberts (2007) attempted to construct a more exhaustive measure, the 'Motivations for Interventions (MFI)' scale, using the RFD (Addis et al., 1995) as their basis. For each causal belief item on the RFD, they developed a congruent item on the MFI; 'I am depressed because I cannot make friends' and 'I would like a therapy to help me to improve my social relationships'. The researchers had problems with this scale, however, in terms of it being under-powered and having one less coherent subscale. Moreover, the researchers did not account for the influence of demand characteristics; it may be that the congruency they found between the RFD and MFI was because the participants worked out which items 'matched' and responded accordingly. Also, as the items on the MFI are not based on real-world modalities (e.g. CBT, Psychodynamic Therapy) results may not be helpful in deciding between a therapeutic modality, but may help therapists distinguish which facets of their therapy to focus on.

Study design

Only one study investigated preferences that determined real interventions; this study therefore had the most ecological validity as the results can be generalised to other real-life settings (Schweizer et al., 2010). RCTs, despite their advantages when looking at intervention outcomes, may not be so helpful in looking at preferences, as participants with strong preferences may not agree to randomisation and therefore not participate (Howard & Thornicroft, 2006). This is reflected in the study by Khlasa et al. (2011) who found that the average rating for all preference subscales was between 'probably not a reason' and 'probably a reason'; indicating not very strong preferences for any of the interventions.

The experimental research undertaken by Kemp et al. (2014) was necessarily artificial, as it would be unethical to deceive participants in a healthcare setting. The results of this study can be thought of as a helpful

approximation of how real life messages about causal attributions in depression may affect people's attitudes towards intervention. However, the results need to be replicated in more naturalistic settings before inferences can be drawn from the results. The researchers also do not give details about how randomisation took place, so there may have been possible selection bias in this study.

Two specific issues have been highlighted in the design of the following two studies which may have biased the outcomes: Dunlop et al. (2012) state that 'the patients endorsing neurochemical causes were equally likely to prefer medication or CBT or no intervention, indicating that acceptance of the medical model does not equate with a greater desire for medication intervention' (p.380). However, at the start of the study (before giving preferences) the participants were told that people with depression are equally likely (on average) to respond to CBT or medication. This could have been a strong influencing factor in participants rating their preferences, and thus have had a significant impact on the results.

One potential problem with the Schweizer et al. (2010) study was that because it was in a naturalistic setting, extraneous variables were difficult to control for. For example, intervention choice was decided in a discussion with the therapist. Although the researchers state that it was meant to be led by participant choice, it would be helpful to know the orientation of the therapist in order to partial out the effects on participant choice.

Summary and conclusions

Summary of findings

The main finding of the studies as a whole is that there is a positive relationship between biological causal attributions and preferring or believing in the efficacy of medication as an intervention. This was found by a number of cross-sectional studies of 'high' and 'moderate' quality (Budd et al., 2008; Gaudino et al., 2014; Iselin & Addis, 2003; Meyer & Garcia- Roberts, 2007; Schweizer et al., 2010; Steidtmann et al., 2012) as well as 'moderate' quality experimental research (Kemp et al., 2014). Three studies of 'high' quality did

not find this relationship: Dunlop et al. (2012), Khalsa et al. (2011) and Houle et al. (2013). These studies had problems with the validity of their measurement of causal beliefs which may explain their findings. Khalsa et al. (2011) hypothesise that this finding might indicate that participants who endorse biological beliefs may have other beliefs that make them wary of medication, such as a fear of the side effects.

In regards to causal beliefs and preferences for and attitudes towards psychological therapy, results were varied. Endorsing 'childhood issues' as a reason for depression increased motivation for childhood-focused interventions in one study (Meyer & Garcia-Roberts, 2007), however this research was of 'moderate' quality. Preference with psychotherapy was found to be associated with 'childhood issues' and 'social' reasons for depression in two 'high' quality studies (Khalsa et al., 2011; Houle et al., 2013). One 'high' quality study found that endorsement of 'intra-individual' causal beliefs was associated with preference for CBT or CBT with medication (Schweizer et al., 2010). Another 'high' quality study found the causal belief 'stress' to be associated with preference for combined intervention (Steidtmann et al., 2012) More generally, endorsement of more 'overarching' and less specific reasons for depression ('psychological', 'autonomous', 'intrapersonal') was associated with preferring (or perceiving to be helpful), 'matching' interventions across a number of studies (Iselin & Addis, 2003; Meyer & Garcia-Roberts, 2007; Schweizer et al., 2010). However, 'interpersonal' reasons were not associated with choosing the congruent intervention of IPT (Schweizer et al 2010). The researchers believe that this may be due to the length of time between participant's onset of depression and choosing treatment.

Research implications

There are some methodological flaws in the studies that indicate a need for future research. More research is needed in naturalistic settings, with a wider range of intervention choices offered and use of the RFD (Addis et al., 1995) to ensure the thorough and careful measurement of causal beliefs. As the link between causal beliefs and psychotherapy is less clearly

understood, research focusing on types of therapy rather than the choice between therapy and medication, would be helpful. Increasing Access to Psychological Therapies (IAPT) services may be ideal places for this to be carried out, given their expansion from purely CBT service into other types of therapy (Department of Health, 2012).

Focusing on populations underrepresented in the samples (men, ethnic minorities) would increase the generalisability of the research. However, as more White women access services for depression, further research may have to be creative in finding representative samples. Online research could help to reach people who may not access services.

As it seems biological attributions are associated with increased preference for medication, research could be conducted on information giving which might alter people's causal beliefs and whether this has any effect on the perceived usefulness of psychotherapeutic interventions. Ethical concerns may however, prevent this taking place in naturalistic settings.

Limitations of literature review

As the review is based in the context of the acceptance of the biopsychosocial model of conceptualising and treating mental health problems, papers were excluded that did not mention specific beliefs such as 'biology'. In the initial literature search, papers were excluded that focused on beliefs that were more generally conceptualised (e.g. internal or external attributions of negative events). Therefore, this review was limited to exploring causal beliefs from a specific perspective. In addition, as this paper was interested principally in the interventions for depression that were medical or psychological, papers were limited to cultures where psychotherapy and medication are considered effective and appropriate interventions for problems.

Clinical implications

Results of the research show that biological causal beliefs are associated with increased belief in and preference for medication. Promotion

of these beliefs may reduce preferences for psychological interventions. For clinicians, it raises the issue of how ethical it is to leave clients with an inadequate understanding about what causes depression (Blease, 2014) given that it affects their intervention choices. Empowering clients would mean giving them more information about the lack of evidence for the biological model, allowing them to make a more informed choice (Blease, 2012).

Although respecting client individual opinions and expertise is in the code of ethics for Clinical Psychologists in the UK, they must also endeavour to support their self-determination (BPS, 2009), and information giving about evidence can be seen as part of this. Clinical Psychologists in mental health care are often placed within multidisciplinary teams with other health and medical professionals. Within this context, working with the team to develop a more co-ordinated view is part of a psychologist's competencies (Division of Clinical Psychology [DCP] 2008). Psychologists are obliged to do this by using clear communication and 'relevant evidence' (DCP, 2008), which would apply to the disseminating of the research behind ideas such as the chemical imbalance theory within the services they work.

Within a therapeutic context, discussing causal beliefs in detail prior to starting therapy could help improve intervention matching. However, this is not always possible in target driven services, or may be an area so in-depth that it constitutes the entirety of the therapeutic work. Meyer and Garcia-Robert's (2007) preference research suggests that no matter what type of psychological therapy is being conducted, focusing on specific parts of the therapy which are congruent with the client's causal beliefs would be helpful. Causal beliefs could be included in formulations that aim to identify the best intervention for the client (BPS, 2011).

Discussion of causal beliefs could also contribute to "collaborative involvement" (Tryon and Winograd, 2011), an important part of the therapeutic relationship. Formulations should be jointly developed (BPS, 2011) and can include a range of causal factors. A thorough formulation

including causal beliefs would help the client feel understood and strengthen the therapeutic alliance.

Conclusion

The ten studies in this literature review were of varying quality; however, seven studies found evidence for a positive relationship between endorsing biological causal beliefs and preference or positive attitude towards medication. The evidence from several good quality studies also suggests that people prefer psychological interventions that 'match' their causal beliefs. More research is needed on causal beliefs and preferences in different therapeutic modalities in more naturalistic settings. Future research could investigate how information giving about causes of depression affects intervention preferences.

Clinically, the findings from this research suggests clinicians should address causal beliefs and attempt to match facets of therapy to meet them. For psychologists, ethical guidelines suggest they have a role in challenging biological explanations of depression in clinical settings.

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Appendices

Appendix 1. Quality Appraisal Checklist

1. Were the aims of the study clearly stated in the title/abstract?
2. Was there a rationale or scientific background for the study?
3. Was the method used appropriate to answer the research question?
4. Were the participants recruited in a way that reduced selection bias?
5. Were the participants randomized in an acceptable way? (experimental research only)
6. Was assessment blind? (experimental research only)
7. Did the study explain how the sample size was arrived at?
8. Was the study sample clearly defined?
9. Was a representative sample achieved?
10. Were the measures used fit for purpose? (reliable and valid?)
11. Were the statistical tests used to assess the main outcomes appropriate?
12. Are the main results presented clearly (with effect sizes and confidence intervals if appropriate)?
13. Was the data analysis sufficiently rigorous?
14. Are results summarised with reference to study aims?
15. Are results interpreted with evidence for and against the researcher's arguments?
16. Is clinical as well as statistical significance discussed?
17. Are limitations of the study discussed?
18. Are the results generalizable to the local population?

Appendix 2. Quality Appraisal Table

Papers	Questions from Quality Appraisal Checklist

Papers	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	Total	%
Budd et al., (2008)	Y	Y	P	P	n/a	n/a	N	Y	P	Y	Y	Y	Y	Y	Y	Y	Y	P	26	81
Dunlop et al., (2012)	Y	Y	N	Y	Y	Y	N	Y	P	P	Y	Y	P	Y	Y	Y	Y	P	28	77
Gaudino et al, (2013)	Y	Y	Y	P	n/a	n/a	Y	Y	P	Y	Y	Y	Y	Y	Y	Y	Y	P	29	90
Houle et al., (2013)	Y	Y	Y	P	n/a	n/a	Y	Y	P	N	P	Y	P	Y	Y	Y	Y	P	25	78
Iselin & Addis (2003)	Y	Y	Y	P	n/a	n/a	N	Y	P	N	Y	P	Y	Y	Y	Y	Y	P	24	75
Kemp et al., (2013)	Y	Y	P	N	UN	UN	N	P	P	P	Y	Y	Y	Y	Y	Y	Y	N	22	61
Khalsa et al., (2011)	Y	Y	P	Y	n/a	n/a	N	Y	Y	P	Y	Y	P	Y	Y	P	Y	P	25	78
Meyer & Garcia-Roberts (2007)	Y	P	Y	P	n/a	n/a	N	Y	P	P	P	P	P	Y	P	Y	Y	P	21	65
Schweizer et al., (2010)	Y	Y	Y	Y	n/a	n/a	N	Y	Y	Y	Y	P	Y	Y	Y	Y	Y	p	28	87

Key: Y=Yes, N=No, P= Partially, UN= Unable to determine, N/A= not applicable

Scoring: Y=2 points, P=1 point, N=0 points, UN=0 points

Appendix 3. Author Guidelines for submission to 'Psychotherapy Research'.

Manuscript preparation

1. General guidelines

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- Manuscripts are accepted in English (for non-English submissions see Manuscript submission section below). Oxford English Dictionary or US spelling are preferred. Please use double quotation marks, except where 'a quotation is 'within' a quotation'. Long quotations of 40 words or more should be indented without quotation marks.
- There is no word limit for articles but authors should include a word count with their manuscript.
- Manuscripts should be compiled in the following order: title page (including Acknowledgements as well as Funding and grant-awarding bodies); abstract; keywords; main text; references; appendices (as appropriate); table(s) with caption(s) (on individual pages); figure caption(s) (as a list).
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- Abstracts of 100-200 words are required for all manuscripts submitted. The abstract should be structured with the following headings: **Objective**, **Method**, **Results**, **Conclusions.**
- Each manuscript should have 5 to 6 keywords.
- Search engine optimization (SEO) is a means of making your article more visible to anyone who might be looking for it. Please consult our guidance here.
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- Description of the Journal's reference style.
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- Guide to using mathematical scripts and equations.
- Word templates are available for this journal. If you are not able to use the template via the links or if you have any other template queries, please contact authortemplate@tandf.co.uk.

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- Figure captions must be saved separately, as part of the file containing the complete text of the manuscript, and numbered correspondingly.
- The filename for a graphic should be descriptive of the graphic, e.g. Figure1, Figure2a.

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Last updated 7 October 2014

Chapter 2: Empirical paper

The role of causal beliefs in predicting self-stigma and prognostic-pessimism in adults with depression

For British Journal of Clinical Psychology (5000-word limit *not including abstract, reference list, figures or tables*)

5000 words (not including abstract, figures or tables, as per journal stipulations)

5905 words (including abstract, tables and figures as per award stipulations)

Contents

Abstract

Objectives

To investigate the beliefs of people with depression about the causes of their depression, and how those beliefs might predict self-stigma, as well as prognostic pessimism. To also investigate the role of self-efficacy in mediating the relationship between these variables.

Design

A cross-sectional design was used and measures were administered online.

Methods

A sample of 184 participants who identified as currently experiencing depression participated. Participants completed self-report measures of depressive symptoms, self-efficacy, self-stigma, prognostic-pessimism and beliefs about the causes of their depression.

Results

Regression analyses identified that self-efficacy and depressive symptoms were significant predictors of self-stigma, accounting for 30% of the variance in scores. No significant predictors of prognostic pessimism were found. As biological causal beliefs were not significant predictors of the criterion variables, mediation analysis was not undertaken.

Conclusions

Causal beliefs had no significant effect on measures of self-stigma or prognostic pessimism in a sample of people with depression living in the UK.

Practitioner Points

Clinical implications

-People with more severe depression might experience more self-stigma, making it possibly harder for them to seek help

-Interventions focusing on the biological causes of depression as a way to reduce self-stigma may not be effective

Cautions

– The data for the outcome variable 'prognostic pessimism' was not normally distributed, suggesting a biased sample.

-The majority of the sample were women of White British origin. The results, therefore are not generalisable to the UK population.

Introduction

Depression

About one in 20 adults will experience an episode of depression each year in the UK (National Institute for Health and Care Excellence, 2015) and it is the third most common reason for a consultation in general practices (Office for National Statistics, 2013). Depression is most often characterised by low mood and loss of pleasure in activities, but can also include a range of other symptoms such as feeling worthless or excessively guilty, insomnia and suicidal thoughts (NICE, 2015). Depression presents a global issue, and is expected to be one of the three leading cause of disease burden worldwide by 2030 (Mathers & Loncar, 2006).

Causal beliefs of depression in the general public

A 2012 meta-analysis of 16 studies examining time trends in public attitudes in Western countries found that increasingly, depression is attributed to chemical imbalances in the brain or genetic causes (Schomerus et al., 2012). In the UK, a 2011 survey undertaken by the Office for National Statistics found that 77% of adults agreed with the statement 'mental illness is an illness like any other' (The NHS Information Centre, 2011). This statement implies that mental health problems have a biological basis, the same as physical illnesses, and should be treated in a similar manner (Albee & Joffe, 2004; Malla, Joober & Garcia, 2015;). The popular media frequently promotes the chemical imbalance theory of mental illness, without citing

references to support these claims (Leo & Lacasse, 2008). An analysis of magazine articles over a 25-year period showed that during the 2000's, depression was consistently portrayed as a medical issue relating to brain functioning (Clarke & Gawley, 2009).

There is research showing that presenting biological reasons as the main cause of mental illness can reduce the blame attached to those with mental health problems by the general public (Phelan, Cruz-Rojas & Reiff, 2002). However, research has also shown that it can lead to more stigmatised attitudes, such as a need for increased social distance (Lauber, Nordt, Falcato & Rossler, 2004) and more pessimistic views about treatment outcomes in lay populations (Phelan, Yang & Cruz-Rojas, 2006). Studies with participants from the general public show that biogenetic attributions are related to higher perceived dangerousness and unpredictability of those with a mental health problem, due their perceived lack of control over their behaviour (Angermeyer, Holzinger, Carta & Schomerus, 2014). Research findings suggest that biomedical causal explanations for depression do not reliably reduce blame and do not increase social acceptance (Kvaale, Gottdiener & Haslam, 2013; Schomerus et al., 2012).

If these patterns are seen in the general public, the question arises as to whether similar views are held by people who experience mental health problems, namely, do biological causal beliefs increase self-stigma and pessimistic views about recovery in this population? Prognostic expectancies are important to investigate as they account for a large proportion of the change observed in treatments for depression (Kirsch, 2010; Rutherford, Wager & Roose, 2010). Self-stigma is being aware of a stereotype, agreeing with it, and applying it to one's self (Watson, Corrigan & Larson, 2007). In this way prejudice and discrimination against people with mental health problems are internalised. A study conducted in 13 European countries found that about 21% of people with a diagnosis of affective disorder experienced self-stigma to a 'moderate to high' degree (Brohan et al., 2010b). Self-stigma is negatively correlated with a range of psychosocial variables (self-esteem, hope, empowerment) and can result in lack of engagement in care and other activities that promote recovery (Corrigan,

Larson & Rusch, 2009); therefore, any insight into potential predictors will be useful (Livingstone & Boyd, 2010).

Causal beliefs in people who experience depression

Research that has looked at the causal attributions of those who have experienced or who are experiencing mental health problems is less extensive than that undertaken with the general public (Lobban, Barrowclough & Jones, 2003). Kemp, Lickel and Deacon (2014) investigated the effects of the chemical imbalance explanation for depression on 73 students who stated that they had previously or currently had depression. They gave participants results of a bogus biological test that indicated that their current or previous depression was a result of a chemical imbalance in their brains. The researchers found that when participants were informed that they had a chemical imbalance, it elicited greater prognostic pessimism. Kemp, Lickel and Deacon (2014) also found that advocating the chemical imbalance theory of depression was found to lower individuals' perceived ability to successfully regulate their depressed moods, a construct closely related to self-efficacy.

Lebowitz, Ahn and Nolen- Hoeksema (2013) looked at the effects of biological attributions of depression amongst adults who were currently experiencing depressive symptoms. 108 participants were recruited online and scored over 16 on the Beck Depression Inventory (Beck, Steer & Brown, 1996) indicating that they had at least 'mild depression' as defined by the measure. They found that heightened depression severity and endorsement of a biological cause of their depression was significantly associated with prognostic pessimism. Brown et al. (2007) investigated the causal attributions of 191 patients in primary care in the US taking anti-depressant medication, and found that those participants with more severe symptoms of depression and who endorsed a heredity cause of depression were more likely to believe that their symptoms would last longer than those not endorsing this belief.

It is hypothesised that biological attributions may elicit greater prognostic pessimism as they may imply that mental health problems are an

intrinsic part of that person's identity (Easter et al., 2012; Nelkin & Lindee, 1995) and reinforce concerns about untreatable nature of mental health problems (Deacon & Baird, 2009; Lam & Salkovskis, 2006). It is possible that this would reduce a person's perceived self-efficacy over their depression. Self-efficacy (Bandura, 1977) is a person's belief in his or her ability to succeed in a particular situation or reach their goals.

All of the research on biological causal beliefs and prognostic pessimism in clinical samples has been conducted in the US, where biological attributions of mental health problems are more widely publicised and psycho-tropic drugs are advertised on television (Park & Ahn, 2013). Little is known about associations with causal attributions amongst people with mental health problems in the UK, despite biological theories having a prominent place in the media and some mental health services (Colomboa, Bendelowa, Fulforda & Williams, 2003; Lenovos & Redman, 2014). Not all of the studies measured symptoms of depression in their samples. As symptoms of depression can include hopelessness about the future and thinking negatively about the self (Beck, Rush, Shar & Emery, 1979) it is expected that depression severity would be associated with prognostic pessimism. More research is needed to consolidate the findings of Lebowitz et al. (2013) and Brown et al. (2007). Although there are associations between stigma and biological causal attributions in the general public, so far research has not investigated whether this is the case in clinical populations.

Study Aims

The aim of this research was to explore predictors of self-stigma and prognostic pessimism in a UK sample and the potential mediating role of self-efficacy on the relationship between biological causal beliefs and self-stigma and prognostic pessimism. It is suggested that reduced self-efficacy will explain how endorsement of the biological model leads people to have increased self-stigma and greater prognostic pessimism.

Hypotheses

1.   Higher depression severity, lower self-efficacy, and biological causal attributions of depression will predict higher self-stigma (SS) and worse prognostic pessimism (PP).

2.   Greater endorsement of a biological model of depression will predict greater SS and worse PP.

3.   Self-efficacy (SE) will act as a mediator between causal attributions of depression and SS and PP. That is, biological causal attributions will lead to decreased self-efficacy, which will lead to heightened SS and worse PP.

Method

Sample

The sample comprised 184 adults who responded to an online advert asking for participants who were 'currently experiencing depression', were over 18, and living in the UK. Due to lack of translation services, participants also had to be able to read and write in English. They were asked to take part only if depression was their 'main problem'. As depression often co-occurs with other mental health problems, such as anxiety (Hepgul et al., 2016; Sunderland, Slade & Baillie, 2010), participants with other mental health problems were included if they perceived their other problems as secondary to depression.

Demographic data was collected on gender, age, and ethnicity to ascertain whether a representative sample was achieved (Table 1).

Table. 1

Participant Demographics; Gender, Ethnicity and Age.

Gender	N	Percentage
Male	49	27%
Female	134	73%
Transgender	1	<1%
Ethnicity		
White British	164	89%
White Irish	4	2%
Any other White Background	5	3%
Mixed race	6	3%
Asian	3	1%
African	1	>1%
Arab	1	>1%
Age		
Mean	35.9	
SD	12.8	
Range	18-67	

Procedure

Ethical approval was obtained from Staffordshire University ethics committee (Appendix 3). Participants were recruited online via an advert placed on social media (Appendix 1). The advert was shared on the following websites; Facebook, Instagram and Twitter. On Facebook, the advert was posted in groups relating to depression or depression support. Potential participants used a link to access further details about the study and the study itself on the secure site Qualtrics.com. On Qualtrics.com, the option to not record IP addresses and other identifying information was activated by the researcher in order to preserve anonymity. If participants wanted to withdraw their data, they were asked to choose a 'password' that they could email the researcher with in order to identify their data. The password and participant number were stored separately from identifying or research data.

Participants were asked to consent to the study and then filled out demographic information and four measures (Appendix 2), which was estimated to take about 15-20 minutes to complete. The participants were also informed at the beginning and end of the study that they would not be able to contact or be contacted by the researcher to discuss their results even if they were feeling very depressed or suicidal. They were provided with information about how to contact NHS services, helplines and support groups, as well as what to do if the participant felt suicidal. This information was taken from the NHS website (NHS Choices, 2015). See Appendix 2 for a copy of this information.

The NHS and the British Psychological Society have published guidance on the use of social media to actively involve people in research (BPS, 2013; Involve, 2014), which the researcher followed. The rationale for choosing to conduct the study online is that it is more convenient for participants with depression (Wise et al., 2016), may overcome the barrier of stigma in face-to-face recruitment (Thompson, Heller & Rody, 1994), and might be more attractive to potential participants due to its anonymity (Temple & Brown, 2011). It also enabled the researcher to source a wider variety of participants with depression and avoid a selection bias of just those who are already in mental health services. This is especially important as 75% of people with mental health problems receive no support at all (Mental Health Taskforce, 2016). Researchers have found that online populations are often more diverse and produce equal quality data as traditional recruitment methods (Shapiro, Chandler & Mueller, 2013).

Assessment measures

Depression severity. 'The Patient Health Questionnaire (PHQ9)',
(Kroenke & Spitzer, 2002) was used to measure depression severity (see
Appendix 2, p.106) The diagnostic validity of the nine-item PHQ-9 was
established in studies involving eight primary care and seven obstetrical
clinics. Questions measure symptoms of depression, and participants are
asked to estimate how often they have suffered these symptoms over the
previous two weeks.

This tool was chosen as it is brief, easy to complete and has reliable
and consistent psychometric properties; Cronbach alphas of .86 and .89
(Kroenke, Spitzer & Williams, 2001). The estimated time for completion is
three minutes. It is widely used in UK clinical services.

Self-stigma. Self-stigma was measured using a questionnaire
developed by Kendra, Mohr and Pollard (2014) (see Appendix 2, p.108). The
measure contains 7 statements on how the respondents feel about 'having
psychological problems' and participants are asked to rate, on a four-point
Likert scale (Strongly Agree, Agree, Disagree, Strongly Disagree) how much
they agree with each statement. Statements include 'I feel ashamed of
myself for having psychological problems'.

This scale was designed to address concerns the researchers had
that other stigma scales lacked face validity for people with depression
(Mittal, Sullivan, Chekuri, Allee, & Corrigan, 2012). In order to develop the
scale, items were borrowed or adapted from previously used and established
measures investigating stigma. Pilot studies and confirmatory factor analyses
indicated good reliability and validity. Cronbach's alpha ranged from .85–.87.
Coefficients above 0.7 are acceptable (Pallant, 2010).

Prognostic pessimism. Previous research has used a variety of
different methods to measure PP as no stand-alone measure has been
developed and validated. The measurement of PP in this study is that used
in a similar study by Lebowitz et al. (2013) (see Appendix 2, p.116). Lebowitz
et al. (2013) used a single item that asked participants 'How long do you

think that you will continue to feel depressed?' The seven possible answers ranged from 'less than 1 week' to 'indefinitely'.

Self-efficacy. Self-efficacy was measured using the 'General Self-Efficacy Scale' (Schwarzer & Jerusalem, 1995). See Appendix 2, p.109 for a copy of the measure) This ten item scale assesses the strength of an individual's belief in his or her own ability to respond to novel or difficult situations, and to deal with any associated obstacles or setbacks. An example item is 'I can remain calm when facing difficulties because I can rely on my coping abilities' and a four point Likert Scale is used ranging from 'not at all true' to 'exactly true'. In terms of internal consistency, Cronbach's alphas ranged from .76 to .90, with the majority in the high .80s (Schwarzer & Jerusalem, 1995).

Causal attributions. Causal attributions of depression were measured using the 'Reasons for Depression Questionnaire' (RFD; Addis, Truax, & Jacobson, 1995). See Appendix 2, p.111 for a copy of this measure. The RFD lists 48 reasons for depression and asks participants to rate how much they agree with these reasons using a four point Likert Scale (definitely not a reason, probably not a reason, probably a reason, definitely a reason). The measure was normed on a UK sample of clinical and non-clinical populations (Thwaites, Dagnan, Huey & Addis, 2004). Cronbach alphas between 0.73 and 0.89 indicate high reliability for all subscales including a further subscale (biological) added since the measure was initially developed. There are nine subscales to the RFD, each categorising reasons for depression as; Characterological, Interpersonal Conflict, Intimacy, Existential, Achievement, Childhood, Physical, Biological and Relationship. Example items include 'Other people don't like me', 'I have no set goals in my life' and 'I have a chemical imbalance'.

Analysis

For a multiple regression with a total of 11 predictor variables (nine types of causal attributions on the RFD, depression severity and self-efficacy), for a medium effect size, 0.15, with power at 0.8 and alpha at 0.05, a GPower calculation (Faul, Erdfelder, Buchner & Lang, 2009) determined

that 122 participants were needed. Other observational research on causal beliefs in clinical samples have used similar sample sizes to this study; Brown et al. (2007) used a sample of 191 participants to undertake regression analysis and Lebowitz et al. (2013) used a total of 148. Dancey and Ready (2002) recommend having at least 15 participants per variable. The sample of 184 in this study was thus deemed sufficient.

Two separate multiple regressions were conducted for each criterion variable (SS and PP) using the predictor variables of causal attributions, depression severity and self-efficacy. Regressions were conducted using SPSS 23 for Windows (IBM Corp, 2012). Normality checks were undertaken, including homoscedasticity, linearity and independence of errors. The Durbin-Watson (Durbin & Watson, 1950) tests demonstrated that the residuals were independent, and VIF and Tolerance statistics showed that there was no multicollinearity between the variables (Neter, Wasserman & Kutner, 1989).

The criterion variable, PP, however, significantly violated linearity and homoscedasticity (see Appendix 5). Significant Shapiro-Wilk and Kolmogorov-Smirnov tests also confirmed that the data significantly deviated from a normal distribution (Corder & Foreman, 2014). Scores on this measure were skewed towards the higher end of the scale (greater prognostic pessimism). Due to these violations, a bootstrapping analysis was conducted to improve accuracy in relation to the confidence intervals and significance levels, which are reported in the results (Hesterberg, Moore, Monaghan, Clipson & Epstein, 2005).

Results

Descriptive statistics

Descriptive statistics for each criterion variable and predictor variable are presented in Table 2. The mean score on the PHQ9 was 15.9, which is in the 'moderately-severely depressed' range of scores (Spitze and Kronke, 2002). 86% of participants scored 10 or over indicating that they would meet criteria for clinical depression within mental health services (Spitze & Kronke, 2001). On The General Self-efficacy scale (Schwarzer & Jerusalem, 1995),

mean score was 23 out of a possible 39. Higher scores on this measure reflect greater self-efficacy. The self-stigma measure (Kendra et al., 2014) is scored out of 28, and the higher the score, the greater the self-stigma. The mean score was 20 out of a possible 28. For prognostic pessimism, the mean score was high on the scale (towards 'indefinitely'), suggesting most participants felt that their depression would last for an indefinite amount of time.

Table 2

Descriptive Statistics for Criterion Variables (Self-Stigma, Prognostic Pessimism & Self- Efficacy) and Depression Severity

Measure	Prognostic Pessimism	Self-stigma	Depression Severity	Self-Efficacy
Mean (SD)	5.9 (1.3)	20.2 (4.0)	15.9 (5.9)	23.3 (5.6)
Range	1-7	9-28	0-27	10-39

Correlations

Bivariate correlations were undertaken to examine which predictor variables were significantly correlated with the criterion variables (Table 3). The Depression Severity predictor variable, was moderately positively correlated with SS (r= 0.512, p<0.01), weakly positively correlated with PP (r= 0.197, p<0.01) and was weakly negatively correlated with self-efficacy (r= -0.358, p<0.01). As expected, participants with more severe symptoms of depression had higher self-stigma, felt somewhat that their depression would last longer and felt somewhat less confident in their ability to assert control over their life. SE was weakly negatively correlated with PP (r= -0.219, p<0.01) and weakly negatively correlated with SS (r= -0.383, p<0.01). Higher self-stigma and worse prognostic pessimism was associated with somewhat lower self-efficacy.

PP was weakly positively correlated with characterological (r=0.245, p<0.05), interpersonal (r=0.210, p<0.05), intimacy (r=0.256, p<0.05), achievement (r=0.228, p<0.05), physical (r=0.159, p<0.05) and existential

beliefs (r= 0.182, p<0.05). SS was weakly positively correlated with interpersonal beliefs (at p<0.01) childhood, achievement, intimacy, interpersonal, characterological and existential beliefs (at p<0.05). SE was moderately negatively correlated with existential beliefs (p<0.01) weakly negatively correlated with interpersonal, intimacy and achievement beliefs (at p<0.01) and weakly negatively correlated with physical beliefs (p<0.05).

Table 3

Pearson's Correlation Matrix for the Dependent Variables (Self-Stigma, Prognostic Pessimism) and the Predictor Variables (Self-Efficacy, Causal Attributions, Depression Severity)

Measure	PP	SS	DS	SE	Character	Existential	Interpersonal	Intimacy	Achievement	Childhood	Relationship	Physical	Biological
Prognostic pessimism	-	.128	.197**	-.219**	.245*	.182*	.210*	.256*	.228*	.123	.085	.159*	.120
Self-stigma		-	.512**	-.383**	.145*	.267*	.366**	.336*	.340*	.234*	.044	.131	-.021
Depression Severity			-	-.358**	.117	.227**	.302**	.310**	.241**	.239**	.107	.199**	.060
Self-efficacy				-	-.104	-.390**	-.379**	-.254**	.287**	-.127	-.129	-.185*	-.125
Character					-	.453**	.334*	.331*	.388*	.186*	.072	.234**	.387**
Existential						-	.397**	.416**	.681**	.174*	.094	.344**	.129
Interpersonal							-	.565**	.565**	.352**	.309**	.176*	.058*
Intimacy								-	.416**	.412**	.301**	.205**	-0.28
Achievement									-	.254**	.085	.270*	.086
Childhood										-	.194**	.208**	.011
Relationship											-	.131	.020
Physical												-	.098
Biological													-

*. Correlation is significant at the .05 level (two-tailed)
**. Correlation is significant at the .01 level (two-tailed
Note: DS: Depression severity (Spitze & Kronke, 2001), SE: Self-efficacy (Schwarzer & Jerusalem, 1995), PP: Prognostic Pessimism, SS: Self-stigma (Kendra, Mohr & Pollard, 2014)

Multiple Regressions

Separate multiple regression analyses were conducted for SS and PP. Each model and results will be outlined separately. For each model, all variables were initially included in order to examine and control for any confounding effects. Predictor variables that were not significant in the initial regressions were removed in order to improve the precision of the model, and the regressions re-run. Table 4 shows the results of the initial regression for SS with all predictor variables included. Table 5 shows the results of the regression model with only the significant predictors included.

Table 4

Summary of Initial Regression Analysis for Variables Predicting Self-Stigma (N=184) with All Variables entered: Unstandardized and Standardised Co-efficients, Significance Levels and Confidence Intervals

	Model 1					
	B	SE	B	Sig.	95% CIs	
					Lower	Upper
Constant (SS)	16.873	2.330		.000	12.274	21.471
DS	.253	.046	.374	.000	.162	.344
SE	-.140	.052	-.194	.008	-.243	-.037
Causal beliefs						
Character	.219	.580	.029	.706	-.927	1.365
Existential	-.295	.475	-.058	.536	-1.232	.643
Interpersonal	.389	.456	.075	.395	-.511	1.288
Intimacy	.469	.435	.088	.282	-.389	1.327
Achievement	.908	.552	.154	.102	-.181	1.997
Childhood	.206	.310	.046	.507	-.405	.817
Relationship	-.399	.313	-.084	.204	-1.016	.219
Physical	-.147	.325	-.030	.653	.789	.495
Biological	-.507	.416	-.082	.225	-1.329	.315

Note. $R^2 = .38$, Adjusted $R^2 = .30$

Table 5

Summary of Regression Analysis for Variables Predicting Self-Stigma (N=184) with only Significant Predictors Entered: Unstandardized and Standardised Co-efficients, Significance Levels and Confidence Intervals

	B	SE	β	Sig.	95% CIs	
					Lower	Upper
Constant	19.421	1.545		.000	16.373	22.469
DS	.291	.045	.430	.000	.203	.380
SE	-.165	.048	-.229	.001	-.259	-.071

Note. R^2 = 30, Adjusted R^2 = 25
Dependent variable: Self-stigma
Predictors: Depression severity, self-efficacy

Self-stigma

The significant predictors of self-stigma were depression severity and self-efficacy. When these predictors were included, the model (Table 5) accounted for 30% (R^2) of the variance in SS, 25% when adjusted (R^2 Adjusted). The model was significant $F(2, 181) = 40.2$, $p<0.001$ meaning that greater severity of depression, and lower self-efficacy, predicted higher SS (see Appendix 7 for ANOVA table). These findings provide partial support for the hypothesis that depression severity and self-efficacy would be significant predictors of SS. However, contrary to the hypothesis, biological causal beliefs did not predict SS.

For mediation analysis to take place, relationships should be significant between the predictor, mediator and criterion variables. As there was not a significant relationship between biological attributions and self-stigma, or biological attributions and self-efficacy, the mediation analysis could not be conducted.

Prognostic Pessimism

As stated, the data for prognostic pessimism violated assumptions (Appendix 5). Therefore, a bootstrapping method was performed (Table 6). There were no significant predictors of PP before and after bootstrapping

was undertaken. The boot strapping demonstrated that the skew in the data and the violations did not affect the significance of the regressions. Table 6 shows the results of the initial regression for PP with all predictor variables included, as well as bootstrapping analysis.

Table 6

Summary of Regression Analysis for Variables Predicting Prognostic Pessimism (N=184): Unstandardized and Standardised Co-efficients, Significance Levels and Confidence Intervals with Bootstrapping Comparisons

	Model 1				95% CIs		Boot strapping				95% CIs	
	B	SE	β	Sig.	Lower	Upper	*B*	Bias	β	Sig.	Lower	Upper
Constant	4.350	.939		.000	2.497	6.203	4.350	-.006	1.069	.001	2.272	6.346
DS	.018	.019	.076	.341	-.019	0.54	.018	.000	.021	.387	-.022	.058
SE	-.037	.021	-.149	.079	-.079	.004	-.037	-.001	.023	.110	-.084	.007
Causal Beliefs												
Character	.423	.234	.164	.072	-.039	.884	.423	-.004	.236	.069	-.018	.896
Existential	-.232	.191	-.133	.227	-.610	.146	-.232	.007	.190	.228	-.583	.164
Interpersonal	-.086	.184	-.049	.638	-.449	.276	-.086	-.015	.162	.577	-.421	.213
Intimacy	.290	.175	.158	.100	-.056	.635	.290	-.007	.164	.083	-.022	.621
Achievement	.285	.222	.140	.202	-.154	.724	.285	.009	.223	.201	-.154	.711
Childhood	-.030	.125	-.020	.807	-.277	.216	-.030	-.006	.133	.800	-.287	.232
Relationship	.014	.126	.008	.914	-.235	.263	.014	.013	.122	.913	-.221	.256
Physical	.104	.131	.061	.429	-.155	.363	.104	.007	.136	.436	-.170	.374
Biological	.084	.168	.039	.618	-.247	.415	.084	-.004	.184	.639	-.311	.425

Discussion

Summary of findings

A range of reasons for depression were endorsed by the sample, reflecting previous research using the RFD (Addis et al., 1995; Thwaites et al., 2004). This study did not find that biological causal beliefs, or any other type of causal beliefs, were significant predictors of self-stigma or prognostic pessimism. No significant predictors were found for prognostic pessimism, but depression severity and self-efficacy were found to be significant predictors of self-stigma. The results of the investigation into the predictors of self-stigma support results from a meta-analysis by Livingstone and Boyd (2010). They established that self-stigma was significantly moderately correlated with symptom severity (r^2 .41, $p < .001$) amongst people with mental health problems. In seven of the studies reviewed in the meta-analysis, there was also a significant negative correlation between self-efficacy and self-stigma (Livingstone & Boyd, 2010).

What this study did not find is that biological beliefs are associated with higher self-stigma. This suggests that the relationship between endorsement of biological reasons for depression and increased stigma seen among the general public (Kvaale, Gottdiener, & Haslam, 2013) were not present in the sample of people experiencing depression in this study. Moses (2010) in a study of predictors of self-stigma in adolescents with mental health problems, also found that causal beliefs were not significant predictors in a regression model. This suggests that variables other than causal beliefs contribute to self-stigma in clinical samples.

This study did not find that biological causal beliefs predict greater prognostic pessimism in the regression analysis. In support of this finding, a qualitative study by Ridge and Ziebland (2006) investigated a UK sample of 38 people recovering from depression and found that 'many' of the sample believed that a chemical imbalance caused their depression. Although these participants tended to define recovery as fixing the chemical imbalance with medication, it did not 'excuse the person from other efforts to bring about his or her recovery' (p. 1043). Biological narratives instead existed alongside of

consideration of psycho-social reasons for depression and use of non-biological treatments such as talking therapy. This suggests that in that study, belief in the biological model did not reduce self-efficacy, and thus increase pessimism about recovery.

However, it is interesting that despite similar study designs, these results differ from the results of Lebowitz et al. (2013), who did find that biological causal beliefs predicted prognostic pessimism in their sample. Participants in both studies were recruited online, had comparable mean scores on measures of depression and the same scale to assess prognostic pessimism was used. Differences could be accounted for by the different causal belief measures used, the nationalities of the participants and the sampling methods. Lebowitz et al. (2013) used the Amazon mTurk website (a website where workers are paid to complete small tasks online) in order to recruit participants, and concerns have been raised about the results of workers participating in conceptually or methodologically similar studies, or sharing information with each other through message boards (Chandler, Mueller & Paolacci, 2014). Prior knowledge about the purpose of the experiment can influence participant response (Brock & Becker, 1966; Edlund, Sagarin, Skowronski, Johnson, & Kutter 2009). People using Amazon mTurk are also already willing to complete online tasks, as they have signed up to do so. Participants in this study were recruited through social media and were not paid, meaning that they would have to have more motivation to complete the study, possibly resulting in different sample characteristics.

Due to the skew in the data for prognostic pessimism, it is necessary to look at the sampling method in this study. What is clear is that the sample in this study were very pessimistic about their chance of recovery from depression. Targeting online depression support groups as well as social media may have increased the likelihood of recruiting participants with different characteristics to other studies. One study of online support groups for people experiencing depression (Houston, Cooper & Ford, 2002) found that people who use online support groups were often socially isolated and had chronic depression. At a one-year follow up, only 33% of users had

'resolved' their depression. If the sample used in this study was similar, it could account for the pessimism seen within this sample. However, a review study found that overall the evidence of who uses these type of groups is mixed with differing results from poor quality research (Griffith, Calear, Banfield & Tam, 2009). Unfortunately, questionnaire burden prevented the researcher from adding in more measures. Having data on length of time of depression, or previous interventions for depression, might have helped to further explain the skew in the sample.

Limitations

This study had a majority of female, White British participants, and the average age was younger than the British average. The gender bias, however, does reflect the difference in reporting of depression in the general population (Kessler, 2002). Caution should thus be used in generalising the results of this study to men and ethnic minorities, especially as studies have found that beliefs can change according to ethnicity (Khalsa et al 2011). Using a Web based study meant that responses were limited to those people who use the Internet and are computer literate, which is more likely to be younger people (Ofcom, 2014).

This study asked for participants to take part if depression was their 'main mental health problem'. Co-occurrence of other mental health problems may have impacted on the results; when people have both depression and anxiety, for example, issues can be more chronic and have a poorer prognosis (Bakish, 1999). Co-occurrence of other mental health problems, as well as the relatively high average score on the measure of depression severity, may have been a factor in the skewing of the prognostic pessimism data.

This study used a measure of causal beliefs that does not separate out genetic or hereditary beliefs from other biologically based ones, for example, chemical imbalance. Genetic causes may be seen as more stable and more immune to change, which could encourage fatalism about recovery (Easter, 2012). Results may therefore have been different had genetic and other biological causes been made distinct from one another.

Future Directions

Researchers have argued that some aspects of stigma are difficult to measure with self-report questionnaires (Teachman, Wilson & Komarovskaya, 2006), stating instead that these components operate in an implicit manner that is not allied directly with conscious processes. Participants may not be aware of the stigma in the way that they could record it on measures, but it may 'show up' unconsciously in other ways. Explicit measures can be vulnerable to social desirability biases as well as being dependent on the participant's awareness of their own beliefs (Monteith & Petit, 2011). Rusch, Tod, Bodenhausen and Corrigan (2010) found that people with 'serious mental illness' who endorsed a genetic model of mental health problems had stronger implicit 'self-guilt' associations. As it is hypothesised that these implicit processes might respond in a different way to anti-stigma attempts (in the general public and amongst people with current mental health difficulties), it is important for research to address these to inform future anti-stigma campaigns (Lincoln, Arens, Berger and Reif, 2008; Stier and Hinshaw, 2007). Future research could address these implicit components of self-stigma with people experiencing depression using methods such as Implicit Association Tasks (Monteith & Petit, 2011).

As the data for one of the criterion variables, prognostic pessimism, was skewed, it could be that sampling or measurement problems have confounded the results. Replicating the research with another population of people with depression, for example, in an Improving Access to Psychological Therapies service and secondary mental health services in order to compare the findings would be helpful. Using a different measure of PP or one that has first been piloted on a similar UK population would be helpful in future research.

This research did not exclude people if they were experiencing other mental health problems such as anxiety. Doing so may have helped define the sample better and given a clearer explanation of the results, but may also have resulted in a sample that did not reflect the population (Hepgul et al.,

2016). In order to overcome this problem, as well as increase diversity within the sample, moving beyond diagnostic categories in similar research may be helpful. Especially as diagnostic categories have been shown to have poor reliability and validity (Boyle & Johnstone, 2016). Instead of operationalising problems using lists of symptoms, using measures of functioning or of wellbeing might result in more representative samples (Kinderman, Read, Moncrieff & Bentall, 2013). Participants could be defined according to their care needs e.g. a sample of people who use a Community Mental Health Team.

Implications for Clinical Psychology

Stigma is an important topic for Clinical Psychology as it remains a huge problem for people with mental health problems (Corrigan, 2005). Unfortunately, participants in this study reported high levels of self-stigma, irrespective of their causal beliefs about their own depression. Clinicians should be mindful that service users with more severe depression will possibly experience self-stigma to a higher degree, which might make it more difficult for them to seek help from services (Schomerus, Matschinger & Angermeyer, 2009).

Although the results of this study might suggest to the profession that the focus on 'causes' in anti-stigma campaigns or initiatives might not be very helpful for those who experience depression, as the findings are contrary to previous studies, more research needs to be undertaken to ascertain this with any certainty. Tentatively, it may be reassuring to the profession to know that the dominance of the medical model within some mental health services (Colomboa, Bendelowa, Fulforda & Williams, 2003) might not be associated with self-stigma in service users.

Conclusions

Depression severity and self-efficacy were significant predictors of self-stigma in this study. Causal beliefs had no significant effect on measures of self-stigma or prognostic pessimism in an online sample of people experiencing depression and living in the UK. A homogenised and skewed

sample may prevent generalisation of these findings. Further research is needed with similar clinical samples.

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Appendices
Appendix 1. Advert for study as posted on social media

Appendix 2. Participant information sheet and questionnaire pack

<u>Participant Information</u>

Hello, my name is Stephanie Davies and I am a Trainee Clinical Psychologist in South Staffordshire and Shropshire NHS Foundation Trust.

I would like to invite you to take part in some research. This research makes up part of a Doctorate Thesis for the Clinical Psychology Training at Staffordshire and Keele University. This research is supervised by Cailzie Dunn (Clinical Psychologist in Central Shrewsbury CMHT) and Dr Helen Scott (Senior Lecturer in Clinical Psychology and Supervisor at Staffordshire University).

Before you decide whether to participate, it is important for you to understand why the study is being conducted and what is involved. Please take the time to read the following information carefully, and discuss it with others if you wish.

We would like to learn about your beliefs about the causes of your depression

Most research in this area has been done with the general public, and little research has been done with people with depression in the UK....

There is evidence from the general public that some beliefs about the causes of mental health problems can increase stigma against people with those problems....

We are interested in finding out your beliefs about the causes of your depression and how they relate to how you feel about yourself and about the future course of your depression....

<u>You are eligible to take part if....</u>

- You are OVER 18

- You are living in the UK

- You can read and write in English

- You are currently feeling depressed

- Depression is currently your only or main mental health problem (for example, you might have some anxiety symptoms, but your depression is more severe).

What does this research involve?

This research involves filling in questionnaires, and will take about 15-20 minutes to complete...

Your information will remain completely anonymous!
We will not ask for your name or other identifying information.
This site does not collect any information from your computer that might identify you, like your IP address. During the collection, storage and publication of this data no-one but you would know that you have taken part in this study.

Please read the following information for all you need to know about this research...

Do I have to take part?

No. You are under no obligation to take part, and if you change your mind at a later date you can contact me to have your data removed from the study. You do not have to give a reason. However, please do this by February 2016. Once the results of the study have been published, it will be impossible to do so. However, so I know that I am removing YOUR information and not someone else's, I will ask you for a password (not your name or anything else that might identify you). You can then email me with this password and I can delete all your data.

How will my information be protected?

Data collected by the questionnaires in this study can only be accessed by the researcher. Your chosen password and email address (if you choose to add it) will be stored in a restricted- access database separate to the questionnaire data, and will not be used in the study. The anonymised data will be stored by the University for five years and then destroyed.

Are there any benefits to taking part in the study?

There will be no immediate direct benefit to you should you participate. However, this research will increase our understanding about the beliefs of people with depression, which could influence information giving about depression and mental health services in the NHS.

Are there any risks to taking part in the study?

Filling in the questionnaires will take up to 15-20 minutes of your time and answering some of these questions might highlight strong feelings of hopelessness. However, as this information is anonymous it will not be possible for anyone to contact you if you report these feelings. If you do feel low or hopeless or experience any suicidal thoughts, please refer to the sources of support below.

What should I do if I want to take part?

Please just continue onto the next question, where you will give your consent to take part in the study!

If anything is not clear, or if you would like more information about any aspect of the study, please contact Stephanie at beliefsresearch@mail.com.

Alternatively, if you prefer not to contact the researcher, please contact the Academic Supervisor of this study, Dr Helen Scott, at H.Scott@staffs.ac.uk

If you are reading this because you are feeling low or hopeless, or have suicidal thoughts, try to ask someone for help. Below are sources of support that you can access should you need to.

Helplines and support groups

www.samaritans.org (08457 90 90 90) operates a 24-hour service available every day of the year. If you prefer to write down how you are feeling, or if you are worried about being overheard on the phone, you can email Samaritans at jo@samaritans.org.

www.depressionalliance.org is a charity for people with depression. It does not have a helpline, but offers a wide range of useful resources and links to other relevant information.

www.studentdepression.org is a website for students who are depressed, have a low mood or are having suicidal thoughts.

www.thecalmzone.net is a resource for young men who are feeling unhappy. As well as the website, CALM also has a helpline (0800 58 58 58).

Do also contact your GP or services that you might already be involved with, as they can advise you about appropriate treatment. Your GP may be able to help you with access to talking therapies.

If you feel that you are in immediate risk, please do not complete this questionnaire. Instead seek help from one of the above sources.

Consent

Research title: *What do people experiencing depression believe are the causes of their depression?*

Name of researcher: *Stephanie Davies*

Please click ALL the answers below to take part in the study

Consent   Research title: What do people experiencing depression believe are the causes of their depression?   Name of researcher: Stephanie Davies   Please click ALL the answers below to take part in the study

❑   I can confirm that I have read the information sheet. I have had the opportunity to consider the information, ask questions and have had these questions answered satisfactorily (1)

❑   I understand that my participation is voluntary and that I am free to withdraw at any time without giving reason, without my medical care or legal rights being affected (2)

❑   I understand that all identifying information will remain anonymous and will be stored securely in keeping with Staffordshire University guidelines (3)

❑   I agree to take part in the above study (4)

In order to withdraw your information from the study, please enter a password below. If you then email me with this password (beliefsresearch@mail.com), I will delete all your information from the study. You can do this at any time until February 2016.

Demographic information. The information that you provide will enable us to to provide an accurate description of the sample. Please select the ONE response which is most descriptive of you Gender:

❍ Male (1)

❍ Female (2)

❍ Transgender (3)

Ethnicity: Please choose one option that best describes your ethnic group or background

❍ White British (1)

❍ White Irish (2)

❍ Gypsy or Irish Traveler (3)

❍ Any other White Background (4)

❍ White and Black Caribbean (5)

❍ White and Black African (6)

❍ White and Asian (7)

❍ Any other Mixed/Multiple ethic background (8)

❍ Indian (9)

❍ Pakistani (10)

❍ Bangladeshi (11)

❍ Chinese (12)

❍ Any other Asian Background (13)

❍ African (14)

❍ Caribbean (15)

❍ Any other Black/African/Caribbean background (16)

❍ Arab (17)

❍ Any other Ethnic Background (18)

Please enter your age

Q1 Thanks for that information! Over the last two weeks, how often have you been bothered by the following problems?

	Not at all (0)	Several days (1)	More than half the days (2)	Nearly every day (3)
1. Little interest or pleasure in doing things (1)	○	○	○	○
2. Feeling down, depressed or hopeless (2)	○	○	○	○
3. Trouble falling or staying asleep, or sleeping too much (3)	○	○	○	○
4. Feeling tired or having little energy (4)	○	○	○	○
5. Poor appetite or over eating (5)	○	○	○	○
6. Feeling bad about yourself, that you are a failure or that you have let yourself or your family down (6)	○	○	○	○
7. Trouble concentrating on things, such as reading the newspaper or watching television (7)	○	○	○	○
8. Moving or speaking so slowly that other people could have noticed? Or the opposite, being so fidgety or restless that you have been moving around a	○	○	○	○

lot more than
usual (8)

9. Thoughts that   ○   ○   ○   ○
you would be
better off dead or
of hurting
yourself in some
way (9)

Q2 Thank you! Please rate how much you agree with the following statements using the scale below

	Strongly Disagree (1)	Disagree (2)	Agree (3)	Strongly Agree (4)
1. I feel ashamed of myself for having psychological problems (1)	○	○	○	○
2. I feel inferior to others who don't have psychological problems (2)	○	○	○	○
3. My self-confidence is NOT threatened because I have psychological problems (3)	○	○	○	○
4. Because I have psychological problems, I cannot live a good, rewarding life (4)	○	○	○	○
5. I am disappointed in myself for having psychological problems (5)	○	○	○	○
6. I feel okay about myself for having psychological problems (6)	○	○	○	○
7. I feel that having psychological problems is a personal shortcoming for me (7)	○	○	○	○

Q3 Thank you! Please rate how much you agree with the following statements using the scale below

	Not at all true (1)	Barely true (2)	Moderately true (3)	Exactly true (4)
1. I can always manage to solve difficult problems if I try hard enough (1)	❍	❍	❍	❍
2. If someone opposes me, I can find means and ways to get what I want (2)	❍	❍	❍	❍
3. It is easy for me to stick to my aims and accomplish my goals (3)	❍	❍	❍	❍
4. I am confident that I could deal efficiently with unexpected events (4)	❍	❍	❍	❍
5. Thanks to me resourcefulness, I know how to handle unforseen situations (5)	❍	❍	❍	❍
6. I can solve most problems if I invest the necessary effort (6)	❍	❍	❍	❍
7. I can remain calm when facing difficulties because I can rely on my coping abilities (7)	❍	❍	❍	❍

8. When confronted with a problem, I can usually find several solutions (8)	○	○	○	○
9. If I am in a bind, I can usually think of something to do (9)	○	○	○	○
10. No matter what comes my way, I am usually able to handle it (10)	○	○	○	○

Q4 Thank you! This next questionnaire presents you with a number of reasons why you might be depressed. Each reason is given as a statement in the form of, 'I am depressed because..' followed by a specific reason. For each statement consider whether or not this particular reason causes you to be depressed.   Rate each reason using the scale. 'I am depressed because...'

	Definitely not a reason (1)	Probably not a reason (2)	Probably a reason (3)	Definitely a reason (4)
I see the world the way it really is (1)	○	○	○	○
I can't accomplish what I want to do (2)	○	○	○	○
I don't feel loved (3)	○	○	○	○
That's just the type of person I am (4)	○	○	○	○
No one really cares about me (5)	○	○	○	○
I can't decide what to do with my life (6)	○	○	○	○
This is the way I've learned to be (7)	○	○	○	○
I haven't resolved some issues with my family (8)	○	○	○	○
I think about things in a depressing way (9)	○	○	○	○
No one really understands me (10)	○	○	○	○
My family treated me poorly as a child (11)	○	○	○	○

My spouse/partner treats me poorly (12)	○	○	○	○
I have not become the person I set out to be (13)	○	○	○	○
Other people isolate me (14)	○	○	○	○
Of certain things that happened to me as child (15)	○	○	○	○
I haven't done anything important in my life (16)	○	○	○	○
Other people criticise me (17)	○	○	○	○
I'm not living up to my personal standards (18)	○	○	○	○
I choose to be depresed (19)	○	○	○	○
I haven't worked through things that happened to me as a child (20)	○	○	○	○
There is no-one to share my innermost thoughts and feelings with (21)	○	○	○	○
I had a difficult childhood (22)	○	○	○	○
I'm not active enough (23)	○	○	○	○
I don't take care of myself physically (24)	○	○	○	○

Q4 cont Almost finished now.. This questionnaire continues with more reasons why you might be depressed. Each reason is given as a statement in the form of, 'I am depressed because..' followed by a specific reason. For each statement consider whether or not this particular reason causes you to be depressed.   Rate each reason using the scale. 'I am depressed because...'

	Definitely not a reason (1)	Probably not a reason (2)	Probably a reason (3)	Definitely a reason (4)
I have a chemical imbalance (1)	❍	❍	❍	❍
I am a pessimist (2)	❍	❍	❍	❍
I inherited it from my parents (3)	❍	❍	❍	❍
It's a biological illness (4)	❍	❍	❍	❍
I don't eat well enough (5)	❍	❍	❍	❍
I am not fulfilling my potential (6)	❍	❍	❍	❍
Other people don't like me (7)	❍	❍	❍	❍
I don't know who I am or what I stand for (8)	❍	❍	❍	❍
I don't get enough exercise (9)	❍	❍	❍	❍
I have always been this way (10)	❍	❍	❍	❍
My nervous system is just wired this way (11)	❍	❍	❍	❍
I've failed to achieve a specific goal I set for myself (12)	❍	❍	❍	❍

I can't make friends (13)	○	○	○	○
I can't get done the things I should be able to (14)	○	○	○	○
I have no set goals in my life (15)	○	○	○	○
People treat me poorly (16)	○	○	○	○
People don't give me the respect I deserve (17)	○	○	○	○
This is the way I respond when things get tough (18)	○	○	○	○
It's basically caused by genetics (19)	○	○	○	○
I'm stuck where I am in life, nothing ever changes (20)	○	○	○	○
I pay more attention to the bad things in life than the good things (21)	○	○	○	○
I'm stuck in a bad marriage or love relationship (22)	○	○	○	○
My spouse/partner doesn't understand me (23)	○	○	○	○
I'm not good at expressing my innermost	○	○	○	○

feelings (24)

Q5 This is the very last question!   How long do you think that you will continue to feel depressed? Please choose one answer

○ Less than 1 week (1)

○ 1 to 2 weeks (2)

○ 2 to 4 weeks (3)

○ 1 month to 6 months (4)

○ 6 months to 1 year (5)

○ More than 1 year, but not indefinitely (6)

○ Indefinitely (7)

○

End of questionnaires!   Thank you so much for taking the time to take part in the study. If you would like to find out the results of the study once it is completed, please type your email address below (this will be stored separately to your answers).   Remember- if you would like to withdraw your data from the study (before February 2016) please email me with your password at beliefsresearch@mail.com

        I hope that completing these questionnaire was not emotionally difficult for you. However, as this information is anonymous it will not be possible for anyone to contact you if you report feelings of hopelessness or if you are feeling suicidal. If you do feel that way, please refer to the sources of support below;

If you are reading this because you are feeling low or hopeless, or have suicidal thoughts, try to ask someone for help. Below are sources of support that you can access should you need to.

Helplines and support groups

www.samaritans.org (08457 90 90 90) operates a 24-hour service available every day of the year. If you prefer to write down how you are feeling, or if you are worried about being overheard on the phone, you can email Samaritans at jo@samaritans.org.

www.depressionalliance.org is a charity for people with depression. It does not have a helpline, but offers a wide range of useful resources and links to other relevant information.

www.studentdepression.org is a website for students who are depressed, have a low mood or are having suicidal thoughts.

www.thecalmzone.net is a resource for young men who are feeling unhappy. As well as the website, CALM also has a helpline (0800 58 58 58).

Do also contact your GP or services that you might already be involved with, as they can advise you

about appropriate treatment. Your GP may be able to help you with access to talking therapies.

If you feel that you are in immediate risk, please do not complete this questionnaire. Instead seek help from one of the above sources.

Appendix 3. Ethical approval for study

STAFFORDSHIRE
UNIVERSITY

Faculty of Health Sciences

ETHICAL APPROVAL FEEDBACK

Researcher name:	Stephanie Davies
Title of Study:	Relationships between causal beliefs, self-stigma and prognostic pessimism in people with depression
Award Pathway:	DClinPsy
Status of approval:	Amendment approved

Dear Stephanie,

Many thanks for your resubmitted form and your letter detailing how you have addressed the committee's comments.

I am pleased to say that you have addressed the comments in full and we can approve your ethics application.

Action now needed:

Your amendment has now been approved by the Faculty's Ethics Panel.

You should note that any divergence from the approved procedures and research method will invalidate any insurance and liability cover from the University. You should, therefore, notify the Panel of any significant divergence from this approved proposal.

You should arrange to meet with your supervisor for support during the process of completing your study and writing your dissertation.

Please note that when your research is complete you will need to submit a completion of research email (details can be found on the ethics BlackBoard site).

We wish you well with your study.

Karen Rodham

Signed: Prof Karen Rodham Chair of the Faculty of Health Sciences Ethics Panel	Date: 5th August 2015

Appendix 4. RFD data and comparisons with previous studies

Belief	Current study, clinical sample n=184	Thwaites et al (2004) clinical sample n=123	Addis et al (1995)
Characterological	.78 (.73- .82)	.88 (.77- .95)	.86
Achievement	.87 (.84- .89)	.80 (.55- .95)	.85
Intimacy	.78 (.72- .82)	.76 (.43- .96)	.79
Childhood	.87 (.85- .90)	.90 (.76- .98)	.84
Existential	.82 (.78- .86)	.79 (.50- .96)	.78
Relationship	.88 (.84- .90)	.83 (.48- .91)	.82
Interpersonal conflict	.87 (.84- .90)	.86 (.69- .97)	.85
Physical	.87 (.84- .90)	.79 (.44- .97)	.79
Biological	.71 (.63- .77)	.80 (.47- .97)	n/a

As seen above, Cronbach Alphas for each sub-scale were similar to Thwaites et al (2004), most being near or over 0.8, showing an acceptable level of internal consistency, as well as having narrower confidence intervals. However, the internal consistency of the 'Biological' subscale is lower than in the previous study, with a range starting at .63.

PHQ9 scores also significantly correlated with Existential, Interpersonal, Intimacy, Achievement, Childhood and Physical reasons for depression. In the original American sample, there was no correlation between depression scores on the Beck Depression Inventory (BDI, Beck) and the subscales on the RFD. However, Thwaites et al (2004) did find significant positive correlations between the BDI and four of the subscales (Physical, Existential, Intimacy and Achievement) in a British sample. Thwaites et al. (2004) considered that due to these correlations being between 0.4-0.6, it did not mean that the 'reasons offered for depression are synonymous with levels of depression'. As the correlations between the levels of depression measure and the subscales of the RFD were all less than 0.4 in this sample, the same can be assumed.

Appendix 5. Violation of normality of PP

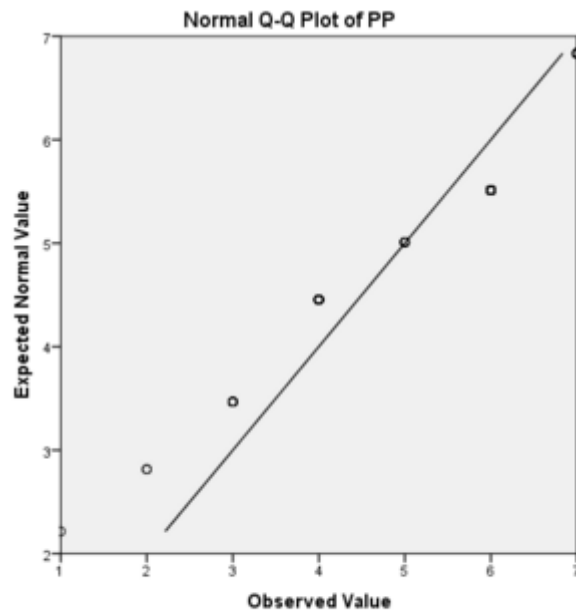Figure 1. Q-Q plot demonstrating violation of normality in PP

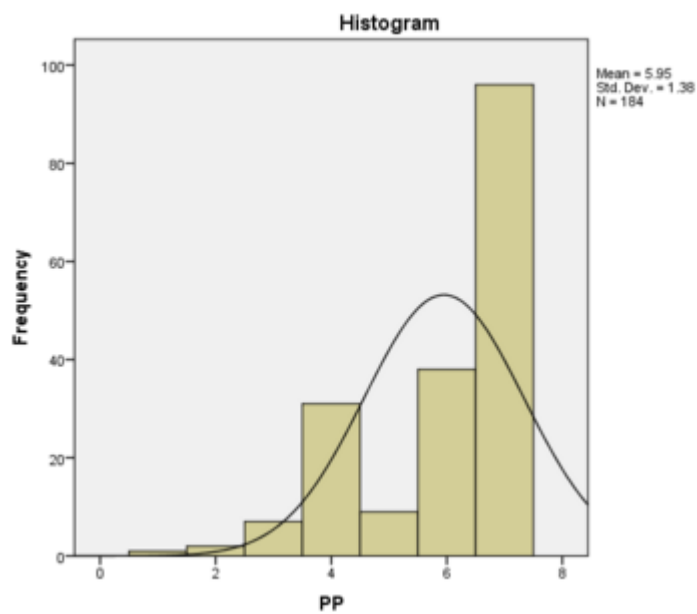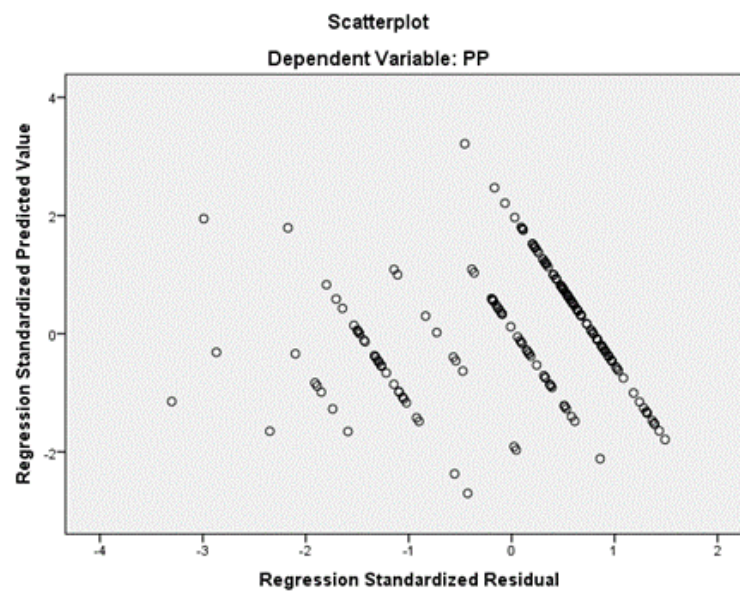Figure 2. Histogram demonstrating sample skew in PP

Figure 3. Scatterplot demonstrating violation of homoscedasticity and linearity

Scatterplot
Dependent Variable: PP

Appendix 6. Journal submission guidelines

Author Guidelines

The British Journal of Clinical Psychology publishes original contributions to scientific knowledge in clinical psychology. This includes descriptive comparisons, as well as studies of the assessment, aetiology and treatment of people with a wide range of psychological problems in all age groups and settings. The level of analysis of studies ranges from biological influences on individual behaviour through to studies of psychological interventions and treatments on individuals, dyads, families and groups, to investigations of the relationships between explicitly social and psychological levels of analysis.

All papers published in The British Journal of Clinical Psychology are eligible for Panel A: Psychology, Psychiatry and Neuroscience in the Research Excellence Framework (REF).

The following types of paper are invited:

• Papers reporting original empirical investigations

• Theoretical papers, provided that these are sufficiently related to the empirical data

• Review articles which need not be exhaustive but which should give an interpretation of the state of the research in a given field and, where appropriate, identify its clinical implications

• Brief reports and comments

1. Circulation

The circulation of the Journal is worldwide. Papers are invited and encouraged from authors throughout the world.

2. Length

The word limit for papers submitted for consideration to BJCP is 5000 words and any papers that are over this word limit will be returned to the authors. The word limit does not include the abstract, reference list, figures, or tables. Appendices however are included in the word limit. The Editors retain discretion to publish papers beyond this length in cases where the clear and concise expression of the scientific content requires greater length. In such a case, the authors should contact the Editors before submission of the paper.

3. Submission and reviewing

All manuscripts must be submitted via Editorial Manager. The Journal operates a policy of anonymous (double blind) peer review. We also operate a triage process in which submissions that are out of scope or otherwise inappropriate will be rejected by the editors without external peer review to avoid unnecessary delays. Before submitting, please read the terms and conditions of submission and the declaration of competing interests. You may also like to use the Submission Checklist to help you prepare your paper.
4. Manuscript requirements

• Contributions must be typed in double spacing with wide margins. All sheets must be numbered.

• Manuscripts should be preceded by a title page which includes a full list of authors and their affiliations, as well as the corresponding author's contact details. A template can be downloaded from here.
• The main document must be anonymous. Please do not mention the authors' names or affiliations (including in the Method section) and refer to any previous work in the third person.

• Tables should be typed in double spacing, each on a separate page with a self-explanatory title. Tables should be comprehensible without reference to the text. They should be placed at the end of the manuscript but they must be mentioned in the text.

• Figures can be included at the end of the document or attached as separate files, carefully labelled in initial capital/lower case lettering with symbols in a form consistent with text use. Unnecessary background patterns, lines and shading should be avoided. Captions should be listed on a separate sheet. The resolution of digital images must be at least 300 dpi. All figures must be mentioned in the text.

• All papers must include a structured abstract of up to 250 words under the headings: Objectives, Methods, Results, Conclusions. Articles which report original scientific research should also include a heading 'Design' before 'Methods'. The 'Methods' section for systematic reviews and theoretical papers should include, as a minimum, a description of the methods the author(s) used to access the literature they drew upon. That is, the abstract should summarize the databases that were consulted and the search terms that were used.

• All Articles must include Practitioner Points – these are 2–4 bullet points to detail the positive clinical implications of the work, with a further 2–4 bullet points outlining cautions or limitations of the study. They should be placed below the abstract, with the heading 'Practitioner Points'.

• For reference citations, please use APA style. Particular care should be taken to ensure that references are accurate and complete. Give all journal titles in full and provide DOI numbers where possible for journal articles.

• SI units must be used for all measurements, rounded off to practical values if appropriate, with the imperial equivalent in parentheses.

• In normal circumstances, effect size should be incorporated.

• Authors are requested to avoid the use of sexist language.

• Authors are responsible for acquiring written permission to publish lengthy quotations, illustrations, etc. for which they do not own copyright. For guidelines on editorial style, please consult the APA Publication Manual published by the American Psychological Association.

5. Brief reports and comments

These allow publication of research studies and theoretical, critical or review comments with an essential contribution to make. They should be limited to 2000 words, including references. The abstract should not exceed 120 words and should be structured under these headings: Objective, Method, Results, Conclusions. There should be no more than one table or figure, which should only be included if it conveys information more efficiently than the text. Title, author name and address are not included in the word limit.

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9. Pre-submission English-language editing

Authors for whom English is a second language may choose to have their manuscript professionally edited before submission to improve the English. A list of independent suppliers of editing services can be found at http://authorservices.wiley.com/bauthor/english_language.asp. All services are paid for and arranged by the author, and use of one of these services does not guarantee acceptance or preference for publication.

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11. The Later Stages

The corresponding author will receive an email alert containing a link to a web site. A working e-mail address must therefore be provided for the corresponding author. The proof can be downloaded as a PDF (portable document format) file from this site. Acrobat Reader will be required in order to read this file. This software can be downloaded (free of charge) from the following web site: http://www.adobe.com/products/acrobat/readstep2.html.

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12. Early View

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Further information about the process of peer review and production can be found in this document: What happens to my paper?

Appendix 7. SPSS output for regression model for dependent variable "Self-efficacy" with only significant predictors entered

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.555[a]	.308	.300	3.347

a. Predictors: (Constant), SETOT, PHq9TOT

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	901.561	2	450.780	40.232	.000[b]
	Residual	2027.999	181	11.204		
	Total	2929.560	183			

a. Dependent Variable: SSTOT

b. Predictors: (Constant), SETOT, PHq9TOT

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	19.421	1.545		12.573	.000
	PHq9TOT	.291	.045	.430	6.491	.000
	SETOT	-.165	.048	-.229	-3.458	.001

a. Dependent Variable: SSTOT

Beliefs about the causes of depression: Author's reflections

(2796 words)

Contents

Abstract

This section contains discussion of reflections on conducting this thesis. Reflection aids learning and self-development (Boyd & Fales, 1983) by identifying areas of concern, openness to new information from different sources, and identifying a changed perspective. Atkins and Murphy (1994) suggest that in order to make a difference to practice, reflection must be followed by commitment to action. This paper therefore includes discussion of the parts the research process which were particularly difficult or positive, re-evaluation of these areas, how my ideas about research or psychology have developed from this process and how I would do things differently in future. These issues are discussed in the hope that it will aid any further research undertaken and improve my abilities as a researcher. This paper is written in the first person, given it relates to my personal thoughts and experiences. As I have already addressed the methodological limitations and generalisability of my findings, they are not included in this section.

Epistemological position

Beliefs about what constitutes valid knowledge and how we can obtain it (epistemological position) have been shown to play a role in how students approach and process new information (Chin & Brewer, 1993; Pintrich et al., 1993). Therefore, it is important to report how my epistemological position may have affected the research process and how my position may have changed as a result of doing this thesis.

My research paper used quantitative methods, which is traditionally a 'positivist' position; that there is objective knowledge in the world that can be captured using scientific methods (Tolman, 1992). At the time of developing my research ideas, I was not at the stage of considering my own 'epistemological position' and chose this method due to it fitting my research question. However, I do not consider myself a positivist as I do not believe that it is fully possible to convert subjective human experience into objective variables without the values, meaning and bias of the researcher and the

societal context being involved. In that way my epistemological position can be thought of as post-positivist; I consider it important to place this research project in context, and not consider it one overall truth. I am probably now more critical of the positivist position due to the difficulties I had in using the approach, but I still consider quantitative research useful when considered as part of a bigger picture. I find it comfortable to do so because it is what I do every day in practice. I use a variety of methods to gain knowledge which I consider to be valid when taken in conjunction; research evidence, supervision, experiences in clinical practice and feelings that arise in sessions with clients (Jones & Mehr, 2007). Evidence supports this idea that epistemological positions can be multi-dimensional and multi-layered (Buehl & Alexander, 2001; Frost & Nolas, 2011). My reflections in this paper are therefore from this multi-layered viewpoint.

Topic and method choice

I knew that I wanted to focus on causal beliefs from an early stage of the research process; my first placement was split between a hospital setting where the medical model of mental health dominated the team, and a CMHT which took a much more critical stance to the diagnosis and treatment of people's distress. Working in such different setting prompted discussions with both supervisors about the possible ramifications on each belief system, and the possible effect on service users. I also worked with service users who believed that their problems were all biologically based, and experienced first-hand the difficulties in thinking psychologically with them when this was the basis of their understanding. In reading, I came across Dorothy Rowe's 'Depression: The way out of your prison' (1983) whose ideas about depression seemed to me to be insightful and useful, but not widely disseminated. I wonder now if one of the reasons for this is because her ideas developed solely out of clinical practice and were popular after initial publication due to 'grassroots, word-of-mouth' publicity. Knowledge gained from positivist methods might be assumed to more rigorous and scientific and so more reliable (Slade & Priebe, 2001) and I wondered if she

was able to test her ideas with validated measures and scientific methods, her work would be more widely known within mental health services.

I decided on a quantitative approach after reading in the area and discovering similar research that had used these methods. When it came to narrowing down to a research question the one I had in mind was not exploratory and so did not suit qualitative methods. I had also not got to the stage in my study where I considered issues like epistemological positions or the drawbacks of positivism in psychology.

Setting

Originally, as I lean towards a more critical stance in regards to the causation and diagnosis of mental health problems, I wanted to use parameters other than diagnosis to define my sample. The initial idea was to use a sample of people who were under the care of the CMHT regardless of 'diagnosis'; this sample could be defined by their care needs instead. In order to assess how suitable this setting would be to conduct the research in, I met with a group of service users from the CMHT. They said although the research seemed needed and valuable, they did not feel that it was appropriate to ask people about their causal beliefs. In fact, they said questionnaires that measured causal beliefs could be 'triggering' for people with complex trauma, so many service users might not consent to take part in the study. They also said that they doubted service users would fill in the amount of questionnaires that were required. Another issue is that the participants would all have different diagnoses and as different amounts of stigma can be attached to different diagnoses (Gaebel, Zaske & Baumann, 2006), this might be a large confounding variable.

It seemed that the sample would have to be defined by their mental health problem, and one which was not severe and enduring. People experiencing depression were chosen as the sample instead. However, as service users within a CMHT would have more severe depression, using this population would not create a representative sample. In addition, if participants were found in primary care service, such as Increasing Access

to Psychological Therapies, it was thought that these participants might endorse only psychological reasons for depression, and would not be a representative sample of all people with depression.

It has occurred to me whilst writing this paper that I did not consider using a sample of service users with depression to consult with them about the research. I wonder if at the time I felt under time pressure to make decisions about the research, or whether I was unsure how I would go about this. As my initial consultation with service users was helpful in making big decisions about the research, it would have been appropriate to do another consultation.

Measures

One of the biggest challenges for me in this research project was choosing appropriate measures to use. In order to fully reflect on why this was, I will expand more on measurement in clinical psychology in general in this section.

Clinical psychology is concerned with people's problems, and often to research people's problems in a quantitative manner, we re-define problems in such a way so that they can be turned into variables (Stam, 2004). 'Self-stigma', is a not a tangible thing which exists inside of people, instead it is a 'functional description of a property' which can be measured (Stam, 2004, p.1261) This is done via a process of reduction, in which a concept or theory is reduced into a measurable entity, so it can be investigated. However, I had the following issues with choosing a measure for self-stigma (these also apply to the other measures used);

1. There are many different measures measuring the same construct, which are all different.

2. Other constructs overlap with the construct to be measured (e.g. self-blame and self-stigma).

3. Some measures included other and similar constructs whilst other measures separated them out (e.g. 'perceived stigma' as a part of 'self-stigma').

4. The construct can be different in different populations e.g. the measures that look at self-stigma in populations of people with 'severe and enduring' mental health problems might not be suitable for a sample with depression.

So, it would appear that we do not all agree on what 'self-stigma' is, how it is defined, how it should be measured, or what it should be called. This shows one of the issues with using positivist methods in psychology; that it is very hard to ignore how societies' values impact on how we define constructs and how we measure them. Often a term or construct is so mainstream in discourse that it is difficult to even begin to question it (e.g. 'depression'). However, sometimes it is more obvious. In looking for a measure of 'self-efficacy' I found a measure that at first seemed suitable for my sample- 'Depression Coping Self-Efficacy Scale' (Perraud, 2000). Items are based on what research says helps people with depression manage their symptoms, as well as nurse's opinions, a construct that they called 'coping behaviour' (users rate how confident they are in doing things that would reduce their symptoms of depression). One of the items was 'take medication the way my Doctor recommends'. This reflects the medical view that an appropriate way to manage mood is with medication. Even the items which were more psychosocial in nature, 'get together with at least one very close person when I am feeling lonely', seem to ignore any social conditions that a person might exist in irrespective of their depression. A person may not be confident to do this because they live alone, or do not have anyone they are 'very close' to.

I also recognise that my own values came into choosing measures. At first, I wanted to only investigate the effects of biological causal beliefs. After discussion with my supervisor, we decided that I should really look at a range of causal beliefs. My role as a psychologist and more personal views had maybe biased me to assume that the only possible effects of more psychosocial beliefs were positive.

In selecting the measures I eventually chose for this research project, I used all the positivist traditions of selecting ones which had statistics which indicated that they were reliable and valid, that they had been normed on similar samples to my own, and that they were used in similar research (Boynton & Greenhalgh, 2004). It is also the case that constructs do not arise out of nowhere, and are based on other forms of knowledge which I consider valid e.g. clinicians experience or qualitative inquiry. From my literature review, I was aware that the causal belief measure was an area that previous researchers had fallen down on, choosing measures that were not appropriate or did not reflect the range of beliefs available. It highlighted to me the value in not just reading previous research, but also critiquing it so you can learn whether their measures were valid or not. Researchers are not required to give reasons for their measure choice, but some did. This was very useful in thinking about whether the measure they had used would be suitable for my research. On this course we conduct the empirical research and literature review simultaneously, but in the future I would do this in sequence. This would mean that I could thoroughly review the papers and the measures that they used before choosing measures for my own.

Recruitment

Recruitment was easier than expected. I joined 'Twitter' a few months prior to recruitment in order to build up followers and identify potential users who might 're-tweet' my research and help me gain a wider audience. I had the most success tweeting people who were well-known and had many followers. Matt Haig, author of 'Reasons to stay alive' (2015), an autobiographical account of his experience with depression, re-tweeted my research. As Alistair Campbell had recently visited the University and sat in during one of our lectures, I tweeted him as well. Although he did not re-tweet my research, many of his followers did. It only took about four days altogether to reach an acceptable number of participants. Many people did not re-tweet my research when asked, including one of my favourite authors. Having people ignore you on social media can actually be detrimental (Tobin,

Vanman & Verreynne, 2014) and I did feel embarrassed and slightly disillusioned when I did not get the re-tweet I was expecting.

My good experience with online recruitment means I would definitely use it again to recruit participants. However, although I targeted some online support groups on Facebook for men and minorities, I did not achieve a balanced sample. From undertaking my literature review, I am aware that this is an issue for many researchers seeking to recruit clinical samples. It has helped me to reflect critically on how we define depression; if depression is conceived as a 'mental health problem' that exists within the sufferer (Pilgrim, 2007) and can affect anyone, yet is present more often in women than men to services (Kuehner, 2003), then how can we be sure that the way we conceive it as a 'mental health problem' is even correct? This has led me to further reading not only on the problems with diagnosis, but the issues with the concept of depression entirely. For example, I learnt that some researchers have argued that as women are the majority of people who experience depression, the conceptualisation of depression as a 'mental health problem' or 'disorder', might instead be women's natural response to an unequal and gendered society (Ussher, 2010). Or that the PHQ9 (Kroenke & Spitzer, 2002) or other measures of depression are nothing but a 'professional reification about human misery, not a fact' (Pilgrim & Bentall, 1999, p.271). Reading about and reflecting upon these ideas is important to me as I am contributing to discourse around depression with this research project; using the term 'depression' in this research project, using a measure to assess its symptoms and reporting my findings, will have added to the legitimacy of the term. At the same time, I cannot ignore all research pertaining to the incidence of a certain type of distress that has specific symptoms and all my clinical experience of working with clients with similar characteristics. Taking a middle position in this regard has been helpful (Busfield, 1996).

Data analysis and results

During data analysis, I was eager to discover whether my hypotheses were reflected in the results. When I did not find a significant effect of causal

beliefs, I was disappointed and had the impulse to try and find some other kind of interesting effect, so as not to 'waste' the data. However, I have been reminded by my supervisor and others that papers that do not find the expected results are vital to the evidence base as building theories requires replication (Ferguson & Heene, 2012). I can now understand why researchers might not submit their results when they conflict with ideas that they are very attached to (Coursol & Wagner, 1986). I also worry that not finding the expected results will make it difficult for me to get this research published due to the publication bias identified in psychology (Levine, Asada & Carpenter, 2009). It also makes me wonder how many other students or researchers have undertaken similar research to mine but not had it published, or how many other students will in the future. It reinforces to me the importance of seeking out grey literature. As my choice to undertake this research project was based on published evidence, it makes me wonder whether I would have made different choices if publication bias did not exit. As evidence grows for negative associations of biological causal beliefs, it might make it even more difficult for research opposing it to be published (Fanelli, 2010).

Conclusion

Although my epistemological position still allows valid knowledge to be gained from quantitative studies, after conducting my own research project, I am more aware of the negative aspects of the positivist approach. Defining and measuring concepts, as well as grouping samples according to diagnosis or symptoms, is much more complicated than I previously thought. When I conduct research in the future, I will probably explore other methods as well as quantitative.

Reflecting upon this paper and thinking about my epistemological position has been valuable, as it has forced me to organise my thoughts on a complicated issue in a coherent way, which is not always easy for me. It has helped me take a step back and view this three-year process as a whole, and to acknowledge the changes in my stance during this time.

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