THE UNIVERSITY OF QUEENSLAND

AUSTRALIA

Depression explanatory models of primary care patients and doctors: Relationship with self-stigma, communication effectiveness, and recovery

Jen Lee Teh
Bachelor of Science (Conservation & Resource Studies)
Master of Communication (Communication for Social Change)

A thesis submitted for the degree of Doctor of Philosophy at
The University of Queensland in 2017
School of Psychology

Abstract

This thesis examines the role of patient and doctor perspectives in primary care depression management. The analytical term chosen to explore these perspectives is Kleinman's explanatory model (EM), defined as "notions about an episode of sickness and its treatment that are employed by all those engaged in the clinical process" (1980, p. 105). This research is exploratory and focuses mainly on patients, with input from a small number of general practitioners (GPs).

I examine self- stigma in depression patients because there has been an attempt in recent years to reduce stigma by describing depression as a medical condition like diabetes. This may have decreased some public stigma, but the association between biomedical explanations for depression and patient self-stigma has not been studied in-depth. Self-stigma is the internalisation of negative stereotypes and prejudices about having mental illness and it can inhibit help-seeking and outcomes more than public stigma. Another factor that can influence depression outcomes is the therapeutic relationship between patient and their doctor. Studies report empathy and good communication as characteristics of caring patient-doctor relationships. Mutual understanding of patient and doctor perspectives could help foster empathy and effective communication, which is framed in this thesis using communication accommodation theory (CAT: Gallois, Ogay, & Giles, 2005; Giles, 1973). My research questions are:

RQ1. What are depression EMs of primary care patients and do these change over time?

RQ2. Does high endorsement of biomedical causation for depression correlate with low patient self-stigma?

RQ3. Does agreement to certain aspects of patient EMs correlate with patient perception of greater GP-patient communication effectiveness?

RQ4. How aware are GPs of their patient's depression EM, how concordant are GP EMs with patient EMs, and how does awareness and/or concordance correlate to depression outcomes?

Two different groups of participants were recruited by convenience sampling – the first consisted individuals aged 18-65 who had sought primary care treatment for depression. I collected longitudinal data using two online surveys spaced six months apart (Time 1 n = 238; Time 2 n = 129). A subset of patients (n = 10) who completed the later survey was interviewed between August 2015 and January 2016. The second group comprised GPs (n = 29) whose details were provided by individuals from the patient survey. The GPs were surveyed once. Ten additional GPs were recruited independently for interviews on EMs between April 2015 and January 2016.

The patient survey contained validated instruments on four areas: patient EM, self-stigma levels, GP-patient communication effectiveness, and symptoms. EM beliefs were measured with Haidet et al.'s (2008) CONNECT instrument modified for this study. It consisted 16 items on causation, internal locus of control, sense of agency, efficacy of non-medical therapies, significance of depression, and preference for GP-patient cooperation. The GPs were surveyed with a version of this instrument on their perception of their patient's EM (to quantify awareness) and the EM they adopt for their patient's depression (to quantify concordance). Patient self-stigma was measured using the Internalised Stigma of Mental Illness scale modified for this study, with items on alienation, discrimination experience, help-seeking inhibition, and social withdrawal. A CAT-based instrument, consisting items for emotional expression, discourse management, interpretability, and interpersonal control, measured communication effectiveness. Participants indicated agreement to instrument items using a Likert-type scale ranging from 1 (*strongly disagree*) to 6 (*strongly agree*). The Clinically Useful Depression Outcomes Scale (CUDOS: Zimmerman, Chelminski, McGlinchey, & Posternal, 2008) was used to track depression symptoms. Patients rated how well the items (e.g., My energy level was low) described them in the past week using a scale of 0 (*Not at all true*) to 4 (*Almost always true*).

Quantitative analysis methods mainly consisted of non-parametric tests for differences between groups and strength of correlations. Interviews were analysed manually and with textual analysis software Leximancer.

RQ1 results showed that most participants had a strong or moderate belief about whether biomedical and/or psychosocial factors caused their depression, and most EM beliefs did not change over time. RQ2 results indicated that biomedical belief was associated with lower help-seeking inhibition but also greater alienation. RQ3 results highlighted that endorsement of GP-patient cooperation was strongly positively correlated with communication effectiveness. Patient interviews yielded exemplars to illustrate these findings. RQ4 results from a paired study (n = 29) suggest that the GPs were aware of and concordant with their patient's EMs. Awareness appears to be more important in determining recovery than concordance. Incidentally, GP interviews showed that doctors mostly accorded importance to knowing their patient's EM but concordance was seen as less crucial.

This thesis gives new understanding about depression EM beliefs that are associated with lower patient self-stigma and more effective GP-patient communication. Results from this research on awareness-concordance were not generalisable due to the small sample size, but future work with larger samples will undoubtedly yield more insights into the importance of EMs in primary care depression management.

Declaration by author

This thesis is composed of my original work, and contains no material previously published or written by another person except where due reference has been made in the text. I have clearly stated the contribution by others to jointly-authored works that I have included in my thesis.

I have clearly stated the contribution of others to my thesis as a whole, including statistical assistance, survey design, data analysis, significant technical procedures, professional editorial advice, and any other original research work used or reported in my thesis. The content of my thesis is the result of work I have carried out since the commencement of my research higher degree candidature and does not include a substantial part of work that has been submitted to qualify for the award of any other degree or diploma in any university or other tertiary institution. I have clearly stated which parts of my thesis, if any, have been submitted to qualify for another award.

I acknowledge that an electronic copy of my thesis must be lodged with the University Library and, subject to the policy and procedures of The University of Queensland, the thesis be made available for research and study in accordance with the Copyright Act 1968 unless a period of embargo has been approved by the Dean of the Graduate School.

I acknowledge that copyright of all material contained in my thesis resides with the copyright holder(s) of that material. Where appropriate I have obtained copyright permission from the copyright holder to reproduce material in this thesis.

Publications during candidature

None.

Publications included in this thesis

No publications included.

Contributions by others to the thesis

Dr Bernadette Watson and Dr David King, my two doctoral advisors, provided feedback on drafts, recommended literature references, advised on study design, and gave input for statistical analysis.

Statement of parts of the thesis submitted to qualify for the award of another degree

None.

Acknowledgements

I want to firstly thank God for seeing me through this PhD process – truly He has provided all I need, especially through the following people to whom I remain ever grateful. They are:

- my parents who were supportive and willing to spend their hard-earned savings on my school fees (Thank you Dad and Mum for believing in me that I could undertake a stressful academic journey even after being diagnosed with a stress-related disorder.)
- my advisors Dr Bernadette Watson and Dr David King who went absolutely above and beyond their duty in so many varied ways
- my sisters who have been an amazing source of support and practical help
- my boyfriend Ben prayed for me, drove me around to clinics to recruit doctors, and inspired me with the hard work he puts in for his degree
- my spiritual family and friends

Special mention also to the following:

- Dr Geoffrey Watson for helping with early analysis of my data
- Dr Yvonne Ho for being my statistics consultant in the final data analysis
- Fellow PhD students who lent a hand in piloting, using Qualtrics, writing advice, and so forth: Dr Danielle Ferndale, Dr Gabrielle Stevens, and Dr Jennifer Setchell
- Dr John Bennett and Dr Amy Kwan who helped me care for myself
- Dr Nancy Sturman, Dr Helen Kerr, and Dr Tony Arklay for useful input on GP-related aspects of my studies, and Dr Rick Sapsford and Dr Ian Williams for facilitating GP recruitment presentations
- Caitlin Sparkes for help with references.

Last but definitely not least, I want to thank my study participants. To the doctors who took time to help in research, this tells me that you always want to be a better doctor for your patients, which is so heartening. To the most important people of all, the individuals with depression, without whom there would be no thesis to speak of. I salute you for wanting to do your part to help improve depression management for others.

This thesis is dedicated to every person who has ever struggled with depression or any other mental illness. It is my prayer that whatever lens you use to look at your struggles, you will always remember to see yourself through God’s eyes. In His eyes, you are deeply, unconditionally, loved.

Jen Lee

Keywords

depression, explanatory model, patient, general practitioner, communication, self-stigma, primary care, mental health

Australian and New Zealand Standard Research Classifications (ANZSRC)

ANZSRC code: 170113, Social and Community Psychology, 60%
ANZSRC code: 111714, Mental Health, 20%
ANZSRC code: 111717, Primary Health Care, 20%

Fields of Research (FoR) Classification

FoR code: 1701, Psychology, 60%
FoR code: 1117, Public Health and Health Services, 40%

Table of Contents

List of Tables

List of Figures

List of Abbreviations used in the thesis

None.

Prologue

In January 2008, I was involuntarily admitted into a psychiatric ward for two weeks and diagnosed with bipolar disorder. The ease of writing this opening sentence belies the struggles I went through in my late-twenties before I started to make sense of what I was undergoing. Even now, it is not easy to put into words what I went through; possibly because I did not have any framework to help process the myriad of information and experiences that can be overwhelming.

One of the terms for such a conceptual framework is *explanatory model*, coined by anthropologist and psychiatrist Arthur Kleinman. Having such a framework is like having a sophisticated filing cabinet that is somehow able to cognitively capture everything that one goes through – thoughts, sensations, emotions, revelations, a jumble of all of the above. In addition, the cabinet sorts out what is important for storage and easy retrieval should a need arise. While explanatory models are relevant with any kind of illness, psychological problems in particular benefit from their use because the latter does not have a convenient physical manifestation that can be easily observed. The X-ray of a broken leg immediately conveys structural bone damage, which leads to treatment to set the bone and later, physiotherapy to strengthen muscles and regain function. In contrast, diagnosing a mental illness relies on self-reporting of symptoms, which can be particularly challenging if the individual suffering from the condition does not even know what to look out for.

This can be especially common in countries with low mental health awareness, such as Singapore where I had been living for many years at the time of my diagnosis. Despite being an information-rich society, the subject of mental illness was taboo in Singapore, as is still the case in many Asian countries today. The stigma surrounding mental illness also makes it difficult to speak about psychiatric symptoms. For example, the term for mental illness in Mandarin, “shen2 jing1 bing4”, is often used in an accusatory or dismissive manner.

Against such a backdrop, I had no idea that my cycles of low and high mood, experienced over at least three years, was symptomatic of bipolar disorder. In fact, I do not think I even knew what the word bipolar meant. If someone asked me then, I probably would have guessed it was something like split personality, which itself is a misnomer. During my low phase, I had little interest in activities that I previously enjoyed, such as photography, poetry, and travel. Although this is a hallmark of a depressive episode, I did not have the metaphorical filing cabinet or explanatory model to recognise it as such. I had believed that a person with depression was someone who was sad and down, whereas I was completely numb on the inside.

The depression stripped me of a clear sense of who I was because I had always navigated according to my feelings. People around me often described me as cheerful and bubbly, and I saw myself as a passionate person who strived to live according to my convictions. For example, I became a vegetarian in university after learning about the ecological unsustainability of the meat industry. Then, suddenly, for no apparent reason, I felt nothing. I could not bring myself to care about anything. Lunch times at work were trying; I knew I had to eat something but I had no mental energy to choose so I would ask my colleague to order for me whatever they were having. It was a far cry from the old me who would have made my meals as ecologically friendly as possible.

Then, inexplicably, I felt the fog over my mind and emotions lifting. I thought that somehow I had managed to get through what some have called the dark night of the soul (Durà-Vilà, Dein, Littlewood, & Leavey, 2010). Only to have the depression return again after a few months, then after a few months it lifted again. This cycle happened about three times, and each time I could not understand why I was back to square one (I called it square zero because it felt worse than before). I tried to assign blame – maybe it was spiritual burnout from serving in too many church ministries? Or perhaps my job as a newspaper journalist was too stressful, or a combination of both? Consequently, I became less actively involved in church and I requested to do backend sub-editing instead of frontline reporting. The first change could have led to me being more isolated, but fortunately I had supportive church friends who did not let that happen. One such friend would come to my home to fold my laundry with me, even though she lived far from me and did not drive. I felt like the worst company in the world, but she persisted to come regularly. In retrospect, her commitment to me as a person when I felt like an empty shell helped me to not give up on myself.

The second change led to me working night shifts at the newspaper. After work, I found I was only able to sleep about four hours each night because I was very sensitive to light and the dawn would inevitably wake me. This went on for almost two months and possibly precipitated my first full-blown manic episode. The lack of sleep coincided with clear symptoms of mania, such as markedly increased energy (which was why sleeping so little was not a problem) and faster speech rate, but these were not deemed negative. On the contrary, I interpreted them as proof that my prayers had been answered and that I had recovered from the long dark spell of not being myself. Indeed, after months of feeling like my thoughts were drowned in molasses, I revelled in the speed of ideas that came to me. I became a regular visitor to the art department of my newspaper, where I found an outlet for my seemingly newfound creativity. The head of the art department even asked if I had fallen in love – that was his explanatory model for the changes he saw in me.

Then one afternoon, while at work, I had a meltdown in the newspaper chief's office. I remember laughing, and then suddenly I started crying. Having studied psychology, my boss sent me home in a taxi and told my family to take me to a doctor. However, it was too late by then because I had lost all insight and did not realise I needed medical intervention. My family still thought I would be fine after some rest, but that night I did not sleep at all and I did not stop talking. I could not make anyone understand me, and in my frustration I ended up leaving the house and somehow in my mind I thought it would be good to board a taxi to go to the airport. Looking back, I shudder to think what would have happened if I had managed to get to the very public Changi Airport and created a scene there. Even if the airport's many security personnel discreetly escorted me away from the crowds, I might have been gone for days with my family having no idea of what happened to me. There was no way to contact my family as I did not have a phone or any form of ID on me.

Fortunately, my parents managed to stop me from boarding a taxi I had flagged down and they dragged me to a GP clinic near our home. I do not recall everything that happened, but suffice to say I was extremely uncooperative. I was eventually rushed to the hospital where I had to be sedated and physically restrained. An MRI scan was done to see if I had a concussion or brain abnormality, and blood tests to determine if I was under the influence of drugs. When both came back negative, by process of elimination, I was diagnosed with bipolar disorder.

It was only then that the pieces fell together and I finally had a name and an explanation for the perplexing highs and lows that I had experienced since childhood. As a child, I had a recurrent thought that I remember even now: "Don't be too happy, because you will only crash and be sad." I tried to be like a robot once, to have no feelings. It did not last very long but I still remember trying to do it – a child's attempt at ordering her inner world. As an adult, having an official diagnosis of bipolar helped me to seek out an explanatory model that could satisfactorily take into account what I had faced for years. It did not necessarily make my condition easier to live with, but it was certainly a key starting point in learning how to manage a very challenging illness that most experts believe requires life-long intervention.

Perhaps I could have been diagnosed earlier if I understood what I had been experiencing. Managing my symptoms before they became so severe that I had to be hospitalised would have saved my family and myself a lot of heartache. There were so many missed cues because no one knew what warning signs for bipolar looked like. One of my colleagues later said that she felt something was amiss in the frenetic pace that I was keeping up but she could not put a finger on it. Like me, she was just glad to see me happy again.

With the help of medication and regular visits to the psychologist I have since been able to better manage my depressive and manic episodes. The months of low mood is like winter, where all seems dead, but I take comfort in knowing that it will pass. When my mood swings the other way it is summertime but just as one is careful not to have too much of the sun, I avoid packing my schedule with activities and make sure I get enough rest. About a year after my diagnosis my psychiatrist and I decided to try lowering my medication dosage. However, I started to have racing thoughts and after one sleepless night I was put back on the original dosage along with another drug, and we were able to prevent my symptoms from worsening. Through open sharing about my condition my friends have been able to help to watch out for what they think are signs of relapse. Sometimes they are off the mark, but I appreciate having insight from those I trust so I do value the times they speak up.

Initially, I was afraid of myself because I believed I could be unpredictable and even violent in the midst of a manic episode. Given how strangely I behaved, and how I was not able explain afterwards why I did the things I did, I thought it entirely possible that I could hurt someone for no reason. It made me nervous to be around my sister's first child, my baby niece who was about four months old when I was hospitalised. What helped me get over this negative attitude about myself (self-stigma) was having honest conversations with people who had seen me during my breakdown. They assured me that they were never in doubt that I was still myself despite the heightened emotions and distorted cognition. My newspaper chief said: "You were not well, but you were still Jen. You wouldn't hurt a fly." I have come to understand that having bipolar does not change the core of who I am and that I can be myself apart from the condition. I used to say "I am bipolar", but now I say "I have bipolar", because the condition does not define who I am.

In fact, I have learnt to use my personal experience to help others who struggle with understanding how to live with bipolar. What I went through has sparked a keen interest in and a desire to improve society's discourses over mental illness. As a single case history in isolation, my personal experience is not enough to test any hypothesis I may have about the importance of perspective. This thesis represents my first step towards seeking answers from extant research and other people's journeys. I hope that having a deeper and more holistic understanding about the role of perspectives in mental illness will highlight the pressing need to foster better dialogue about mental health.

Chapter 1: Introduction and Aims

Approximately 350 million people worldwide are affected by depression (Marcus, Yasamy, Van Ommeren, Chisholm, & Saxena, 2012). Depression manifests itself in a variety of symptoms but the latest Diagnostic and Statistical Manual of Mental Disorders (DSM-V, APA, 2013) requires the presence of persistent low mood and anhedonia (loss of interest or pleasure) for diagnosis of major depressive episode (for a full list of diagnostic criteria see Appendix A, p. 266). The World Mental Health survey of 17 countries reported that 1 in 20 individuals on average would have had depression in the past 12 months. Depression's early age of onset, negative impact on functionality, and recurring nature has made it the number one cause of years lost to disability (Marcus et al.). In Australia, over 650,000 Australians in any given year are afflicted by depressive episodes, whether mild, moderate or severe (see Appendix A for category definitions) (Australian Bureau of Statistics, 2007). More recent figures from the 2011-2012 Australian Health Survey (ABS, 2012) show that one in 10 people in Australia have depression.

Depression diminishes quality of life by negatively impacting personal relationships (Östman, 2008) and work productivity (Simon, Chisholm, Treglia, & Bushnell, 2002). It increases the risk of heart attacks (Malach & Imperato, 2004) and is more disabling than chronic diseases like angina, arthritis, asthma and diabetes (Moussavi et al., 2007). People with depression have shorter life expectancies than those without depression, partly because they are at greater risk of medical illnesses and suicide (Chang et al., 2011). The World Report on Violence and Health (Krug, Mercy, Dahlberg, & Zwi, 2002) states that four out of every five people who killed themselves had depressive symptoms.

The Problem

Despite the prevalence of depression and its enormous burden of disease, the treatment of depression remains somewhat unsystematic. Relapses appear to be the norm rather than the exception. A 15-year longitudinal study showed that only 20% of those experiencing depression recovered and stayed well; 60% recovered but had further episodes and the final 20% either committed suicide or were continually unwell (Kiloh, Andrews, & Neilson, 1988). These statistics could be due to ineffective treatment. Most depression patients are prescribed pharmacotherapy alone (Olfson & Marcus, 2010; Olfson et al., 2002; Robinson, Geske, Prest, & Barnacle, 2005) despite evidence-based recommendations to combine drugs and psychotherapy when treating depression (Robinson et al.).

Antidepressant overuse is partly due to the portrayal of depression as primarily caused by neurotransmitter imbalance (Clarke & Gawley, 2009). The biomedical view is simplistic because the causation of depression is known to be complex and multifactorial (Lacasse & Leo, 2005), involving bio-psychosocial factors. One journal article listed 27 possible causes for depression (Street, Sheeran, & Orbell, 1999). Correspondingly, there is no one-size-fits-all approach to treating depression. However, the biomedical view of depression and over-emphasis on antidepressants in depression treatment still prevails for several reasons.

Firstly, pharmaceutical companies push the biomedical explanation (Clarke & Gawley, 2009; Lacasse & Leo, 2005) because it helps to sell drugs. Secondly, doctors can be biased towards biomedical causations due to their training (Andersson, Troein, & Lindberg, 2001). Finally, not-for-profit mental health organisations promote the biomedical model because they believe that describing depression and other mental illnesses as medical conditions, comparable to diabetes or cancer, reduces stigma towards the sufferer (Botha & Dozois, 2015). While some researchers have found this to be partly true (Schreiber & Hartrick, 2002), others have concluded differently (e.g., Mehta & Farina, 1997) and more research is needed, particularly with respect to self-stigma. Self-stigma arises when patients internalise the negative stereotypes and prejudices associated with having a mental health condition (Corrigan & Watson, 2002a). Some researchers have found that self-stigma inhibits help seeking more than anticipated stigma from others (Schomerus, Matschinger, & Angermeyer, 2009). As such, it would be important to see if a biomedical view of depression causation reduces self-stigma among people with depression as this could potentially improve treatment outcomes.

Another aspect of treatment that could affect outcomes would be the therapeutic relationship between patient and their health provider. Previous research has shown that factors such as good patient-physician communication and strong therapeutic alliance are linked to positive treatment outcomes (Street, 2001, 2003). Good communication is so critical that the code of conduct for doctors developed by the Australian Medical Council includes nine components of effective communication (Medical Board of Australia, 2014). In addition, a recent literature review by Mikesell (2013) revealed that building supportive and caring relationships with patients requires doctors to be there for the patient. If doctors were aware of how their patients viewed their depression, it would make it easier for them to communicate well and to empathise effectively. This is another reason why it is important to study how people with depression view the condition.

I have indicated why it may be important to know to what degree a person with depression subscribes to the biomedical view of depression. How a depressed person views their illness, especially in terms of its causation, influences their treatment choices and recovery journey. It has been demonstrated that an individual's belief about their depression influences their help-seeking behaviour (Okello & Neema, 2007), illness experience and treatment choices (Buus, Johannessen, & Stage, 2012). Perhaps the effectiveness of depression treatment can be improved if we first investigate how individuals with depression perceive the condition. This is the approach of my thesis, which looks at how depressed individuals in primary care explain their depression.

My Approach

The analytical term that I use to examine how patients talk about their depression is explanatory model (EM). It was coined by Arthur Kleinman to refer to culture-specific conceptualisations that influence people's communication about their health issues (Kleinman, Eisenberg, & Good, 1978). There are EMs for other medical conditions; for example, diabetes is explained in terms of insulin. Researchers have called for clinically relevant studies to systematically assess the impact of specific EMs on satisfaction with patient-clinician consultations, therapeutic alliance between patients and doctors, and intervention outcomes (e.g., Waite & Killian, 2009). Kleinman et al. intended for patient and doctor EMs to be compared so that doctors can deal with major differences that may cause challenges for clinical management.

Research into depression EMs is particularly important to depression management because there is so much more debate about the causation of the condition. As previously mentioned, this debate ultimately affects outcomes. Furthermore, patients experiencing mental distress may not have physiological changes to show their doctors, so patient perception is especially relevant. This research is exploratory in nature and focuses mainly on patients, with input from a small number of general practitioners (GPs). I chose the primary care setting because that is where the majority of depression cases are treated without referrals to specialists (Harman, Veazie, & Lyness, 2006; Katon & Schulberg, 1992). I explored the two key aspects affecting patient recovery that I cited earlier, namely patient self-stigma levels and GP-patient communication effectiveness. I will briefly explain my approach to these two aspects now.

As mentioned previously, mental health organisations such as the National Alliance for Mental Illness (NAMI) and the National Association for Research on Schizophrenia and Affective Disorders (NARSAD) described depression as a biomedical condition in the hopes that it would lessen mental illness stigma. NARSAD's public education campaign centred on the slogan "depression is a flaw in chemistry, not character" (Goldman, Nielsen, & Champion, 1999) and up to 2013, NAMI described depression as a chronic medical condition (Kemp, Lickel, & Deacon, 2014). My thesis will test whether biologically oriented EM among patients with depression is associated with lower self-stigma than those with a psychosocial EM. To examine communication between patients and their GPs I will use communication accommodation theory (CAT: Gallois et al., 2005; Giles, 1973). Developed by Howard Giles, CAT is a general theory of communication that an individual's cognitions and motivations influence their communication behaviour. It recognises that people modify their communication due to situational, personal or interactional variables (Williams, 1999). This study examines whether EMs espoused by both depressed patients and their doctors correlate with perceptions about GP-patient communication.

Aims and overall structure of thesis. This thesis aims to examine depression EMs in primary care and how this affects patient self-stigma, and patient-GP communication, as these in turn influence treatment outcomes. Specifically my research questions (RQs) are:

1. What are depression EMs of primary care patients and do these change over time?
2. Does higher endorsement of biomedical causation for depression correlate with lower patient self-stigma?
3. Does agreement to certain aspects of patient EMs correlate with patient perception of greater GP-patient communication effectiveness?
4. How aware are GPs of their patient's depression EM, how concordant are GP EMs with patient EMs, and how does awareness and/or concordance correlate to depression outcomes?

The next chapter reviews existing depression literature and research, with the following three chapters detailing each theoretical concept driving the present study: explanatory models, self-stigma, and communication accommodation theory (Chapter 3-5). In Chapter 6, I explain the rationale for my research methodology. In Chapter 7, I describe how I addressed the RQs and provide details about participants, materials, and procedure. Chapters 8-11 focus on answering each of the RQs with results from the quantitative surveys and interview quotes to illustrate the survey findings where relevant. The EMs of depression patients are in Chapter 8. Results from both the initial survey and follow-up survey are presented and discussed. Chapter 9 is the longitudinal correlational study of patient belief in biomedical causation for depression and their self-stigma levels. Chapter 10 consists of the correlational study between patient EM beliefs and perceived GP-patient communication effectiveness at Time 1. Chapter 11 presents the study to answer RQ4, while

Chapter 12 is the results of the GP interview study. I conclude with an overall discussion of the major findings from my research in Chapter 13, along with directions for future research.

Chapter 2: Depression

Understanding how depression has been viewed in the past and continues to be viewed today provides the necessary context for grounding my thesis which, as briefly mentioned in Chapter 1, examines the importance of patient and physician perspectives of the condition. In this chapter I discuss the history and development of the understanding of depression, demonstrating the complexity and diversity of depression theories. The myriad of possible ways to comprehend depression suggests that it is more therapeutically useful to discuss the specific depression perspectives of patients and their doctors instead of trying to find one unified theory of depression. The chapter consists of a historical overview of depression theory (including a brief description of the anti-psychiatry movement, whose proponents would argue that the experience of depression is not the problem, but rather its labelling and treatment), followed by depression classification, diagnosis, epidemiology and treatment.

Depression Through The Ages

5th century BC to the 15th century AD. William Styron referred to depression as the "ancient affliction" in his memoir Darkness Visible (1990, p. 17). Depression has been systematically studied since at least the time of Greek physician Hippocrates in fifth-century BC (Andrews, 2010). Theories about the causes of depression (or even what to call it) and how to treat it have been evolving from these early times. Particularly from the late 20th century onwards there has been a burgeoning of depression theories and treatment options. Andrews (pp. xxxiii-xxxix) provides a brief overview of how various thinkers have conceptualised depression over 2,500 years and demonstrates the complexity around the articulation of what depression is and what it is not. Over time, public opinion would swing back and forth like a pendulum between physiological and psychological explanations of depression (Leventhal & Martell, 2006).

The earliest physiological explanations for depression among Western European thinkers were based on the humoral theory (Andrews, 2010). Hippocrates theorised that the human body was filled with four basic fluids called humors, which corresponded to specific temperaments. He applied this notion from the ancient Greek belief that the physical world was comprised of earth, air, fire and water – which had arisen from even older civilisations in Egypt, India and China. The word melancholia is derived from Ancient Greek – μέλας (melas) means dark or black, and χολή (kholé) means bile. Black bile was linked to the melancholic temperament, and an excess of black bile resulted in symptoms such as despondency, restlessness, and lack of appetite. Andrews noted that the humoral theory was also adopted in Roman, Islamic and European medicine and it persisted for centuries.

In second century AD, Greek physician Galen elaborated on earlier Hippocratic ideas by proposing the existence of various souls governing bodily functions (Leventhal & Martell, 2006). In particular, he wrote of an *animal soul* that directed sensation and feeling through the brain and central nervous system. Andrews (2010) argues that Galen's animal spirits were the ancient world's equivalent of neurotransmitters, which were only discovered centuries later. Many modern antidepressants target the imbalance of neurotransmitters, which has been theorised as the cause of depression. However, whether depression is caused by chemical imbalance continues to be hotly debated today by patients and physicians alike. Further discussion of chemical imbalance theory and antidepressant efficacy appears later in this chapter.

The humoral theory of melancholy even extended to lycanthropy, the transformation of a human into a wolf in appearance and behaviour. Many in the Western world today consider the werewolf a purely mythical creature, popularised in books and movies but not taken seriously. However, the ancient Greek and Roman physicians sincerely believed lycanthropy to be a form of melancholy. Poulakou-Rebelakou, Tsiamis, Panteleakos, and Ploumpidis (2009) excerpted works by six prominent Byzantine physicians (AD330-1453) who helped to preserve the texts of Hippocrates' and Galen's writings, which have since been lost in their original form. Five of the Byzantine physicians such as Oribasius (personal physician to the Emperor Julian the Apostate in the 4th century) and Michael Psellus (a philosopher, politician, and historian in the 11th century) considered lycanthropy to be a type of melancholy, while the sixth believed it was a type of mania. Byzantine medical knowledge subsequently influenced Western European and Arab medicine, but this view of lycanthropy was eventually replaced by attributions to demons and witchery during medieval and Renaissance times (Poulakou-Rebelakou et al.).

Besides the humoral theory of mental disorders, the Greeks and Romans also believed that moral or psychological lapses were to blame, and accordingly the treatment for mental disorders included controlling one's emotions and living virtuously, in addition to dubious physical remedies such as bloodletting and purging (Leventhal & Martell, 2006). These ideas continued into the Middle Ages and Renaissance. The church's view became prominent that mental disorder arose from the battle between God and Satan for the human soul. Physical ailments were the realm of medical doctors and medicine, while mental disorders were the domain of clergyman, who employed prayer and fasting initially as preferred remedies. Unfortunately harsher treatments arose as demons came to be seen as the cause of mental illness; for example, flagellation was the most common treatment in the 13th century to dispel demons (Leventhal & Martell).

The 16th century to 1950s. In 1597, French physician André du Laurens described melancholia as a form of delirium without fever, where people became delusional (Andrews, 2010). He gave several examples that he had encountered, including one man who thought himself made of glass, and another who feared he had turned into a rooster. According to Andrews, Du Laurens' work was prominent in Europe and frequently cited up to the 18th century. In 1628, William Harvey discovered the principles of blood circulation and for the next 100 years, numerous theories arose linking melancholic states to circulatory problems; for example, thickening of the blood led to sluggish circulation in the brain, which in turn affected the flow of animal spirits. Benjamin Rush (1745-1813), who has been called the father of American psychiatry (Ferguson, 2006; Shryock, 1945), theorised that mental disorder could be rectified with better blood circulation to the brain, but his treatment methods using bloodletting, emetics and purgatives (Leventhal & Martell, 2006) were similar to the ancient Greeks.

In the late 18th century, depression theories began to de-emphasise physiology and shift into the realm of psychology (Andrews, 2010). French physician Philippe Panel, one of the important early thinkers of modern psychiatry, linked melancholia to delusional thinking and attributed such a wide range of symptoms to the disorder that it covered practically all chronic psychotic conditions, including schizophrenia. Pinel's student Esquirol divided melancholia into narrower disorders called monomanias, where the mind focused on a single subject. He coined the term *lypémanie*, or sad-madness in Greek, for the monomania focused on sad moods, which he considered to be primarily an emotional disturbance. However, the term lypémanie was not taken up. It was another French psychiatrist Louis Delasiauve who first used the term depression to refer to a psychiatric symptom in 1856. This term, from the Latin verb deprimere (to press down) has since replaced the word melancholy.

The discourse on depression became more sophisticated, especially in the 20th century, making a leap from demonic activity or suspected witchcraft to psychological theories focused on the individual's inner reality. One theory prominent in its day was Austrian neurologist and psychiatrist Sigmund Freud's psychoanalytic theory that emphasises the role of childhood events in shaping adult experiences (Freud, 1917/1959). Freud posited depression as a reaction over the loss of someone for whom the depressed person had mixed feelings. To minimise the sense of loss, the depressed person takes on some characteristics of the person who was lost, but anger that would have originally been expressed towards that person is now turned inwards. This was a new approach on the link between interpersonal loss and depression, a link that dates back 700 years because from as early as the late 13th century, Arab physicians had identified lovesickness from unrequited love as a cause of profound melancholy. The condition was easily cured if the object of one's affection was available, but otherwise it was lasting, dangerous and even fatal (Andrews, 2010). Andrews noted that depression treatment with psychoanalysis has generally declined due to lack of empirical evidence for its efficacy.

At around the same time Freud first began publishing his theories, German psychiatrist Emil Kraepelin developed his theory of inherited temperament of how a component of one's personality that emerges at a young age and has impact throughout one's lifetime (Leventhal & Martell, 2006). Considered a pioneer of the modern classification system of mental disorders, Kraepelin (1921) described the depressive temperament, arguing that people with this temperament tended to develop depression because they were generally serious, self-critical and guilt-conscious. He attempted to find distinct groupings of symptoms that would help differentiate between disorders but failed because there was too much overlap in the symptoms from one condition to another.

Modern Theories of Depression

Cognitive theory. In the 1960s, US psychiatrist Aaron Beck developed a cognitive theory of depression (Beck, 1967) that has since become one of the most prominent and extensively researched psychological theories of depression (Clark, Beck, & Alford, 1999). The cognitive model delineates how an individual's thoughts and perceptions can determine their emotions and behaviours. Beck's theory holds that people with depression think differently than people without depression and it is this difference that gives rise to depression. He described underlying, persistent, thought patterns called schemas that are formed by early negative life experiences such as rejection by peers or criticism from authority figures. These are latent until triggered by adverse events that resemble the original conditions of the learned schema. When triggered, the schema distorts an individual's information processing system, unleashing automatic negative thoughts about oneself, the world and the future – resulting in and exacerbating depression.

Beck's cognitive approach went against the psychoanalytical approach of Freud which was based on gaining insight into unconscious drives, and it initially also conflicted with the approach of behaviourism, which focused on stimuli and behavioural responses (Trull, 2004). In the 1970s the behavioural modification techniques and the cognitive approach became joined, resulting in cognitive-behaviour therapy (CBT) (Wilson, 2008). CBT for depression, first set out by Beck and colleagues (Beck, Rush, Shaw, & Emery, 1979) became increasingly influential in the late 20th century as it was widely taught to health professions including general practice, nursing and occupational therapy (King, 2002). It is the most studied psychotherapy for depression (Cuijpers et al., 2013) and the main treatment modality employed today for all mental disorders (Field, Beeson, & Jones, 2015). The goal of CBT is to help patients overcome their difficulties by harnessing cognitive shift as the key mechanism of change – changing how an individual thinks will in turn result in different behavioural and emotional responses. As Beck et al. (pp.118-119) put it:

> The impact of the therapeutic techniques derived from a strictly behavioural or conditioning model is limited because of the restriction to observable behaviour and selective exclusion of information regarding the patient's attitudes, beliefs, and thoughts – his cognitions…. For the behaviour therapist, the modification of behaviour is an end in itself; for the cognitive therapist it is a means to an end – namely, cognitive change.

Numerous randomised control trials have shown that CBT is efficacious in treating depression, especially when combined with pharmacotherapy (Cuijpers et al., 2013). It remains highly relevant today as more cognitive-based therapies such as preventive cognitive therapy (Bockting et al., 2005; Bockting et al., 2015) and mindfulness-based cognitive therapy continue to be developed (Abolghasemi, Gholami, Narimani, & Gamji, 2015; Teasdale et al., 2000). In CBT, the therapeutic relationship between the patient and the clinician is key because they must work as a team to identify and evaluate the patient's automatic thoughts. It is vital for the therapist not only to gain the trust of the patient, but to also convey an accurate conceptualisation of the patient's disorder, so that it resonates with the patient (Wright & Davis, 1994). The different frameworks that are relevant when patients and doctors discuss medical conditions such as depression will be further discussed in the next chapter on explanatory models. Due to the importance of effective communication in building the therapeutic relationship, this thesis will also include a separate literature review chapter on communication.

Theory of learned helplessness. In the 1970s, around the same time Beck's theory of cognition was gaining prominence, the theory of learned helplessness by US psychologist Martin Seligman also came to the fore in trying to explain certain types of depression that were characterised by passivity and a pessimistic mindset. In a laboratory setting, Seligman (1972) discovered that restrained dogs became conditioned to accepting pain from electric shocks. The animals learned that they could not avoid or escape from the painful stimulus and so they eventually gave up trying. This giving-up response became apparent in other situations where the dogs behaved helplessly (whining and not moving) even though they could have effectively escaped (Miller, Roselini, & Seligman, 1977; Seligman, 1975). In humans, the theory predicts that some people become depressed after exposure to adverse events that are or believed to be beyond their control to avoid. Various studies over the years have confirmed that uncontrollability of a negative stimulus does have a depressive effect (e.g., Goodman & Hess, 1999; Hiroto & Seligman, 1975). Goodman and Hess studied patients with implantable cardioverter defibrillators, which discharge electrical shocks without warning whenever there are irregularities in the patients' heart rate. The study found that the total number of discharge shocks significantly predicted depressive symptoms in patients without a history of psychological distress before the implant. The explanation was that patients learned through the increasing number of intermittent shocks that they were not able to control the discharges, causing them to feel helpless and resulting in more depression symptoms.

In a critique and reformulation of the learned helplessness theory, Abramson, Seligman, and Teasdale (1978) addressed why some people responded to the uncontrollability of adverse events with learned helplessness while others did not. Also, the old theory could not explain why people with depression suffered from low self-esteem, why they tended to blame themselves for failure and why they reported variation in severity and length of symptoms. The revised theory states that what the uncontrollability is attributed to influences if, how and to what extent helplessness manifests as an outcome (Abramson et al.). This attributional framework, based on refinement of the attribution theory (cf. Heider, 1958; Weiner, 1972, 1974) posits that the cause of uncontrollability can be perceived to be stable or unstable, global or specific, and internal or external.

A stable and global attribution of uncontrollability gives rise to helplessness that is chronic and broad, resulting in repeated severe depression that impacts on many aspects of the individual's life, whereas internal attribution of uncontrollability results in low self-esteem and self-blame (Abramson et al., 1978). In other words, people who have stable, global and internal attributions tend to have a pessimistic outlook or explanatory style – a phrase that was later incorporated into the learned helplessness theory by Peterson and Seligman (1984) – and these in turn are more likely to suffer from learned helplessness and depression. They see adverse events as permanent (e.g., "it will never change"), pervasive (e.g., "nothing goes well for me") and personal (e.g., "it is because of me") (Peterson, Maier, & Seligman, 1993). Clearly, such an explanatory style can be a factor that predisposes, precipitates, and prolongs depression. If the way people explain negative events in their lives can make them more likely to be depressed, it is highly probable that the way people with depression explain their depression would have far-reaching implications as well. Indeed, explanatory models do impact on many aspects of an individual's illness (Cabassa, Hansen, Palinkas, & Ell, 2008; Laws, 2016; Makanjuola et al., 2016), and the chapter on explanatory models will further elaborate on this with respect to depression.

Attachment theory. In the 1980s, John Bowlby's attachment theory arose in the discourse about depression after his third volume on attachment and loss focused on the link between attachment and depression (Bowlby, 1980). According to the theory, babies are born with an innate attachment behavioural system that spurs them to bond with significant others in their environment (e.g., their parents or caregivers) and to look for these "attachment figures" in times of distress (Bowlby, 1969, 1973, 1980). If the attachment figure reliably provides comfort and assurance to the distressed infant, this promotes a stable sense of security in the infant and a positive model of self and others. If the attachment figure is unavailable and unsupportive, the opposite ensues – the infant feels insecure and develops a negative view of self and others (Bowlby, 1973). Today, attachment theory is arguably one of the leading theories in the study of early childhood behaviour, treatment of children, child mental health and associated fields. The generalised models from early life result in patterns of relational expectations, behaviour, and emotions in adulthood, which Hazan and Shaver (1987) termed attachment styles.

Traditionally, attachment styles were classified into secure, anxious, and avoidant and attachment instruments correspondingly categorised attachment according to these three types (e.g., Hazan & Shaver, 1987). However, recent developments indicate that adult attachment orientations are more accurately assessed along the two independent dimensions of anxiety or avoidance (Brennan, Clark, & Shaver, 1998; Mikulincer & Shaver, 2003). People who score high on the anxiety dimension are fearful of rejection, whereas those who score high on the avoidance dimension strive to maintain emotional and psychological independence (Brennan et al.). Studies with regionally or nationally representative samples have shown that individuals who exhibit anxious and/or avoidant attachment are significantly more at risk of depression and depressive symptoms (Cooper, Shaver, & Collins, 1998; Palitsky, Mota, Afifi, Downs, & Sareen, 2013). Other studies have also found associations between insecure attachment orientations and depressive symptoms (e.g., Cantazaro & Wei, 2010; Simpson, Rholes, Campbell, Tran, & Wilson, 2003; Wei, Heppner, & Mallinckrodt, 2003).

Researchers have uncovered several mediators that explain how insecure attachment results in depression (Rholes et al., 2011). Lee and Hankin (2009) theorised that insecure attachment results in dysfunctional attitudes that negatively influence an individual's expectations of others, as well as assessment of interpersonal events, all of which contribute to a “cognitive vulnerability” to depressive symptoms (p. 230). These consequences of insecure attachment overlap with and could potentially be exacerbated by the stigma experienced by many individuals suffering from mental health problems. As mentioned in Chapter 1, one of the aims of my thesis is to examine the association between stigma and certain perspectives of depression. A separate literature review chapter on stigma will provide further details on this.

Besides the cognitive vulnerabilities to depression that is caused by insecure attachment, insecure individuals may also be more at risk of depression because their reactions to stress are unhelpful. Berant, Mikulincer, and Shaver (2008) noted extensive evidence that highly anxious people tend to perceive stressful events as more threatening than less anxious individuals. People with avoidant attachment do not have this problem of over-perceiving threat, but their reactions to stress are similarly counter-productive; for example, they are less likely to seek support (Simpson, Rholes, Nelligan, & Miller, 1992) and instead they use ineffective strategies such as avoidance, denial, distraction, and repression (Gjerde, Onishi, & Carlson, 2004; Vetere & Myers, 2002). The implications of a patient’s attachment orientation on therapy have also been studied. Bettmann (2006) asserts that a comprehensive understanding of a client’s attachments results in more appropriate treatment. A review of research on adult attachment and psychopathology by Mikulincer and Shaver (2012) found that improving attachment security of individuals would reduce the probability and intensity of psychiatric symptoms. They added that this evidence highlights the healing effects of having supportive attachment figures, including therapists.

The monoamine hypothesis. In the midst of the many psychological theories for depression in the 20th century, one key physiological theory arose that hypothesised the cause of depression as a deficiency in a group of brain chemicals called monoamine neurotransmitters (Andrews, 2010). The three main monoamines found in the brain are serotonin, dopamine and norepinephrine (Nutt, 2008). The monoamine hypothesis for depression proposes an underlying biological basis for the mood disorder (Hirschfeld, 2000). It followed a few observations made serendipitously from researchers in several different fields. The first observation was from examining the action of the hallucinogen lysergic acid diethylamide (LSD), an important research area in neuroscience in the 1950s (Hirschfeld). Researchers found that LSD blocked peripheral serotonin receptors, and this observation, combined with prior knowledge of LSD as a mood altering substance and the presence of serotonin in the brain, led to initial postulations about the role of serotonin in mood disorders (Woolley & Shaw, 1954).

The second key observation that supported the link between serotonin and depression was from research into hypertension treatment. The anti-hypertension drug reserpine was effective in lowering high blood pressure but researchers found that it caused depression and anxiety symptoms in some patients (Muller, Pryor, Gibbons, & Orgain, 1955). The effect of reserpine in animals, namely drowsiness and psychomotor impairment, seemed similar to depressive symptoms in humans. When reserpine treatment stopped, these symptoms ceased in both animals and humans. In addition, when monoamine precursors were administered to humans, the psychological effects of reserpine were reversed (Degkwitz et al. cited in Hirschfeld, 2000). Further research discovered that reserpine depleted serotonin levels in the brain and increased concentrations of a main breakdown product of serotonin (5-hydroxyindoleacetic acid) in urine (Shore, Silver, & Brodie, 1955). This was seen as evidence for correlating low serotonin levels with depression symptoms.

The third key observation came from the accidental discovery that iproniazid, an anti-tuberculosis (TB) drug, improved the mood of TB patients (Crane, 1956; Kline, 1958). Researchers found that iproniazid prevented the degradation of serotonin and norepinephrine, another monoamine neurotransmitter, by inhibiting the enzyme that breaks down monoamines (monoamine oxidase). This led to higher concentrations of the monoamines, which correlated with the observed alleviation of depression symptoms such as improved energy levels. Subsequently, monoamine oxidase inhibitors (MAOIs) were developed to treat depression (Hirschfeld, 2000).

Another drug, imipramine, originally developed to treat panic in psychotic patients, was ineffective for that purpose but instead imipramine reduced depressive symptoms in certain patients who had concurrently been experiencing fatigue, sense of heaviness, hopelessness, guilt, despair and psychomotor retardation (Kuhn, 1958). Further experiments showed that imipramine inhibited the re-absorption of norepinephrine and serotonin (Herting, Axelrod, & Whitby, 1961). Imipramine became classed as a tricyclic antidepressant, the first of a group of antidepressants that differed from MAOIs in how its pharmacological mechanism increased synaptic concentrations of monoamines. Although TCAs were used to treat depression for many years, the medication had a wide range of negative side effects (Cookson, 1993). This was partly due to their chemical structure, which caused them to inhibit the reuptake of numerous neurotransmitters (Carlsson as cited in Hirschfeld, 2000). Hirschfeld said re-uptake inhibitors were developed that were selective to a single monoamine system to improve the safety and tolerability of antidepressants. Probably the most well known of these is the class of antidepressants known as selective serotonin reuptake inhibitors (SSRIs). Unlike the other types of antidepressants that were discovered by chance, SSRIs were designed specifically for depression treatment and have been found to be effective in some patients previously resistant to therapy (Åberg-Wistedt, 1989). Although they tend to be better tolerated than TCAs, they can still cause uncomfortable effects such as headache, nausea and vomiting.

The monoamine hypothesis has enjoyed considerable support because it attempts to explain how antidepressants work. However, it does not explain why the drugs increase monoamine levels within hours but there is a delay of several weeks before any improvement can be observed in patients. Patients often feel worse after starting on antidepressants because the adverse side effects of the antidepressants can manifest within hours or days. This naturally causes significant problems with treatment adherence (Hirschfeld, 2000). Furthermore, researchers have found that monoamine deficiency does not consistently cause depression (Iversen, 2005; Ruhe, Mason, & Schene, 2007). Conversely, increasing serotonin levels have been found ineffective at alleviating depression by some researchers, pointing to inconsistency and inconclusiveness of evidence for the efficacy of antidepressants such as TCAs and SSRIs. For example, a review study by Turner, Matthews, Linardatos, Tell, and Rosenthal (2008) showed that antidepressant efficacy is smaller when all Food and Drug Administration (FDA) trials are included instead of only published studies. Publication bias made it appear as if 94% of the trials were positive, but the FDA analysis showed only 51% were positive. Moreover, meta-reviews involving placebos show that patients with mild depression are unlikely to benefit from antidepressants (Barbui, Cipriani, Patel, Ayuso-Mateos, & Van Ommeren, 2011) compared to patients with more severe depression (Fournier et al., 2010).

Most importantly, the exact causation mechanism between low monoamine levels and depression remains unknown despite decades of research. Even as researchers continue to develop new theories (Baudry, Mouillet-Richard, Launay, & Kellermann, 2011; Nestler et al., 2002), the neurotransmitter imbalance explanation remains a mainstay of biological models for depression today (Schomerus et al., 2012). This is largely due to pharmaceutical companies' advertising campaigns that further oversimplify the already limited monoamine hypothesis when presenting depression to the public (Lacasse & Leo, 2005; Leo & Lacasse, 2008). The biochemical explanation for depression has also been adopted by mental health advocates in the belief that such explanations will reduce public stigma (see Chapter 4 on stigma).

Anti-psychiatry movement. At around the same time researchers started proposing the monoamine hypothesis in the 1960s was also when the anti-psychiatry movement came to the fore. The proponents of anti-psychiatry challenged the validity of psychiatric classification, the subjectivity of diagnostic processes and the apparent inhumanity of treatment options such as electroconvulsive therapy and long-term involuntary hospitalisation (Grob, 2011; Staub, 2011). Psychiatrists like R. D. Laing (1969) and Thomas Szasz (1960) were key players in the movement, although they disliked the term *anti-psychiatry*, because they desired reform instead of revolution in the discipline (Whitley, 2012). They were joined by social scientists like Foucault (1961/1965) who contended that psychiatry was predominantly an institution to wield social control. This has continued to recent years. Horwitz and Wakefield (2007) argue against mainstream psychiatry pathologising ordinary human sadness and wrongly categorising it as clinical depression. Psychologists have also joined in the anti-psychiatry movement. For example, Kirsch (2010) documented meta-analyses showing that antidepressants are not significantly more effective than placebos. He concluded that the theory of depression as a chemical imbalance was a myth because there is insufficient evidence to back such a theory.

The sampling of perspectives from the anti-psychiatry movement alludes to the considerable challenge faced by people experiencing depressive symptoms in understanding their condition and how to manage it. The disconnect that would exist between a patient who does not believe they have depression and their physician who believes firmly otherwise would seriously jeopardise the therapeutic relationship even before a single word is exchanged between both parties. This underscores the importance of elucidating depression perspectives of patients and doctors. Certain criticisms of the anti-psychiatry movement fuelled changes that attempted to improve the classification and reliability of mental illness categories by specifying the use of standardised clinical interviews and diagnostic processes in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) (Whitley, 2012). The next section will cover the classification of depression beginning from DSM-I to the latest DSM-5.

Classification of Depression

Goldstein and Anthony (1988) argue that the classification of depression began with Kraepelin, who has been called the founder of modern scientific psychiatry (Engstrom & Kendler, 2015). Kraepelin initially viewed depression as a mood disorder with endogenous, organic causes but eventually he noted evidence of psychogenic depression, which he considered to be distinct from endogenous depression. By the end of the 1920s, psychiatrists were split in two camps over the classification of depression – the nature school focused on heredity, anatomy, and pathogens, while the nurture school was concerned with the impact of early life experiences, current stressors, loss, and unconscious processes (Goldstein & Anthony). It would be reasonable to expect this division in public opinion as well, impacting patient perspectives.

DSM system of classification. In 1952, the American Psychiatric Association (APA) published the first DSM (DSM-I). According to Houts (2000), who reviewed 50 years of psychiatric nomenclature, DSM-I's development came after the mass involvement of US psychiatrists in choosing, assessing, and treating soldiers for World War II. A committee of these psychiatrists developed a classification scheme called Medical 203 that was issued in 1943 as a War Department Technical Bulletin. This was adopted by all Armed Forces during the war and afterwards it began to be used in many clinics and hospitals by psychiatrists who had served in military duty. The Veterans Administration (VA) also implemented a modified version of the Medical 203. At the same time, the psychiatric categorisation subsection of the US general medical guide, called the Standard Classified Nomenclature of Disease, was also in use. The APA and the New York Academy of Medicine had prepared this document before the war (Greenberg, Shuman, & Meyer, 2004). In addition, the World Health Organization (WHO) in 1949 had published the sixth revision of the International Statistical Classification of Diseases, Injuries and Causes of Death (ICD-6), which had a section on mental conditions for the first time, so this was consulted too, according to Houts.

The use of the many different documents was confusing, therefore in 1950, an APA Committee on Nomenclature and Statistics was tasked to review and develop a classification system specifically for the US (Houts, 2000). The committee circulated an adaptation of the Medical 203, the VA system, and the Standard's Nomenclature to 10 per cent of APA members, of which 43% replied and 93% of these approved. After some revisions, the DSM-I came into being, with much of its structure and conceptual framework being the same as in the Medical 203 – in fact, many sections of text were identical. The context of how the DSM came about, with its bias towards military rigidity, could partly explain why there has been debate over its use in clinical encounters with much greater patient diversity than in the Armed Forces. This has implications over how depression is managed because a doctor who is concerned with deciding on a classification could overlook certain aspects of the patient's experience or explanatory model of the disorder.

The DSM, or any classification system for that matter, is fluid – as shown by the inclusion of homosexuality in the DSM-1 (APA, 1952) as a sociopathic personality disturbance and its removal in 1974 (Mayes, Bagwell, & Erkulwater, 2009) after prolonged public uproar. The DSM-I, containing the term *depressive reaction*, classified depression as subcategories of neuroses (a problematic term that means different things to different people), psychoses, and personality disorders. However this was criticised for being too basic and for overlooking the difference between unipolar and bipolar depression (Goldstein & Anthony, 1988). The DSM-II published in 1968 did not address the latter; it was similar to DSM-I except it included the term *depressive neurosis*, defined as a disproportionate reaction to internal conflict or an identifiable event (APA, 1968).

The classification of depression changed fundamentally in DSM-III (APA, 1980) as it eliminated the diagnoses of depressive reaction and neurotic depression, which had been in DSM-I and DSM-II. For the first time, DSM-III distinguished between manic-depressive (bipolar) disorders and unipolar or major depressive disorders (Gruenberg, Goldstein, & Pincus, 2008). Aetiology, precipitating events, or psychotic features, were not taken into consideration as the DSM-III offered an atheoretical approach to classification. It focused on descriptions that were easily defined, reliable, and valid (Goldstein & Anthony, 1988) because there was a perceived need for reaffirm psychiatry's medical identity and commitment to science (Parker, 2005). The work of Feighner et al. (1972) and Spitzer, Endicott, and Robins (1975) greatly influenced the listing of explicit symptoms as inclusion criteria for the classification of depression. Both diagnostic systems included the time factor (how long symptoms had been present for) and exclusion criteria. To make the DSM-III diagnosis of a major affective disorder there must be depressed mood or loss of interest in activities. In addition, at least four of eight symptoms, based on Feighner et al.'s criteria and Spitzer et al.'s Research Diagnostic Criteria (RDC), must be present for a fortnight or more. The list of exclusion criteria included delusions, hallucinations, bizarre behaviours, schizophrenia or paranoid disorders, and bereavement (APA, 1980). Goldstein and Anthony noted that the DSM-III for the first time distinguished between bipolar disorder versus unipolar depressive disorder, arising from research into genetic, familial, biochemical, physiological, and pharmacological differences in these two conditions.

The third revised edition of the DSM (DSM-III-R) was published in 1987 with some minor changes; for example, the term *mood disorder* took over *affective disorder* (Goldstein & Anthony, 1988). As a result of research into cyclical depression that began in winter and ended in spring, the concept of a seasonal pattern to depression was added. Although DSM-III and DSM-III-R had taken into account research studies in their formulation of what constitutes depression, their atheoretical approach meant that there was no longer any distinction between psychogenic (i.e., reactive) and endogenous depressions – a change that Goldstein and Anthony found questionable because they felt the distinction had therapeutic implications. In addition, the terms psychoneurotic depression and psychotic depression were both omitted in DSM-III and DSM-III-R, which makes it hard to classify a neurotically depressed individual. Goldstein and Anthony concluded that it was hard to determine whether the later DSMs were generally better than the earlier ones with respect to depression, perhaps because "[d]epression is far from figured out" (p. 193).

In the DSM-IV, published in 1994, there was additional criterion that symptoms of a major depressive episode should "cause clinically significant distress or impairment in social, occupational, or other areas of functioning" (APA, 1994, p. 523). However, Parker (2005) argues this definition is too vague and it poses a risk to reliability estimates. He also takes issues with DSM-III being atheoretical, arguing that it potentially results in limited explanatory power because a classificatory system that is not based on a valid explanatory model is essentially flawed. He considers the term *major depression* to be problematic because research has shown that "its natural and treatment history are difficult to predict at the individual level, while cause and response to treatment are again more related to factors in the individual sufferer rather than being integral to the disorder" (Parker, 2005, p. 469). In an earlier review, Parker (2004) showed that randomised control trials of a wide variety of treatments for mood disorders (from antidepressants to psychotherapy to St John's Wort) had the same overall efficacy levels of between 50-55%. This lack of differential treatment outcomes is tied to the lack of specificity in the diagnostic category (Parker, 2005). Imagine a patient diagnosed with major depression asking what is the best way to treat it and the doctor replies that the vastly different treatments all have a 50/50 chance of working. This would understandably impinge on the patient's confidence over the efficacy of treatment, which could in turn undermine treatment adherence and outcomes.

Controversies over depression classification continued with the publication of DSM-5 in 2013. The issue that sparked the most debate was the removal of the bereavement exclusion criteria (Pies, 2014). Critics argue that this medicalises ordinary grief and leads to over-prescription of antidepressants. Supporters argue that there is no clinical basis for excluding someone from a diagnosis of major depression simply because the condition occurs after the death of a loved one. In his commentary, Pies noted that although bereavement-related grief and major depression share similar features, they do have distinct characteristics that enable them to be differentiated. Bereavement does not guarantee that someone will not develop depression; in fact the former is a common precipitating factor of depression. Therefore, given the potential risks of unrecognised depression, including suicide, Pies concluded that the removal of the bereavement exclusion criteria was a reasonable decision.

While Pies' argument is convincing, Mario Maj, chairperson of the WHO Working Group on the Classification of Mood and Anxiety Disorders, believes that removing the bereavement exclusion for depression may be "perceived as a further step in psychiatry's attempt to pathologise normal human processes" (Maj, 2008, p. 1374). In fact studies do show that the public holds a different view than mental health professionals. Carta et al.'s (2013) population-based survey in Sardinia (n = 1,200) found that respondents presented with vignettes containing information on loss events (e.g., death of spouse, betrayal by romantic partner, and job loss) were less likely to view depressive symptoms as evidence of depression. They also found that respondents were less willing to recommend professional help, and more likely to depend on self-help and support by family and close friends. This clearly shows that diagnostic decisions are not solely the realm of clinicians, but they directly impact whether an individual believes they have depression, and their help-seeking intentions. Carta et al.'s findings were similar to a population survey carried out in Vienna by Holzinger, Matschinger, Schomerus, Carta, and Angermeyer (2011). Given that Sardinia and Vienna differ in many respects (the former is traditional and rural, while the latter is a capital city considered the birthplace of psychoanalysis, and it has a high density of people working in mental health), Carta et al. concluded that the similar results reflect a divide between the public's perspectives of depression versus the DSM's perspective. Furthermore, the divide is not limited to a specific culture, but appears to be a more widespread phenomenon. Such a divide is likely to impact clinical encounters and therapeutic relationships, as patient views differ from that of physicians with respect to mental illness.

DSM vs. ICD classification. A brief mention should be made here about the ICD-10, published in 1992 by the WHO and used as the official coding system in the statistical departments of many countries, including Australia (ABS, 2007). The DSM, although developed by the APA for the classification of mental disorders in America, has become widely accepted among mental health professionals in many other countries (Andrews, Slade, & Peters, 1999). Therefore both operational diagnostic systems, which categorise known disorders based on number of criteria symptoms, are relevant to depression classification. One study examining the correspondence between ICD-10 and DSM-IV using the Composite International Diagnostic Interview (CIDI) – a structured set of questions systematically addressing each ICD-10 and DSM-IV criterion – showed a high level of concordance as 83% of those diagnosed with depression using either classification met criteria on both (Andrews et al.). The study's participants were drawn from a "disorder-enriched population sample combined with a clinic sample" (p. 3) – a phrase that was not adequately explained. Another similar study, but with a community population in Japan (Saito et al., 2010), concluded that ICD-10 seems more sensitive to mild cases of depression compared to DSM-IV. There was greater concordance for the more depressed respondents, as most of those diagnosed with severe or moderate depressive episode under the ICD-10 were also diagnosed with major depressive episode under the DSM-IV (Saito et al.).

Nonetheless, both the APA (Rounsaville et al., 2002) and the WHO (International Advisory Group for the Revision of ICD-10 Mental and Behavioural Disorders, 2008) have called for the two systems to be harmonised, as have researchers like First (2009). As the ICD-11 is only expected to be launched 2018 (Boerma, 2017), it is unknown whether this will be accomplished. Kessing (2004) noted that the ICD-10's severity subtypes for depressive episode (mild, moderate, and severe) have clinical utility because they predict risk of relapse and suicide, so he joins Paykel (2002) in arguing that ICD-10's way of categorising severity should be retained in future revisions. Clinicians would benefit from incorporating this aspect of classifying in their diagnosis of patients with depression because knowing their patients' risk of relapse and suicide could change the way they help their patients manage the condition.

The challenges of classifying depression could potentially be lessened if the condition was associated with biomarkers that could provide an objective diagnostic process. As it stands there are no definitive biological tests that can confirm major depression. A 2014 study by Redei et al. was touted as the first blood test to diagnose adult depression (Paul, 2015). The study found that blood biomarkers from 32 depressed primary care patients differed from 32 age-, gender-, and race-matched non-depressed controls. However, Redei et al. noted that larger studies were needed to confirm that the biomarker test does reflect vulnerability to depression. In recent years, there has been an upsurge of interest over biological markers for psychiatric conditions, fuelled in part by the desire to reverse the long-standing view of psychiatry as a "second-class citizen in science and medicine" (Singh & Rose, 2009, p. 202). Finding such biomarkers from genetics, neuroimaging, and physiology, might result in more accurate diagnostic practices and help align psychiatric classification with classification systems used in other fields of medicine (Moffitt et al., 2008). Hahn et al. (2011) developed a decision tree model based on interpreting a series of functional magnetic resonance imaging (fMRI) scans taken during various activities. The method had a sensitivity (ability to correctly identify individuals who have depression) of 80% and a specificity rate (ability to correctly identify individuals who do not have depression) of 87%. However, the authors noted that the sample size was small (30 patients with depression and 30 healthy volunteers) therefore extensive further study is needed before this method can be adopted clinically. Also MRI scanning will have to be considerably more affordable and accessible than it is now if this method is to be used widely. For now, clinicians utilise a diagnostic process that is based on behavioural symptoms and course of illness, as outlined below.

Depression Diagnosis

As most depression cases are treated in primary care (Harman et al., 2006; Katon & Schulberg, 1992), this section will elaborate on the diagnostic process used by GPs. The GP will first conduct a physical examination and interview, asking for the individual's current circumstances and symptoms ("Diagnosis of depression," 2015). Questions will cover family history of depression, onset and duration of symptoms, symptom severity, and prior treatment if any. It is important to check for thoughts of self-harm, suicide or death. The aim is to understand the biological, psychological and social factors that may affect the patient's mood – including the use of alcohol and/or drugs. The doctor may order laboratory tests to rule out medications or medical conditions (such as thyroid disorder or virus infections) that can cause symptoms similar to depression.

The psychological examination may include the use of depression rating scales which are psychiatric measuring instruments containing descriptive words and phrases to reflect the severity of depression symptoms within a time period, usually in the last week or month. These are not used to diagnose depression but rather to provide an indication of symptom severity so that patients who score above a certain threshold will undergo more thorough evaluation for depression. Numerous rating scales have been developed and they vary in length and format of questions. For example, the Beck Depression Inventory (BDI) contains 21 items (Beck, 1972) whereas the Patient Health Questionnaire-2 has only two questions (Whooley, Avins, Miranda, & Browner, 1997). Some rating scales are in the public domain and free to use such as the Clinically Useful Depression Outcome Scale (CUDOS) (Zimmerman et al., 2008) whereas others are copyrighted such as the BDI, where the use of each copy incurs a fee and photocopying is prohibited. The CUDOS can be completed in less than three minutes (Zimmerman et al.), has been validated and found to be a reliable measure to track symptoms (Beidas et al., 2015; Trujols et al., 2013; Zimmerman & Martinez, 2012).

Research indicates that GPs and other non-psychiatric physicians find it challenging to diagnose and treat depression: a meta-analysis by Cepoiu et al. (2007) showed that less than 40 per cent of depressed patients were recognised by their doctor. Fortunately, based on the later studies in the review, the authors were able to conclude that the rate of correct diagnosis had improved. Another meta-analysis by Mitchell, Vaze, and Rao (2009) found that GPs could generally identify about 50% of genuine cases. However, the authors also found that for every 100 cases in primary care, there are more false positives (n=15) than either missed (n=10) or identified cases (n=10). Based on the 41 studies in their review, Mitchell et al. discussed why GPs have difficulty in diagnosing depression. One reason is the relatively low rate of depression in primary care (about 10-20%), which favours the identification of non-depressed cases. Another is that patients may focus on somatic symptoms at the expense of psychological symptoms (Nuyen et al., 2005) or feel they have insufficient time to fully disclose their problems (Pollock & Grime, 2002). In light of this, Mitchell et al. proposed a multi-step assessment process to improve the accuracy rate. Instead of relying on a one-off assessment, individuals who might have depression are re-assessed over a period of 3-12 months. This aligns with other studies that have shown diagnostic recognition improves with better therapeutic relationships, more experienced clinicians, and increased GP-patient contact (Bushnell, 2004; Hickie et al., 2001; Wittchen, Hofler, & Meister, 2001).

Epidemiology of Depression

In all the countries where epidemiological surveys have been conducted, major depression has been identified as a commonly occurring disorder (Kessler & Bromet, 2013). Researchers have noted gender differences in depression (e.g., Blazer, Kessler, McGonagle, & Swartz, 1994; Piccinelli & Wilkinson, 2000; Weissman et al., 1996). A 10-nation survey by Weissman et al. involving 38,000 adults showed that the condition affects more women than men in each country, with the ratio being 2:1 in the US (Blazer et al.). Reviewing the global epidemiology of major depressive disorder (MDD) is complicated by the problem of heterogeneity in the data from various studies (Ferrari, Somerville, et al., 2013). A study by Ihara, Muraoka, Oiji, and Nadaoka (1998) showed that point prevalence for MDD in older Japanese men was 0.05%, whereas the same statistic for females in Afghanistan aged 15 and above was 73% (Cardozo et al., 2005). It is unclear how much of the variability in prevalence was due to true differences in epidemiology of the different populations or to differences in data collection methodology.

The World Mental Health Survey (WHMS) Consortium attempted to reflect true regional variability in epidemiology of major depressive episodes (MDE) through population surveys in 28 countries using a standardised method for collecting and assessing data (Bromet et al., 2011), using the WHO Composite International Diagnostic Interview (CIDI) Version 3.0. MDE differs from MDD in that MDE includes depressive episodes occurring in bipolar disorder whereas MDD excludes bipolar depression. Results showed regional variation in the 12-month prevalence of a MDE, from 2.2% in Japan to 10.4% in Brazil. Developed countries averaged 5.5%, similar to developing countries, which averaged 5.9%. The use of the CIDI, a standard data collection method, appears to have reduced the variability in prevalence to a more reasonable range compared to previous studies. However, the CIDI is arguably a severe limitation in itself because it has only been validated in western countries so it may not be reliable or valid in cross-cultural settings (Bromet et al.).

A different approach by Ferrari, Somerville, et al. (2013) involved a meta-analysis of primary data from community-representative studies. The aim was to explore the global distribution and sources of heterogeneity for MDD using a meta-regression. Unlike the WMHS, which tried to minimise heterogeneity through a standard protocol, Ferrari and colleagues adjusted for variability caused by methodological and design differences where feasible. They extracted point prevalence (i.e., current or past-month prevalence), 6- and/or 12-month prevalence data from 116 studies. Lifetime prevalence data was excluded because the authors deemed these to be more prone to recall bias (Moffitt et al., 2010). Although the focus was on MDD, they included for comparison estimates of depression not otherwise specified. They found that prevalence varied according to prevalence period, gender, year of study, depression subtype, survey instrument, region, and age. The median age of onset for depression and other mood disorders is, according to the WHO World Mental Health Survey of 28 countries, between 25 and 45 (Kessler et al., 2007) – which means depression often occurs during what may be considered the prime years of people’s lives.

Reports on duration of depressive episodes can vary depending on the definition of recovery used, the sample population, and when data was collected (Richards, 2011). For example, the first worldwide Global Burden of Disease (GBD) survey estimated mean episode length to be six months (Lopez & Murray, 1996), whereas other studies like the NEMESIS study (Spijker et al., 2002) and the 2008 Baltimore Epidemiologic Catchment Area Survey by Eaton et al. report a median episode length of three months. Without treatment about half of depressive episodes remit spontaneously (Mueller et al., 1999). However, Mueller and colleagues found six months after remission about 20-30% of patients will continue to suffer from residual symptoms that can be disabling and cause distress. In addition, between 50% and 85% of people who experience one major depressive episode will experience another in the future.

The widespread prevalence and onset of depression causes significant burden of disease, particularly in middle- and high-income countries (WHO, 2008). The consecutive GBD studies of

1990, 2000, and 2010, consistently identified depressive disorders (major depressive disorder and dysthymia) as a leading cause of burden (Ferrari, Charlson, et al., 2013). The GBD studies used disability adjusted life years (DALYs) to measure the global burden caused by diseases and injuries. One DALY is the loss of a healthy year of life and it is calculated by adding up years of life lived with disability (YLD) and years of life lost due to premature death (YLL) (Lopez & Murray, 1996). The GBD 1990 report showed that depressive disorders were the fourth leading cause of burden, responsibly for 3.7% of DALYs in 1990. A decade later depressive disorders accounted for 4.3% of DALYs and were the third leading cause of burden (Üstün, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004). The GBD 2010 study was broader in scope than earlier studies as it quantified the direct burden of diseases and injuries, as well as the burden attributable to risk factors in 187 countries (Ferrari, Charlson, et al.). Ferrari and colleagues calculated the overall burden of depressive disorders – the sum total of direct burden from MDD and dysthymia, as well as indirect burden as a risk factor for suicide and heart disease – to be 3.8% of global DALYs in 2010. By 2030 depression is projected to be the second leading cause of disease burden worldwide, second only to HIV/AIDS (Mathers & Loncar, 2006).

Co-morbidity between depression and medical illness, ranging between 10 to 40% (Goodnick & Hernandez, 2000), adds to depression's burden of disease. A depressed person is more likely than the general population to suffer from diseases such as cerebrovascular disorders, diabetes, irritable bowel syndrome, and some types of cancer (Jakovljevic cited by Uzun, Kozumplik, Topic, & Jakovljevic, 2009). Depression decreases quality of life (Östman, 2008) and increases the risk of heart failure by up to 40% (Gustad, Laugsand, Janszky, Dalen, & Bjerkeset, 2014). Depression is also often associated with other mental conditions such as anxiety disorders, substance abuse and eating disorders (Kessler et al., 2003; Weissman et al., 1996). People with depression have shorter life expectancies than those without (Chesney, Goodwin, & Fazel, 2014; Zivin et al., 2012). Premature deaths linked to depression can come in the form of illness or suicide (Wulsin, Vaillant, & Wells, 1999). Given the common and debilitating effects of depression there is a large body of research about treatment options, which will be covered in the final section of this chapter.

Depression Treatment

Numerous studies have researched the efficacy of different treatments for depression. By one count, there have been at least 60,000 empirical publications on depression since 1980 (Richards & Perri, 2002). Black Dog Institute, an Australian research organisation focusing on depression, categorises depression treatments into physical treatments, psychological treatments, and the third group consists of self-help and alternative therapies. Physical treatments include medication (antidepressants) and electroconvulsive therapy (ECT). The institute's factsheet on depression treatments said depression cases that are more biological in their origins (i.e., melancholic depression and psychotic depression) are more likely to need antidepressants; however, antidepressants should not be a first-line treatment option for children and adolescents. The factsheet also added that ECT is a relatively safe procedure. Despite its controversial image, ECT is recommended for severe cases of melancholic depression or post-natal depression where there is risk of suicide and significant impaired functioning (Black Dog Institute, n.d.).

There is a wide range of psychological treatments, the most well known of them is cognitive behavioural therapy (CBT). The Black Dog Institute website also lists mindfulness meditation, interpersonal therapy, psychotherapy and counselling. The psychosocial approach to treating depression sees it as a disorder of thoughts and emotions. Past and present risk factors are evaluated for its impact on an individual's mental wellbeing. Social class differences (i.e., poverty or lack of education), severe life events involving loss that the individual feels shame for, feelings of being trapped in negative circumstances (Brown, Harris, & Hepworth, 1995), childhood trauma and absence of mitigating factors such as social support and social capital, can precipitate, exacerbate and prolong depression. Individually tailored talk therapy is the mainstay of psychosocial treatments. However, patients may lack knowledge about psychotherapy and psychotherapists (Thompson, Bazile, & Akbar, 2004). A study by Cullinan, Veale, and Vitale (2016) showed that GPs also have limited knowledge about specific psychotherapies. This could be due to physicians' medicalised understandings of the illness, despite research showing that doctors do recognise the influence of social factors in depression (McPherson & Armstrong, 2012).

The third group of therapies, which can work alone or be a useful supplement to other treatments, include good nutrition, alcohol and drug avoidance, exercise, bibliotherapy, omega-3 (fish oil supplements), St John's Wort, light therapy, yoga, and acupuncture. Systematic reviews examining the effects of antidepressants, psychotherapy and alternative therapies have questioned the claimed superiority of these methods over placebos in treating various types of depression (Cooney et al., 2013; Cuijpers, Van Straten, Bohlmeijer, Hollon, & Andersson, 2010; Fournier et al., 2010; Smith, Hay, & MacPherson, 2010). The criticism against antidepressants is particularly strong from some researchers and popular media because roughly half of clinical trials found no differences between active treatments and controls (Kirsch & Sapirstein, 1998). In addition there is evidence of selective reporting in studies funded by pharmaceutical companies to make the drugs appear more efficacious than they really are (Melander, Ahlqvist-Rastad, Meijer, & Beermann, 2003; Turner et al., 2008).

A systematic review found that combining antidepressants and psychotherapy is slightly more efficacious for acute depression than either treatment alone (Khan, Faucett, Lichtenberg, Kirsch, & Brown, 2012). The study, believed to be the first of its kind, also found that antidepressants and psychotherapy on their own are not significantly different from alternative therapies such as exercise or acupuncture or active intervention controls such as bibliotherapy or sham acupuncture. This suggests that it does not matter which type of treatment is offered as long as the depressed patient is involved in an active therapeutic programme (Khan et al.). The authors proposed that treatment type be chosen based on clinical presentations, risks, and patient preferences and acceptance. I argue that these factors can only be understood and evaluated by clinicians if they successfully elicit patient perspectives.

Reducing the impact of depression through effective and timely treatment is a challenge because there appears to be no single common pathway for depression causation (Schotte, Van Den Bossche, De Doncker, Claes, & Cosyns, 2006). The beginning of this chapter on depression theory provides much evidence of this. In addition, depression understandings are highly individual in nature (Cornford, Hill, & Reilly, 2007). While there are overarching aspects of depression that can be said to be generally true of patients, every individual's experience with the condition is unique and deeply personal. This gives rise to challenges when communicating about depression and has implications on how well GPs and patients understand each other's perspectives during consultations. A recent review by Haslam and Kvaale (2015) provide evidence that the increasingly prevalent biological and genetic explanations of mental disorders (Pescosolido et al., 2010) affect the perspectives of patients and clinicians, as well as the public.

A vignette study (Reavley & Jorm, 2011) involving a national sample of over 6,000 participants in Australia found that 86% of people thought "chemical imbalance" was a likely or very likely cause of depression. However, in the same survey, 97% of people also thought that "day to day problems" was likely or very likely to cause depression. Yet the non-biomedical view of depression appears to be ignored as depression is often treated with antidepressants alone (Olfson & Marcus, 2010; Robinson et al., 2005). This has resulted in drastic increases in antidepressant prescription rates – Australia saw a 95.3% increase between 2001 and 2011 (Stephenson, Karanges, & McGregor, 2013). As mentioned earlier, the effectiveness of antidepressants in reducing the symptoms of depression remains contestable (Casey, 2013). Patients have also expressed concerns about the effectiveness of antidepressants in addressing the root cause of depression (Givens et al., 2006). In addition, several international studies, including one that involved 16 European regions, have concluded that rates of suicide and attempted suicides have not consistently fallen despite increased antidepressant use (Safer & Zito, 2007; Van Praag, 2002).

New treatments for depression are constantly being developed especially for treatment resistant depression, a clinical term used to refer to MDD cases that do not show remission after receiving two different antidepressants (Wijeratne & Sachdev, 2008). Some examples include deep brain stimulation (Lakhan & Callaway, 2010), repetitive transcranial magnetic stimulation (Euba, Panihhidina, & Zamar, 2015; Lefaucheur et al., 2014), and ketamine infusions (Bobo et al., 2016). One of the more unorthodox treatments involves injections of botulinum toxin A (BTA), commonly known as botox, to reduce glabellar frown lines, which are most produced when expressing negative emotions (Kruger & Wollmer, 2015). Kruger and Wollmer cited three randomised control trials that have shown significant improvement in depression symptoms in participants who received BTA injections compared to those who received saline placebos. The proposed mechanism of action is that the BTA paralyses the muscles required for frowning and thereby interrupts a feedback loop from the patient’s face to the brain that would usually strengthen and sustain the negative emotions linked to depression. Although some of these newer treatments are promising, it is unlikely that a magic cure will ever be found that can eliminate depression completely for all individuals. The nature of depression is simply too complex for that. In light of this, it makes more sense for GP and patients to develop a shared understanding that promotes greater treatment adherence (regardless of what treatment is chosen) and better therapeutic alliance, which ultimately leads to improved outcomes.

Importance of Perspective

As mentioned previously, the combination of pharmacology and psychotherapy remains the best approach for treating depression (Karyotaki et al., 2016; Schotte et al., 2006) but this requires a biopsychosocial depression model that integrates its biological and psychosocial aspects (Schotte et al.). The biopsychosocial model was promoted by Engel, who posited that biological, psychological, and social factors all play a role in health and disease (1977, 1980). A constructive biopsychosocial view of depression that is clearly communicated between clinicians and patients (and their families) would provide the rationale for combination treatments and conceivably improve treatment adherence and effectiveness.

Researchers have begun to compare the perspectives of depressed patients with their GPs. A qualitative US study comparing GPs' and patients' views on depression care found that one of the three key processes that give rise to satisfactory care was the patients' search for a personal understanding of their depression (Keeley, West, Tutt, & Nutting, 2014). This demonstrates that patient understanding of their condition is of prime importance and my research will further expand on this. The importance of patient perspective supports the rationale for not using verified diagnoses of MDD as an inclusion criteria. Sections of this chapter have shown that the definition, diagnosis and classification of depression are arbitrary at best. Instead, my research will involve the perspectives of participants who self-report consulting a GP for what they consider to be depression, and also the perspectives of their GPs (more details in Chapter 7). Patient perceptions are key because they influence patients' help-seeking behaviour (Okello & Neema, 2007), illness experience, and treatment choice (Buus et al., 2012). GP perceptions are equally important because both patient and doctor viewpoints should be integrated to establish a strong therapeutic alliance (Nosè et al., 2012). Ardito and Rabellino (2011) have argued that it does not matter what kind of psychotherapy a patient is given because a good therapeutic alliance is a stronger predictor of positive clinical outcomes than treatment type. Doctor and patient conceptualisations of depression will interact dynamically during the clinical encounter so there is a need to research the significance of differing depression conceptualisations in GPs and patients. The framework I use to study the perspectives of patients and GPs is Kleinman's concept of explanatory models (Kleinman et al., 1978), which I will explain in the next chapter.

Chapter 3: Explanatory Model

A significant portion of the previous chapter elaborated on the different understandings of depression. In investigating how individuals think and talk about illnesses, researchers have tried to establish what theoretical frameworks are suitable for capturing such perspectives (Kokanovic, Butler, et al., 2013). In this chapter I elaborate on psychiatrist and anthropologist Arthur Kleinman's explanatory models (EMs) approach and how the EM framework is particularly suited to my research on depression. I also explain the rationale for choosing the analytical term of EMs to explore patient and doctor perspectives instead of other approaches. The final portion of this chapter will be a review of previous research using EMs and how these inform my studies.

Explanatory Models

EMs are defined as the different "notions about an episode of sickness and its treatment that are employed by all those engaged in the clinical process" (Kleinman, 1980, p. 105). Hallenbeck

(2003) gives the example of how Western medicine views the body as a machine and illness as a malfunction to be repaired by the physician whose role is similar to that of a mechanic. This mechanistic view differs from non-Western views of illness as an imbalance of forces (e.g., the Chinese concept of yin-yang). Different beliefs on the causation or nature of sickness that stem from cultural differences may lead to poor communication and misunderstandings between patients and doctors, which may strain the therapeutic relationship and negatively affect treatment outcomes. Clinicians wanting to develop culturally appropriate therapies require an adequate understanding of how cultural disparities influence illness behaviour, help seeking, patient expectations, and perceived quality of care (Mumford, 1995). It is important to note that the Chinese example of yin-yang provided earlier is solely for illustrative purposes because in reality, culture is dynamic and not simply a matter of ethnicity, language, or nationality (Kleinman & Benson, 2006). This is why Kleinman and Benson argue that cultural competency training for physicians is more than prescribing how to treat patients of certain ethnicities.

Such ethnicity-based guidelines are at best assumptions. Instead, clinicians need to value and intentionally elicit each individual patient's EM so as to understand the nature of the illness from the patient's perspective and the major concerns he or she has. To facilitate such discussions, Kleinman et al. (1978, p. 256) recommended asking simple open-ended questions about the top five clinical issues that are important to patients, that is, aetiology, symptoms onset, pathophysiology, course of illness, and treatment. These questions provide a basis for other queries to help explore the patient's views so clinicians have a solid foundation and understanding from which to negotiate a mutually agreed treatment plan. From a medical anthropology perspective, the EMs approach enable a clinician to undertake a "mini-ethnography" (Kleinman & Benson, 2006, p. 1674) on the patient's view of their condition. Ethnography is a central methodology in anthropology where a researcher describes knowledge from the point of view of the subjects. The focus on EMs is congruent with patient-centred models of service delivery (Williams et al., 1999) and EM approaches have been used in a range of patient populations suffering various conditions such as adolescent spina bifida (Kinavey, 2006), schizophrenia in India (Charles, Manoranjitham, & Jacob, 2007), low-income African American women (Waite & Killian, 2009), and immigrants with depression in Australia (Kiropoulous & Bauer, 2011).

Besides taking into account the patient's EM, it is also important to understand the EMs of health professionals. Kleinman et al. (1978) explained that comparison of patient and doctor EMs allows doctors to recognise severe discordance that could lead to challenges in managing their patient's condition. Comparing of EMs also helps the doctor to understand which portions of his EM needs to be more fully explained to patients, and their carers, and to choose the most suitable form of patient education. In addition, EM comparisons aid in resolving conflicts that may arise, not due to unequal information, but because of differences in priorities and values. Kleinman et al. believe that an essential component of the clinical encounter involves negotiations between patient and doctor EMS that have been expressed. The act of negotiating is necessary for all clinical encounters, not just those involving patients and doctors from ostensibly different cultural backgrounds. With respect to understanding depression, research suggests that doctors tend to have more biomedically oriented perspectives compared to their patients (e.g., Casini et al., 2013; Elliot, Maitoza, & Schwinger, 2012).

While this thesis is focused on the role of depression perspectives in primary care, it is important to note that how society views depression and other mental illness has wider ramifications. For example, when there was an increase in suicides among older men in the UK, it sparked debate whether mental illness can be caused by social factors such as job insecurity, unemployment, and welfare reforms. While acknowledging that not every depression case has economic or political roots, political theorist Mark Fisher (2012) criticised the dominant approaches to depression that attribute the disorder to individual brain chemistry and/or early childhood experiences. Consequently, pharmacotherapy and most forms of psychotherapy were inadequate because they ignore the social causation of depression. Fisher also wrote about his struggles with depression, and news of his suicide (Doran, 2017) during the writing of this thesis (mid-January 2017), tragically underscores the importance of holistically addressing depression.

Before further discussion of current literature involving depression EMs, I will overview other analytical terms that I could have invoked and which are used by other researchers to encapsulate perspectives about illness. I then provide justification for my decision to use EMs.

Other Theoretical Frameworks

Health belief model. There are different ways to capture illness perspectives in current literature but not all are concerned with the patient's subjective reality, which is the focus of my thesis. One of the early approaches that preceded the EM framework is the health belief model (HBM). Developed in the 1950s by social psychologists employed in the US Public Health Service, the HBM aimed to explain and predict behaviour with respect to the utilisation of health screenings (Rosenstock, 1966). The model states that an individual's perception of their susceptibility to a health condition, the severity of the condition, the benefits of taking preventative actions, and the barriers associated with the preventative behaviour, will determine whether they adopt preventative measures. Since then the HBM has been widely tested and found to be a robust model of health behaviour (Carpenter, 2010; Glanz & Bishop, 2010; Janz & Becker, 1984). The HBM's component of perceived seriousness parallels the question about severity and course of illness identified by Kleinman et al. (1978) and discussed above. The perceived benefits component of the HBM could arguably be compared to the question of what patients would like to achieve through treatment.

However, because the HBM was originally developed to counter the low uptake of tuberculosis screening services (Carpenter, 2010), it is strongly focused on responses to preventative public health interventions (Glanz & Bishop, 2010). It is therefore more suited to research into objective health behaviours to avert a medical condition than the topic of my thesis, which is on subjective perspectives of both primary care patients who have depression and their doctors. I would contend that EMs could be considered a prequel to HBM because EMs focus on illness perceptions while HBM focuses on actions that flow out of an individual's illness perceptions. Arguably this emphasis on a patient's understanding and not actions is why Kleinman's EM includes the dimension of perceived aetiology or causation of a condition, while HBM does not. The importance of aetiology cannot be overlooked as it influences treatment options; for example, Iselin and Addis (2003) showed that beliefs about depression causation affect perception of treatment effectiveness. Hence, the absence of aetiology within the HBM framework limits its suitability for my thesis.

Exploratory maps. Another approach proposed by Williams and Healy (2001) involves the use of exploratory maps. Williams and Healy interviewed 15 individuals who had been newly referred to a community mental health service in rural northern Wales (UK) and found these patients expressing a variety of perceived causes for their illness within the same interview. The researchers argued that the concept of explanatory models was too fixed for such new patients, as it was more suited for those with an established diagnosis of depression. Based on the fluidity of beliefs among their participants, they argued that the term exploratory map was more suited. Exploratory maps outline how individuals try to understand the reasons behind their distress with initial vague concepts that eventually get clarified, similar to how a cartographer may draft out a physical map to give a general sense of the geography of the land and then fill in more details after further exploration. While the concept of an exploratory map may be a good analogy for the gradual way that a patient's perspective becomes clearer over time, there does not appear to be further structure or process in Williams and Healy's framework to make it immediately applicable and operationally useful for my thesis. In contrast, research using Kleinman's EMs framework can be operationalised using the topics and questions that he proposed for clinical encounters, as explained in the beginning of this chapter.

When Kokanovic, Butler, et al. (2013) examined published studies and extracts from interviews of primary care patients in Australia they found that patients used EMs, exploratory maps, and illness narratives to talk about depression. An illness narrative differs from the other two conceptual frameworks in that its analytic focus is chronology, or the arrangement of events associated with change according to the temporal order in which they happened (Hyden, 1997). Kokanovic, Butler, et al. contend that patients used all three frameworks concurrently but opined that EMs would only be useful for patients who feel that a clear biochemical explanation for their depression aids their recovery by helping them make sense of their symptoms.

However, I would argue that the EM concept could be applied in such a way as to incorporate the fluidity of exploratory maps and the temporal nature of illness narratives. It depends on how EMs are discussed in the clinical encounter, such as a GP who understands that a newly diagnosed patient is likely to have several changeable beliefs about the illness can revisit the patient's EM over a few consultations. As noted in Chapter 2, increased frequency of GP-patient contact improves diagnostic accuracy (Bushnell, 2004) so this brings about twin benefits of the GP being more confident of making the correct diagnosis, while the patient has more opportunities to express different aspects of their depression EM. Similarly, a GP who believes in the importance of illness narratives can extract answers to Kleinman's proposed questions from the stories that patients tell about what their illness means to them. Compared to the patient focus of exploratory maps and illness narratives, the EM concept can be used more broadly to explore explanation of disease from the perspective of doctors (Kleinman, 1980), making it a useful construct for my study on depression patients and their GPs.

Common sense model. Around the same time that EMs approach was introduced, Leventhal and colleagues (Leventhal, Meyer, & Nerenz, 1980; Leventhal, Nerenz, & Steele, 1984) proposed the common sense model of illness representations (CSM), also known as the self-regulatory model. The CSM outlines how people form subjective interpretations when reflecting on their physical or psychological symptoms. These illness representations have implications for how patients conceptualise and manage their condition (Leventhal et al., 1980; Leventhal et al.,1984). Originally Leventhal and colleagues had listed four dimensions as the building blocks of the illness representations. They were identity (label and perceived symptoms of the illness), timeline (perceived duration of the illness), perceived cause of the illness, and perceived consequences of the illness. The fifth dimension of control/cure, that is, the perceived controllability and/or curability of the illness, was proposed by Lau and Hartman (1983), and subsequently acknowledged by Leventhal and Nerenz (1985) to be an additional constituent of most illness representations.

Leventhal, Diefenbach, and Leventhal (1992) applied the CSM to understand why patients would undertake change to improve their condition. Leventhal et al.'s focus was mainly on threat recognition and treatment adherence, beginning their article by quoting from a diabetic adolescent who does not monitor his blood sugar levels because he feels well and who believes insulin does not help him because the injections make him feel unwell. However, their observations are relevant to my thesis because they underscore the role of perspective and, perhaps even more importantly, the negotiation of perspectives – which, as mentioned earlier, is also highlighted in Kleinman's EM concept. The CSM derives its name from Leventhal et al.'s argument that self-regulative systems require coherent integration of the key components of various treatments – they need to "fit together and make sense!" (p. 153). The components are cognition-interpretation of the problem (Beck, 1967), sense of self-efficacy from rehearsing effective actions (Bandura, 1977), and criteria to evaluate outcomes. These components are similar to aspects of Kleinman's EM concept explained earlier; for example, the cognitive interpretation of the problem can be arguably addressed through Kleinman's proposed question on causation and the criteria for outcome evaluation can be derived from the question on what patients hope to achieve from treatment.

Leventhal et al. (1992) also listed three domains in which issues of coherence often arise, and the second of these – coherence at the cultural and interpersonal levels (particularly between the individual and their health providers) – is especially relevant to depression. This is because the way depression is treated and managed, both in clinical and non-clinical settings, is highly dependent on perceptions of the illness, and perceptions of possible treatments and coping mechanisms. Leventhal et al. observed that an individual who is sick will have to negotiate with loved ones, colleagues, and doctors, over topics like being excused from work and family responsibilities, and choosing the right treatment for a recovery. Although complex, the negotiation process can be so familiar and instinctive that is not recognised as a negotiation. However, the ease of negotiation depends on whether similar perspectives on the illness and its management are held by the person who is ill, and their family, friends, and doctors. Leventhal and colleagues added that non-Western patients who use both Western and traditional medicine often do not have shared perspectives with their Western-trained practitioners, and cited Kleinman describing storefronts of traditional and

Western practitioners in Taipei’s old Lung-Shan district. This was where patients sought care according to how well they perceived their symptoms and self-diagnoses fitted within traditional or Western models of disease.

I can validate this perspective with respect to unshared viewpoints. During my childhood (in Malaysia) I was taken to Western-trained doctors and admitted to hospital frequently for my severe asthma, but when I sprained my ankle, the Chinese traditional bonesetter treated me. With a few sharp twists, my ankle would be set right and I would be able to walk normally after a few days. In contrast, Western-styled treatment often seemed to only address symptoms with painkillers and cold compresses. This resulted in a perception that Western doctors did not address the main underlying problem, and consequently such injuries were expected to have a much longer recovery period. My cousin sprained her ankle in a camp and was put in a cast by the camp’s first-aid personnel, but after the camp she was taken to a bonesetter who immediately broke open the cast and proceeded to twist the ankle back into place.

While Leventhal et al. (1992) only cited the storefronts example from Kleinman when explaining the CSM, other researchers have more readily adopted Kleinman’s approach and terminology. Bhui and Bhugra (2002) wrote on the clinical practice and research implications of EMs for mental disorders. These authors did not include any discussion about why they chose to use the term EMs. Sumathipala and colleagues (2008) also appeared to prefer the term EM in their work on patients with medically unexplained symptoms. Although they mentioned that the different terms used to refer to illness perspectives, such as the illness perception model, self-regulatory model, parallel process model, all originate from Leventhal et al.’s CSM, Sumathipala et al. chose to use the term EM in the title of their article. This suggests that perhaps the term is most easily understood.

In sum, the CSM largely articulates the same principles of Kleinman’s EMs. However, the latter does not presume that what is common sense to one individual is common sense to another. Thus I use Kleinman’s terminology.

Explanatory Models Concordance

Notwithstanding the use of different terms for illness perspectives by different researchers, many have underscored the importance of concordance between patient and physician perspectives. Leventhal et al. (1992) said treatment adherence appears to be high when doctor and patient share the same perspectives, agree on treatment choice, and use the same criteria to appraise outcomes. Similarly, Bhui and Bhugra (2002) said understanding EMs enables clinicians to appreciate their patients' response to illness, develop a better therapeutic relationship based on empathy, and more effectively communicate their own interpretations and treatment recommendations. The opposite is true when doctor and patient perspectives differ. Individuals from a variety of backgrounds, and not just non-Western cultures as commonly thought, can perceive psychiatric disorders such as depression as somatic conditions (Bhugra & Mastrogianni, 2003). Leventhal et al. noted that psychological labels such as depression are rejected due to their stigmatising effect (the next chapter will elaborate more on mental illness stigma). In the case of depression, the pattern of preferring physical or physiological over psychological has important consequences as it influences interpretation of symptoms (e.g., feeling tired instead of sad), perception of causation (e.g., pathogens rather than stress), and choice of treatments (e.g., medication and not psychotherapy). When there is interpersonal inconsistency blocking the formation of a coherent system, the patient may keep searching for alternative explanations for their disorders and they are likely to stop psychiatric treatment eventually (Leventhal et al.). With respect to depression, even if patients continue with their doctors, there may be limited therapeutic outcomes if perspectives are not shared or negotiated.

A review of published research on medically unexplained physical symptoms (MUPS) in primary care by Burton (2003) showed that patient satisfaction is highest when patients feel empowered by their therapists. The relevance of MUS to depression is based on Burton's observation that the causative factors for MUS are similar to those for depression and anxiety. Another link is the finding of Burton, Mcgorm, Weller, and Sharpe (2011) that patients referred to specialists for their MUS often experience depression and anxiety. Patients with MUPS felt that empowering medical explanations, where clinicians provided "tangible, non-judgmental explanations which legitimised the patient's suffering and offered opportunities for self-management", were helpful (Burton, 2003, p. 237). Such explanations were foundations upon which patients could build a recovery plan or at least develop coping strategies for their condition in cooperation with their doctors. Empowering explanations are markedly different from the other two types of explanations identified by Salmon, Peters, & Stanley, 1999 – rejecting (where doctors are seen as negating the reality of their symptoms) and collusive (where doctors give in to the patient's explanation of symptoms but lose credibility for doing so). A fundamental reason my thesis focuses on how patients with depression and their doctors perceive and explain the condition is because research has shown that medical explanations, as a part of psychoeducation, play a key role in helping individuals cope with their symptoms. Colom (2011) described the aim of psychoeducation as the establishment of holistic understanding of affective disorders in patients so they can better manage their condition. A systematic review by Tursi, Baes, Camacho, Tofoli, and Juruena (2013) on the benefit of psychoeducation for unipolar depression found that greater awareness about depression and its treatment was linked to an improved clinical course of depression in individuals. Tursi et al. noted a paucity of research into the value of psychoeducation on adults with depression, despite the fact that psychoeducation has been extensively described as a psychosocial treatment that supplements pharmacotherapy. Nevertheless, the authors concluded from the 15 studies they reviewed that there is evidence suggesting that psychoeducation can better the prognosis, treatment compliance, and functioning of patients with depression.

So far in this chapter, I have explained what EMs are and why I have chosen the EMs framework to study depression perspectives in primary care patients and doctors. I now review the current literature on depression EMs. In so doing, it becomes apparent that there is insufficient research that compares the depression EMs of patients and GPs. My thesis addresses this gap.

Depression Explanatory Models

Patients' explanatory models. Kleinman's EM concept has been the theoretical foundation of different studies into the depression EMs of patients. An example of a patient study conducted in a Western context was Schreiber and Hartrick's (2002) interview of 43 women who had been treated for depression in Canada. Research has consistently demonstrated that women are more at risk for depression than men, whether they are in high or low-income countries (Piccinelli & Wilkinson, 2000). Schreiber and Hartrick investigated coping strategies adopted by women with depression and distilled key aspects of women's depression EMs (including perspectives on causation, symptoms, physiology, illness duration, and treatment). The researchers found that women used the biomedical EM to manage certain aspects of stigma associated with depression, such as the inner shame associated with being depressed and the fear of being judged or discriminated because they had depression. However, the same EM reinforced other aspects of stigma, namely anticipated stigma from others. The different facets of stigma and its relevance to depression and EMs will be elaborated in the next chapter.

Schreiber and Hartrick (2002) also found that having a more biologically oriented perspective had unintended effects that seemed to hinder the patients' recovery from depression. For example, since depression was seen as a biomedical problem, it logically required a biomedical solution, which shifted the ownership of the problem away from the patient and undermined the patient's inter- and intrapersonal efforts to manage it. For example, the non-medical treatments that the participants had been engaged in were seen as ineffective since they were not able to directly change the neurotransmitter levels or genetic factors that were deemed responsible for the depression. Another inadvertent effect of adopting the biomedical EM was that it caused patients to focus on managing the more observable and definable symptoms of depression instead of taking a more holistic view. Schreiber and Hartrick's study thus shows that the EM adopted by a patient can strongly influence the patient's recovery journey. The authors observed that adopting the biomedical EM for depression tended to overshadow recognition of the complexity of the women's experiences. This resulted in an over-emphasis on treating the biological aspects of depression while ignoring the intricate interconnections between the different aspects of the participants' lives. I would argue that such an approach is unlikely to lead to sustained recovery from depression.

Schreiber and Hartrick's (2002) study involved highly educated urban women and their findings differed from Hauenstein's (2003) observations of women living in the impoverished Appalachian region of the southern United States. Hauenstein's review of the limited literature on depression in rural women, supplemented with insights gained from over a decade of research with this population, concluded that poor Southern women do not subscribe to the biomedical EM of depression. They are unlikely to interpret unusual behaviour as symptoms of mental illness and more likely to believe that God or spirits have a role in disease causation and recovery. In light of this, Hauenstein suggested involving a member of the clergy in helping to mitigate depressive symptoms. This makes sense given the common practice among people in distress to seek help from religion and religious figures (Koenig & Larson, 2001). In the previous chapter, I had mentioned that the church's chequered past in managing depression. Fortunately, in recent years, anecdotal evidence suggests that there is a move to equip churches on ministering to people with depression and other mental illnesses.

A study of adult hospital patients in a non-Western setting had similar findings to Hauenstein's rural Western study in concluding that the biomedical EM was not prevalent (Grover et al., 2012). Grover and colleagues interviewed 164 patients in North India who was admitted with first episode depression according to ICD-10. The researchers evaluated the patients' EMs using the causal models section of the Explanatory Model Interview Catalogue. Using an open-ended query "People explain their problems in many different ways, sometimes ways that are different from what their doctors or other family may think. What do you think is the cause of your problem?" (p. 252), patients were encouraged to describe as many causes as possible for their depression symptoms. The most frequently cited reasons were from the karma-deed-heredity category (77.4%);

karma refers to the Hindu and Buddhist belief that the sum of a person's deeds in this and previous lives will determine their fate in future reincarnations. The next most frequently cited reasons for depression were psychological causes (62.2%), weakness/nerves (50%) and social causes (40.2%). The patients expressed many different EMs simultaneously; one-fifth of them had two EMs, while three-fifths believed three or more EMs. The mean number of explanations was 2.85 (SD -1.48, range 1-6). When asked to choose the most important cause of their depression, two-fifths of the patients were not able to do so. The others most frequently gave psychological causes as the most important explanation for the depression, followed by social causes, and the karma-deed-heredity category. Grover et al. stressed the importance of clinicians being aware that their patients have multiple EMs so that they can tailor treatment accordingly for optimum adherence and outcomes.

Grover et al.'s (2012) study also looked at factors that may affect a patient's EM or EMs. There were significant differences in EMs associated with gender. Males more frequently cited alcohol ingestion and work problems as reasons for their depression compared to females. Females more frequently cited the evil eye and other supernatural causes for their depression compared to males. Education status was another factor associated with significant differences in EMs. Patients who had less than 10 years of formal education were more frequently citing general weakness as a reason for depression than patients with more than 10 years of education. Interestingly, the more educated patients did not allude to biomedical reasons for their depression, contrary to Schreiber and Hartrick's findings previously mentioned. Instead, Grover et al. found that patients with more than 10 years' education more frequently blamed pollution and supernatural causes for their depression than patients with less than 10 years' education. Gender and educational levels are important predictors of EMs and will be included in my study's demographic questions (further details in Chapter 7). The study will also ask for other participant characteristics such as age, ethnicity, and socioeconomic status, which have been the focus of other depression EM studies (Alemi, Weller, Montgomery, & James, 2016; Lawrence et al., 2006; Pereira et al., 2007). Participants also reported health characteristics such as whether they had a medical diagnosis of depression and presence of co-morbidity, as EMs could be affected by these factors as well.

As mentioned earlier, patient EMs do affect their recovery. Some EM studies have sought to understand how patients' beliefs influence their treatment adherence. For example, Buus et al. (2012) interviewed 16 depressed patients in their first year after being discharged from a hospital in southern Denmark and found that having a sense of control was paramount. The patients generally had psychosocial reasons for their depression, which gave them a sense of direct and personal influence over their recovery. Although medicine was not central to these patients, they took antidepressants initially because doing so increased their sense of certainty, control, and hope for future recovery. Over the course of the study, nine of the 16 patients gradually recovered and planned to continue taking medication out of fear of relapse. The other patients who felt the antidepressants were ineffective or had intolerable side effects began to de-medicalise their perspective. They adopted EMs that aimed to rationalise non-adherence and other personal actions to regain their sense of control over the issues they were facing. As mentioned earlier in the CSM discussion, perceived controllability is an important aspect of illness representations (Leventhal & Nerenz, 1985). My thesis takes into account that patients may have differing ideas about how much control they have over their depression (more details in Chapter 7).

In fact, Kokanovic, Bendelow, and Philip (2013) found that lay accounts of patients diagnosed with depression fluctuate between medical and non-medical discourses, highlighting the limitations of the biomedical approach in depression diagnosis and treatment. However, even though there are these limitations, it is critical to take into account the biomedical perspective because it is so prevalent. One way to take into account the biomedical perspective of depression is to determine to what extent patients and their doctors engage in the biomedical EM, which is the approach that I take in my thesis. Through awareness, it would then be possible to negotiate alignment.

Laypersons' explanatory models. Depression EM studies not only involve patients. Dejman et al. (2010) interviewed participants who did not have depression from Iran's three largest ethnic groups (Fars, Kurds, and Turks) about depression EMs. Although the study involved a non-patient population, it was beneficial to show how caregivers, family members and friends feel and perceive individuals with depression in their midst, and what measures they would take should they experience depression at some point. Through focus group discussions on a case vignette describing a woman with DSM-IV major depression, Dejman et al. found that participants perceived depression symptoms as temporary and caused by external stressors such as family conflicts and war (particularly among the Kurdish participants who had experienced war with Iraq in the 1980s). This finding suggests a more psychosocial EM of depression. A few of the participants who were more highly educated expressed a more biomedical EM, citing biological factors such as hormonal problems and pregnancy as the cause for the depression. Dejman et al. found that participants' views on aetiology of depression influenced their views about help seeking as most of them felt the woman in the vignette should empower herself to cope with life problem. At the same time, participants did perceive that consulting a psychologist could be helpful as an avenue for expressing emotions and to increase one's internal fortitude against challenges. However, the majority of participants believed that the most essential solution, regardless of the cause of depression, was to rely on God for greater strength. Medication was seen as a last resort only if symptoms were prolonged or worsening. Many participants were hesitant to propose medication due to fear of stigmatisation and adverse side effects; perhaps for that reason, the female Turkish participants in the Dejman et al. study expressed a preference for herbal medication. Again this finding highlights the importance of stigma, which is a key variable in this thesis.

In Australia, Kokanovic, Dowrick, Butler, Herrman and Gunn (2008) interviewed Anglo-Australians and East African refugees and found differences in lay accounts of depression. Anglo-Australians see depression as a socially isolating, individual experience that is associated with adverse life events. In contrast, Ethiopian and Somali refugees view depression as a disorder that is communally derived and experienced (Kokanovic et al.). The authors and other researchers, such as Shaw (2002) and Skultans (2003), argued for the term lay accounts over lay beliefs. Shaw contends that the latter suggests an immutable analytical category, whereas in reality lay people in Western society are frequently exposed to professional representations of disease. For example, Skultans found that medical consultations aid in acculturation to biomedical EMs and shape a layperson's experience of depression. For this reason, Kokanovic et al. (2008) contend that lay accounts are necessarily contextually defined through a "negotiation of identity" between the individuals communicating (p. 455). Again, the aspect of negotiation of perspectives is pertinent, as mentioned earlier in this chapter.

Doctor's explanatory models. Research has found that the match between patient and clinician EMs is highly influential in the treatment process (Cohen, Tripp-Reimer, Smith, Sorofman, & Lively, 1994). Studies show variation between the EMs of patients and medical professionals in physiological conditions such as diabetes (Cohen et al.) and hypertension (Blumhagen, 1980). There could well be greater variation for psychological conditions like depression, which have no biomarkers. Few studies have focused directly on doctors' understanding of depression (Thomas-Maclean & Stoppard, 2004) and most of these appear not to use the term explanatory model from Kleinman. McPherson and Armstrong's (2012) systematic review of qualitative research examining GP management of depression found 10 studies, representing a total of over 200 GPs, did not use the specific term explanatory model in their database search. However, I included their study in this literature review on Kleinman's EM approach because one of the main themes they identified overlaps with Kleinman's EM questions that were listed previously. For example, under the main theme of negotiating the nature of depression, McPherson and Armstrong identified the following subthemes in the GP studies: (1) "'normal' response" (p. 1152) where depression is perceived as a natural response to stressors and social circumstances – this maps onto Kleinman's causation and onset questions, (2) "medicalisation, dissonance, and 'true' depression" (p. 1152) – this maps onto Kleinman's pathophysiology and severity questions, (3) "curability" (p. 1153) – this maps onto Kleinman's treatment-related questions).

Elaborating on the second subtheme, McPherson and Armstrong (2012) highlighted two studies where GPs preferred the biomedical EM. One was a study of 17 Swedish GPs by Andersson et al. (2001) and the other was a study of 20 Canadian GPs by Thomas-Maclean and Stoppard (2004). The GPs described depression as a biological disorder caused by a biochemical imbalance or other organic causes including chronic pain. However, in both studies, GPs characterised a condition as "true" (Thomas-Maclean & Stoppard) or "proper" (Andersson et al.) depression only when there were no social factors in their patients' lives that could explain the symptoms. This spurious distinction between what is real depression and what is not appears to be a weak strategy to try and resolve the conflict between the psychosocial aspects of depression and the biomedical view of depression. This unresolved inconsistency has been highlighted in another systematic review of 13 qualitative studies on physicians' attitudes regarding depression diagnosis in primary care (Schumann, Schneider, Kantert, Löwe, & Linde, 2012). The studies involved 239 primary care providers from seven countries (UK, USA, the Netherlands, Sweden, Australia, Canada and Germany). Schumann et al. said most GPs believed that "depression is the condition we diagnose in normal people with overwhelming problems. It is mostly reactive, social" (p. 258).

Thomas-Maclean and Stoppard (2004) concluded that the dissonance arises from the influence of Cartesian dualism inherent in the GPs' medical training and hinders them from viewing depression holistically. Other studies reflected this opinion. For example, Rogers, May, and Oliver (2001) argue that although general practice professes to provide holistic care and individual doctors recognise the unique social and psychological experiences that lead to the patient's symptoms, the pathology of depression remains at the core of what doctors do. Defining depression as a medical condition first and foremost necessarily means that people with depression are just like any other patients with a medical problem (Rogers et al.). Similarly, in a study by Johnston et al. (2007), GPs portrayed depression as a "highly individual concept, imbued with moral and cultural values" (p. 874), but at the same, they encouraged their patients to consider it as a treatable illness like any other medical condition. The reason for doing so was supposedly to minimise stigma. As mentioned earlier, stigma is a key factor in this thesis. One of my aims is to examine the relationship between biomedical EM for depression and stigma, in particular self-stigma. The different facets of stigma will be explained in the next chapter.

As mentioned earlier in this chapter, patient depression EMs become de-medicalised when antidepressants are ineffective (Buus et al., 2012) and this is true for GP EMs too. McPherson and Armstrong (2009) interviewed 20 GPs on how they would conceptualise depression when discussing patients whose condition did not improve with antidepressants. This study, included in the 2012 review by the same authors, was conducted in the UK where antidepressants are considered the medical frontline treatment for primary care cases of depression. The doctors did not attempt to use pharmacology to explain why the antidepressants were ineffective in non-responsive patients – instead they reflected a psychosocial perspective, which implied that medication only works when the correct psychosocial circumstances are present. McPherson and Armstrong (2009, p. 1141) observed that the absence of effective medical treatment seemed to result in the label of depression becoming less strongly attached to such persons. Instead the non-responsive patients are constructed as having socially deviant features such as disagreeable personalities and controlling tendencies. One GP who had been practising for over 18 years said:

> [T]hey call them heart sink patients because they are always complaining of something, and never getting any better and never you know improving and never happy of anything you do for them...patients which are not typically depressed but just a nuisance.

This quote, along with others in the study, reflects a loss of empathy for non-responsive patients and has fundamental implications for the GP-patient therapeutic relationship (McPherson & Armstrong, 2009).

McPherson and Armstrong's review (2012) did not include studies on depression EMs of GPs where the study focus was considered too narrow, such as studies on postnatal depression or depression among people with other health conditions such as HIV. However, I include here a study on depression discourses of Australian GPs working with high caseloads of homosexual men (Körner et al., 2011) because it highlights the strong link between GP perspective and treatment. In a qualitative interview study that was part of a multi-stage mixed method study of both homosexual patients and their GPs, Körner et al. identified three distinct discourses of depression among the 16 GPs interviewed. The first was depression as constructed in mainstream psychiatry using the DSM classification, focusing on decontextualised symptoms such as low mood and lethargy. This biomedical discourse or EM was explicitly refuted and rejected. For example, one GP countered the DSM-IV construction of depression repeatedly: “I don’t subscribe to a formula. I don’t look at the DSM-IV. I’m familiar with it in a loose way, but I tend to just base it on history” (p. 1056).

The second discourse involved the centrality of the patient’s world and experiences in diagnosing and managing depression. As one GP puts it, “You are an absolute idiot if you are a doctor and if you don’t think about what your patients are thinking” (Körner et al., 2011, p. 1056). This second discourse of depression focuses on external actions and events, differentiating it from the first discourse that focuses on the internal world of emotions or mood. The third discourse constructs depression as complexly linked to the larger socioeconomic forces that the patients have limited or no control over. For example, gay men with HIV develop depression because of prolonged period of unemployment. This goes against the perspective that the men become unable to work because of depression (Galasiński, 2008) and it also replaces pharmaceutical and psychotherapeutic treatments with a more effective management strategy centred around employment. Körner et al. concluded that there is a synergy between the GPs’ depression discourses and their gay patients’ experiences of depression that differs from mainstream medical perspectives. This synergy allows the GPs to be decidedly successful in helping their patients cope with mental health issues.

Comparing Explanatory Models

Most studies on EMs of patients and doctors tend to focus on just one group and not the other – very few look at the intersection between patient and doctor perspectives (Haidet et al., 2008). Ogden et al. (1999) surveyed 90 GPs and 681 of their patients on what they believed to be symptoms, causes, and treatments for depression. They reported that both groups were similar in their ratings of mood symptoms, psychological causes, and non-medical treatments. However, the doctors rated the somatic symptoms (e.g., lack of appetite, decreased libido), medical causes, and medical treatments higher than the patients. Essentially the doctors favoured a more medical perspective compared to the patients. A later study by Wittink, Barg, and Gallo (2006) aimed to understand concordance and discordance between older patients (aged 65 and above) and their physicians with respect to depression. However, the study had limitations because the researchers only asked the physicians two fixed-answer questions – one on their patient’s level of depression (none at all, mild, moderate, severe) and the other on how well they knew their patient (very well, somewhat, or not at all). In contrast, the patients were assessed using several scales and they were also interviewed in-person by trained professional interviewers to examine their perceptions of encounters with their doctors (Wittink et al.). Naturally, most of the data only reflected the patients' perspectives, with no in-depth discussion about their doctors' perspectives. Nonetheless the fact that 21 out of 48 participants who self-identified as depressed were rated as not depressed by their doctors on their first visit is further evidence that there is a difference in how patients and GPs perceive depression. Clearly there is a need to consider both perspectives and to examine the impact of differing perspectives, which my thesis sets out to do.

Studies involving participants with diabetes (Hampson, Glasgow, Toobert, & Matthews, 1990), heart failure (Horowitz, Rein, & Leventhal, 2004) and hypertension (Meyer, Leventhal, & Gutmann, 1985) point to the importance of patients and physicians having a shared definition of health problems for optimum management. Increasingly, researchers are suggesting that discordance between patient and doctor EMs hinders collaborative management of chronic illness and results in poor treatment outcomes (Schouten & Meeuwesen, 2006; Von Korff, Gruman, Schaefer, Curry, & Wagner, 1997). However, the degree and nature of discordance between patient and doctor EMs have not been established through large-scale studies of EMs (Haidet et al., 2008). This gap is due to the complex nature of EMs (particularly true in depression) and the lack of suitable tools to systematically characterise and measure differences between EMs of patients and their doctors. Earlier instruments were either too narrowly focused; for example, the Disease Concept Scale (Linden, Nather, & Wilms, 1988) reflected only the patient's perspective, or they took too long. For instance, the Explanatory Model Interview Catalogue (EMIC) takes two hours on average (Jadhav, Weiss, & Littlewood, 2001). As noted in Chapter 3, interviews are time-consuming. Lloyd et al.'s Short Explanatory Model Interview (1998), as the name suggests, is shorter and can be used in a semi-structured manner to elicit causal and other health beliefs. However, Bhui and Bhugra (2002) criticised the SEMI for not having an agreed system of managing and analysing data (unlike the EMIC), which could result in the loss of large amounts of qualitative information. Besides taking too much time and resources, in-depth interviews present conceptual challenges when identifying the parameters on which patient/doctor pairs are similar or different (Haidet et al.). In other words, patients and their physicians may hold differing views on a wide range of topics but it is difficult to decide which topic is relatively more important and needs to have patient-doctor concordance. Haidet et al. (2008) set out to address this problem by systematically identifying which EM dimensions would be important for a patient and doctor to review and recognise each other's perspectives, and then devising a method to quantify concordance. Their work resulted in the development of the CONNECT instrument.

CONNECT instrument. Haidet and colleagues (2008) first conducted a literature review on EMs of illness and 16 qualitative interviews with primary care patients in Houston, Texas. The review highlighted researchers such as Kleinman and Leventhal et al., whose work has been described earlier in this chapter, and the topics that they proposed based on general observations included cause, treatment, severity, meaning of illness, identity, timeline, control/cure, and consequences. Other articles dealing with specific illnesses also identified areas for patient-doctor agreement in addition to those listed above, such as symptoms, coping resources and locus of control. The topics from the literature review were used to generate questions for the patient interviews, which also used Kleinman's proposed method for mini-ethnographies in clinical settings (1980). The patients interviewed came from African American, Hispanic and European ethnic backgrounds, and suffered from a range of enduring health problems such as progressive blindness and infertility.

From the review and interviews, Haidet et al. (2008) identified eight dimensions in which patient-doctor concordance might influence treatment in unpredictable ways if not made explicit. This was then followed by item development for each dimension – the items were based on actual statements from the qualitative interviews where possible and they used a six-point Likert response scale ranging from 1 (*strongly disagree*) to 6 (*strongly agree*). The items were reviewed to eliminate repetitive or unclear statements, and then pilot-tested with 10 patients who answered the items with respect to the last health issue that they had consulted a doctor for. The draft instrument was then field-tested, refined and validated in two separate studies involving 575 patients and 31 doctors from 14 primary care clinics. The wording of the item statements for the physicians paralleled that of the patients; for example, "I am sick because my body is having problems" in the patient's version of the CONNECT instrument was worded "The patient is sick because their body is having problems" in the doctor's version of the instrument. The authors named the final product, which had six dimensions (three of the original eight dimensions were condensed into one after the field testing), the CONNECT instrument because it is supposed to measure how much 'connection' exists between patients and their doctors with respect to the most salient aspects of illness EMs (Haidet et al.). The dimensions of biomedical cause, patient fault, patient control, effectiveness of natural treatments, meaning, and preference for partnership (Haidet et al.) consisted of three to four items each, so the total number of items was 19. The internal consistency estimates, or Cronbach alphas, for the dimensions ranged from .68 to .81 for the patient's CONNECT instrument, while the doctor's CONNECT instrument had alphas between .65 to .85. Haidet et al. noted that these values were approaching or exceeding the recommended value of .80 for group comparisons, citing Nunnally and Bernstein.

Results from the second of the two studies mentioned earlier showed statistically significant differences in all six EM dimensions of the CONNECT instrument (Haidet et al., 2008). On average, the degree to which patients perceive a biological cause for their illness is less than their doctors. Also compared to doctors, the patients also feel greater fault for their illness, have greater sense of control over their illness, believe in greater effectiveness of natural treatments, derive greater meaning from their illness, and desire greater preference for partnership, compared to their doctors. The fact that these findings were consistent with other qualitative research reporting differences in patient and doctor perspectives (e.g., Kleinman, 1980; Schouten & Meeuwesen, 2006; Von Korff et al., 1997) is further evidence of the validity and usefulness of the CONNECT instrument. This is particularly in primary care research where GPs face substantial time constraints.

The instrument has since been used in a study by Street and Haidet (2011) that involved a convenience sample of over 200 patients and 29 GPs from 10 primary care clinics in Texas. Patients and their doctors individually completed the CONNECT instrument after their consultation. The doctors also completed the measure again on how they thought the patient would respond. Helman (1985) had highlighted the need to examine the doctor's belief about the EMs adopted by their patient because it may expose preconceptions or false knowledge of the patient and their perceptions. These suppositions are as important as the patient's socio-cultural background or diagnosis, in that they "will influence what is said in the consultation, how it is said, what is heard and what is heard and interpreted" (Helman, p. 924). I term this the GP-perceived patient EM, that is, the EM that the doctor thinks their patient adopts, and it is also a feature of my study (for operational details, see Chapter 7). Street and Haidet's cross-sectional observational study set out to establish doctors' awareness of their patients' health beliefs by comparing between the patient's EM and their doctor's GP-perceived patient EM for the six CONNECT dimensions. Secondly, they analysed the similarity between the GP EM and the GP-perceived patient EM to see whether doctors tended to think that their patients think like they do. Thirdly, they examined communication, relationship, and demographic factors to determine how they correlate with physician understanding of patient EMs.

Street and Haidet (2011) found significant differences between patient EMs and GP-perceived patient EMs in the CONNECT domains. With the exception of the biomedical cause dimension, where doctors overestimated the extent to which patients' believed their illness was due to biological reasons, doctors underestimated patients' beliefs in all the other dimensions (i.e., about being at fault, having control, finding meaning in the disease, expecting natural treatments to be effective, and seeking collaboration with the doctor). Comparing between doctor EM and GP-perceived patient EM, there were significant differences in the biomedical cause and control over condition dimensions. Doctors' perceptions were in the opposite direction of the patients' beliefs. For example, compared to their own EM, doctors thought that patients subscribed to a more biomedical EM for their illness and that patients felt less in control over their health condition. In reality, patients were less convinced of a biomedical EM and they believed they had more control than what their doctors assumed (Street & Haidet). Significant predictors of the concordance between patient EM and GP-perceived patient EM for the dimensions of meaning, control, natural remedies, and partnership with physician, centred around active patient participation. The more patients asked questions, expressed their concerns, and stated their preferences and views, the greater the concordance. Clearly, communication behaviour plays a key role, which is why communication is another key variable in my thesis (more in the next literature review chapter on communication).

My study is partly based on Helman's (1985) work and I use a modified version of the CONNECT instrument to examine the EMs of depressed individuals and their doctors as well as GP-perceived patient EM. In this chapter, I have explained Kleinman's EMs and the CONNECT instrument as a framework to assess patient and GP depression EMs, and the degree to which these perspectives are similar or different. The next chapter will review and discuss the concept of self-stigma, particularly how it can impact GP-patient communication and treatment outcomes for depression.

Chapter 4: Stigma

In this chapter, I define stigma and discuss its relevance to depression. Researchers have identified that stigma is an important barrier to the treatment of depression (Halter, 2004; Sirey, Bruce, Alexopoulos, Perlick, Raue, et al., 2001). I elaborate on the different manifestations of stigma and explain this thesis' focus on self-stigma. A review of the current literature on self-stigma in depression follows and demonstrates the gap in research examining the link between depression explanatory models (EMs) and self-stigma. The chapter will conclude with the hypothesis that a biomedical EM of depression aetiology correlates with lower self-stigma.

Defining Mental Illness Stigma

The meaning of stigma is derived from a similar sounding verb in two different languages: stigmatizare, to brand or tattoo in Medieval Latin, and stigmatizein, to blemish in Greek. In Goffman's seminal work (1963/1986), he defined stigma as a "deeply discrediting" attribute (p. 3) and "undesired differentness" (p. 5) that sets an individual apart and causes others to perceive that individual as flawed. As a result, such individuals feel ashamed for not meeting social standards and have to manage impressions of themselves to protect their identities. According to Goffman, the main strategy used is through concealing of one's flaws due to the fear of being shunned. This desire for suppression is one reason why stigma prevents people with mental illness from seeking help, which can lead to symptoms becoming more severe. The US Substance Abuse and Mental Health Services Administration (SAMHSA) estimated that only two-thirds (59.6%) of people with serious mental illnesses reported getting treatment in 2011 (SAMHSA, 2012). In Australia mental health service utilisation rates have been reported to be even lower. Using data from the Australian national mental health survey, Andrews, Issakidis, and Carter (2001) found that only one-third (32%) of individuals who met the criteria for a mental disorder had sought treatment for their condition.

Corrigan, Druss, and Perlick (2014) said understanding stigma could help reduce its negative impact on help seeking and sustained treatment engagement. Social science research in stigma increased in the 1980s and 1990s, especially in social psychology, where researchers demonstrated how people conceptualise cognitive groupings and impose stereotypical beliefs on these groups (Link & Phelan, 2001). However, the concept of stigma was criticised for being ill-defined and focused on individual experiences, so Link and Phelan set out to conceptualise stigma more clearly. They posited that stigma comprises of (a) differentiating and labelling of an out-group; (b) cultural beliefs associate the labelled differences with negative attributes; (c) the differences reinforce the disconnection between "us" and "them"; and (d) the labelling and separation result in loss of standing and discrimination.

Around the same time as Link and Phelan's (2001) work, Corrigan (2004) elucidated four social-cognitive processes that contribute to mental illness stigma. They are cues, stereotypes, prejudice, and discrimination. First, Corrigan explains how the public usually uses four types of cues to infer mental illness. They are psychiatric symptoms (e.g., psychotic or bizarre behaviour), lack of social skills, poor personal appearance, and labels. While the first three types of cues can be concealed successfully to avoid stigma, studies show that labels are far more pernicious in causing stigmatising responses. Labels may be obtained from others (e.g., a psychiatrist informing someone about their patient being mentally ill) or from association (e.g., someone coming out of a psychologist clinic may be assumed to have a mental illness).

Cues lead to the second stigma process of stereotyping (Corrigan, 2004). Stereotypes are collective beliefs held about groups. Hamilton and Sherman (1994) explained that stereotyping is an efficient way of processing information because people can rapidly form impressions and assumptions of those from the stereotyped group. People with mental illness are frequently stereotyped as violent/dangerous, incompetent, and weak individuals whose flawed character is to be blamed for their condition. A negative stereotype may be known but not endorsed.

When someone endorses a negative stereotype and consequently experiences adverse emotional responses such as fear of people with mental illness, this is the third stigma process of prejudice. Prejudice as a cognitive and affective reaction leads to the fourth stigma process of discrimination, that is, negative behaviour against the out-group. An example would be landlords refusing to rent to people with mental illness or employers rejecting job applications from people with mental illness. Many studies, whether from the Western industrialised nations, or from low- and middle-income countries confirm that mental conditions are highly stigmatising (Ghanean, Nojomi, & Jacobsson, 2011). The effects of stigma include loss of status and self-esteem, and eroded social networks. This leads to poor outcomes such as unemployment, delays in seeking treatment, prolonged course of illness and avoidable hospitalisations (Link, Struening, Neese-Todd, Asmussen, & Phelan, 2001; Sirey, Bruce, Alexopoulos, Perlick, Friedman, et al., 2001).

Self-stigma and Its Effects

Besides elucidating the four processes of mental illness stigma, Corrigan (2004) differentiated between public stigma and self-stigma. Public stigma is the typical societal response by members of the public who are prejudiced against people with mental illness, and has been referred to as cultural stigma by Quinn and Chaudoir (2009). In contrast, self-stigma is how people with mental illness respond to being in the stigmatised out-group, the lowering of their self-esteem and sense of self-worth because they do not see themselves as full members of society (Ritsher, Otilingam, & Grajales, 2003). Livingston and Boyd's (2010) systematic review of 45 studies found robust inverse correlations between self-stigma and psychosocial variables of self-esteem, hope, self-efficacy or empowerment, social support, and perceived quality of life. Livingston and Boyd used the term *internalised stigma*, which Ritsher et al. defined as the "devaluation, shame, secrecy and withdrawal triggered by applying negative stereotypes to oneself" (p. 32). Ritsher et al. had chosen the term *internalised* partly because it aligned with research on internalised racism and internalised homophobia, which similarly examines the adverse psychological impacts of public perceptions. People with mental illness who experience internalised stigma marginalise themselves, making the external discrimination from others even more challenging to overcome.

Corrigan (2004) and Ritsher et al. (2003) are referring to the same construct but I prefer Corrigan's term of self-stigma because it is more succinct. Also, Corrigan's explanation of the different stereotype-prejudice-discrimination pathways of public stigma and self-stigma provides a clear pathway (see Figure 1 that is based on a diagram by Corrigan, p. 617). Corrigan and Watson (2002b) have noted that public stigma and self-stigma are likely to interact with and amplify each other.

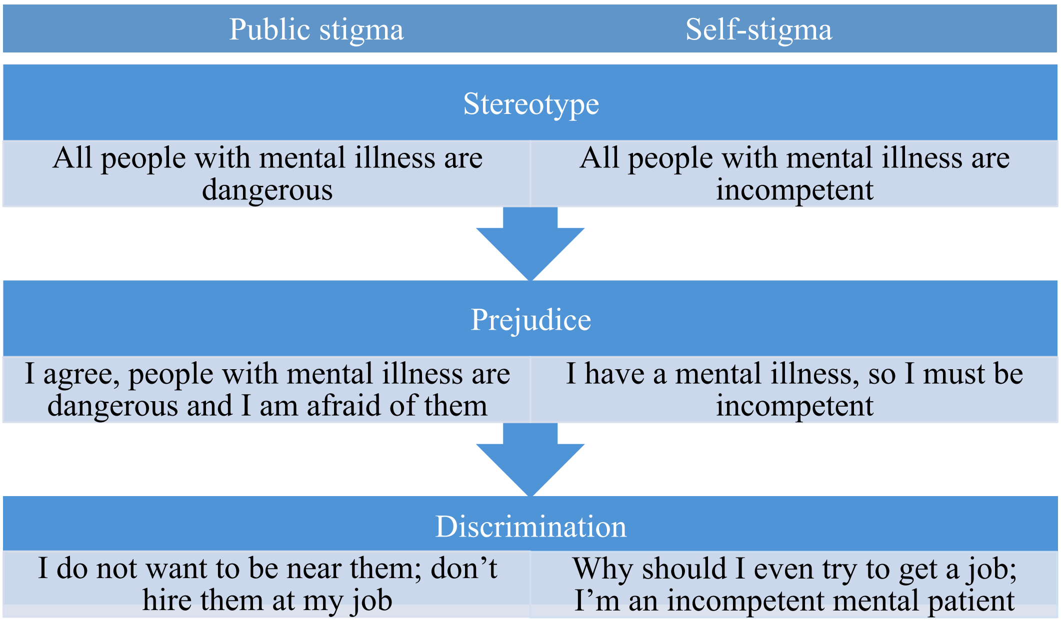


Figure 1. The difference between public stigma and self-stigma.

Adapted from "How Stigma Interferes with Mental Health Care" by P. Corrigan, 2004, *American Psychologist*, *59*(7), p. 617. Copyright 2004 by the American Psychological Association.

Earnshaw and Chaudoir (2009) provided further insight into how individuals experienced self-stigma when they differentiated between the mechanisms of anticipated, enacted, and internalised self-stigma. Anticipated self-stigma is where individuals expect to experience discrimination or social rejection. Enacted self-stigma is where individuals have experienced discrimination and rejection. Internalised stigma is where individuals endorse negative feelings (e.g., shame and self-loathing) about themselves. Anticipated and enacted stigma represents stigmas that are perceived to be directed at the self from others, while internalised stigma is stigma directed at the self from the self (Chaudoir, Earnshaw, & Andel, 2013). Reviewing HIV stigma mechanism measures, Earnshaw and Chaudoir gave examples of how these different aspects of self-stigma lead to negative psychological, behavioural, and health outcomes in people living with HIV/Aids. Their observations can be applied to individuals with depression, for example someone with a high degree of anticipated self-stigma is less likely to disclose that they have depression, which in turn affects their willingness to seek any help. An individual who has experienced enacted self-stigma may be subject to psychological distress, and a worsening of their depressive symptoms as a result. Those who have internalised self-stigma may lose their sense of agency and feel helpless about being able to improve their depression, which leads to poor engagement in any therapy or management strategies.

It is important to note that Earnshaw and Chaudoir's (2009) internalised stigma is more specific than Ritsher et al.'s (2003) construct by the same name, which was explained at the beginning of this section. Ritsher et al.'s internalised stigma would encompass all three of Earnshaw and Chaudoir's self-stigma mechanisms. In stigma research, it is common to come across similar terms that have different meanings, which adds to the challenge of understanding empirical evidence on the nature of stigma and its impact. Nevertheless, there is sufficient evidence that self-stigmatisation can set off a vicious circle when it induces a change in an individual's identity and results in reduced self-confidence (Corrigan & Watson, 2002a). This then leads to a tendency to perceive others as being discriminatory even if they are not (Latalova, Kamaradova, & Prasko, 2014) and leads on to further erosion of self-esteem. In other words, even if public stigma is diminished, people with self-stigma will still be suffering. Such evidence points to the need to address self-stigma, which is why I focused on self-stigma in depression. Another study conducted in the US found that self-stigma in adults over 60 years and living with depression is associated with a negative attitude towards seeking help, more so among African American participants compared to their white counterparts (Conner et al., 2010). Race and age are among the demographic information collected from participants in my thesis. Participant gender was included because research has shown that males seem to have higher levels of self-stigma than females (Latalova et al.). Endorsing of dominant masculine norms (e.g., "boys don't cry", p. 1399) leads to self-stigmatisation in men who feel that they should be able to manage their depression without professional intervention.

Another perspective about mental illness self-stigma comes from Tucker et al. (2013) who identified two subgroups of self-stigma – the first involves feeling ashamed and embarrassed over having a mental illness, and the second involves further loss of self-esteem when a person labels themself as someone who needs psychological treatment. Tucker and colleagues collected data from 217 college students suffering clinical levels of mental distress and 324 community members with self-reported history of mental illness. Clear evidence from confirmatory factor analyses showed that self-stigma of mental illness and self-stigma of seeking psychological help are empirically distinct constructs in the sampled populations. Multiple regression analyses in both samples indicated that the two self-stigmas uniquely predict differences in theoretically related stigma components such as shame, self-blame and social incompetence. Help-seeking self-stigma was found to be the only significant predictor of self-blame, and not mental illness self-stigma. The authors discussed this finding in light of research investigating perceived controllability. Having a mental illness may be seen as less controllable than actively seeking mental health treatment (Vogel & Wade, 2009). Accordingly, Corrigan et al. (2000) have established that mental illnesses that appear to be more controllable (e.g., substance abuse) are more stigmatising than those that seem to be less controllable.

Tucker et al. (2013) also found that help-seeking self-stigma accounted for a larger amount of variance in help-seeking attitudes and intentions than mental illness self-stigma. These findings were similar to the findings by Schomerus et al. (2009), who investigated the correlation between intentions to seek help for depression and treatment stigma. These researchers developed two scales, one on the anticipated discrimination by others when seeing a psychiatrist, and the other on the desire for social distance from someone seeing a psychiatrist. They then conducted a representative population survey in Germany (n = 2,303) where adult participants aged 18 years and older were asked to imagine they had seen a GP for symptoms of depression as described in a vignette. Respondents were asked to rate how likely were they to follow their GP's recommendation to consult a psychiatrist and they also completed the two scales of anticipated discrimination and desire for social distance. Finally, the respondents provided demographic information, history of prior treatment from a psychiatrist/psychologist, and completed the German version of the patient health questionnaire to determine if they had current depressive symptoms. Schomerus and colleagues found that in both non-depressed and depressed participants the personal desire for social distance significantly hampered help-seeking intentions, whereas anticipated discrimination from others had no effect. Other factors that increased the likelihood of help seeking were female gender and prior contact with psychiatric treatment or psychotherapy.

Consistent with findings from Germany, Jorm and Wright's (2008) study of young Australians and their parents found that those who have received psychological treatment scored higher on 'stigma perceived from others' than those who have not sought treatment. It appears to be a case of stigma begetting stigma; help-seeking self-stigma being associated with anticipated or enacted self-stigma – as per Earnshaw and Chaudoir's (2009) terms mentioned previously. This study's findings is similar to a depression vignette study by Ben-Porath (2002), which showed student participants rating depressed individuals who have received psychotherapy as less emotionally stable and less confident than those who were not treated for their depression. Ben-Porath's study involved undergraduates enrolled in an introductory psychology course and did not differentiate between participants who may have experienced mental illness. Therefore its findings can be said to reflect public help-seeking stigma.

It is worth noting how the public help-seeking stigma could potentially negate real benefits from seeking psychotherapy. For example, a depressed individual who undergoes effective psychotherapy may be more emotionally stable and more confident compared to a depressed individual whose symptoms worsen because he or she refuses to seek help. However, the one who sought help could still be perceived as the less emotionally stable and less confident simply due to the insidious effects of help-seeking stigma. Being in a society that endorses help-seeking stigma, an individual with depression may accept and internalise the same stigmatising attitudes and beliefs that they are exposed to (Link and Phelan, 2001), resulting in persistent self-stigma over seeking psychological help. The self-stigma study in my thesis will examine whether participants with depression feel embarrassed over seeking professional treatment for depression (more details in the Methods section on help-seeking inhibition and other self-stigma subscales). The next section will elaborate on the different self-stigma scales that currently exist in literature.

Self-stigma Measurements

Measuring mental illness self-stigma. King et al. (2007) observed that there were fewer stigma scales for people with mental illness compared to scales to measure the general public's stigma towards mental illness. They cited a study by Judge on help-seeking stigma of receiving psychotherapy as being among the first of such scales, followed by a second measure by Link et al. on the shame and withdrawal experienced by people with mental illness. King et al. designed a standardised measure of mental illness stigma from the experience and perspective of mental health service users in the UK; called the Stigma Scale for Mental Illness. A few years earlier in the US, Ritsher et al. (2003) had developed the Internalized Stigma of Mental Illness (ISMI) scale using input from people living with mental illness. The ISMI was designed to be as comprehensive as possible (Ghanean et al., 2011), with subscales measuring Alienation, Stereotype Endorsement, Perceive Discrimination, Social Withdrawal, and Stigma Resistance (Ritsher et al.). The 29-item questionnaire has been widely used and is available in multiple versions to cater for different languages, ethnicity, and medical conditions including depression (Boyd, Adler, Otilingam, & Peters, 2014). In a multinational review of the 47 available ISMI versions, Boyd et al. found that the ISMI was reliable and valid across an extensive array of languages, cultures, and writing systems. The most frequently reported findings of studies using the ISMI are that self-stigma is associated with higher levels of depression, lowered self-esteem, and more serious symptoms.

A similar scale to the ISMI is the Self-Stigma of Mental Illness Scale (SSMIS), which was developed by Corrigan, Watson, and Barr (2006) to assess levels of internalised and perceived stigma of individuals with a mental illness diagnosis. However, in a review of self-stigma instruments by Stevelink, Wu, Voorend, and Van Brakel (2012), the SSMIS was given indeterminate ratings for construct validity and reliability. The ISMI had relatively better ratings, even though none of the 21 self-stigma scales reviewed by Stevelink et al. fulfilled all seven criteria that the authors were purported to be using. The criteria were part of a quality framework by Terwee et al. (2007) on what constitutes good psychometric properties, and an international panel of experts had reached consensus on the precise terminology, classification, and definitions of these properties (Mokkink et al., 2010). The properties were content validity, internal consistency, construct validity, criterion validity, reproducibility, responsiveness, floor and ceiling effects, and interpretability. The ISMI rated positively for three out of seven criteria (only one other scale scored three out of seven in Stevelink et al.'s review) and has been used in at least one study on self-stigma in depression (Brown et al., 2010).

However, I felt it was necessary to review self-stigma instruments for depression if they were available, especially given this thesis' focus on depression. Also, a longitudinal study by Crisp, Gelder, Goddard, and Meltzer (2005) involving a representative population sample in Great Britain found that the nature of depression stigma is different from stigma related to other types of mental illness. In 1998, more participants felt it was hard to talk to someone with depression (62%) compared to someone suffering panic attacks (33%), schizophrenia (58%), dementia (60%), eating disorder (38%), and alcoholism (59%). Five years later, the percentage was significantly lower ($p < .005$), having fallen to 56% but this was still higher compared to the other conditions listed above, except dementia, where the percentage was the same. I consider Crisp et al.'s findings on depression stigma to be an example of public stigma because the study did not target participants diagnosed with mental disorders. Since the study shows that public stigma about depression is different from public stigma of other mental illnesses, it is reasonable to expect that self-stigma of depression should also be studied separately from self-stigma of mental illness as a whole. This is why I argue that scales for depression self-stigma are necessary.

Measuring depression self-stigma. Two scales have been specifically developed for self-stigma in people with depression – the Depression Self-Stigma Scale (DSSS) by Kanter, Rusch, and Brondino (2008) and the Self-Stigma of Depression Scale (SSDS) by Barney, Griffiths, Christensen, and Jorm (2010). The DSSS contains 32 items in five subscales: general self-stigma (negative stigmatising attitudes of the respondent and perceived stigma from others that have been internalised), secrecy (tendency to hide depression from others), public stigma (respondent's negative attitudes about other depressed individuals), treatment stigma (negative attitudes about being treated for depression), and stigmatising experiences (past incidents of feeling stigmatised because of depression). I propose that the public stigma subscale, made of items such as "Other people with depression are morally weak" is problematic in a scale that is supposed to focus on self-stigma. It would have been preferable if the wording had been changed to reflect an internalised perspective (e.g., "Having depression means I am morally weak"). In addition, I concur with Barney et al.'s assessment that the DSSS is more focused on perceived stigma from others than internalised stigma on a personal level. For example, 25 of the 32 statements in the DSSS refer to stigma in relation to others, or people, or someone (Kanter et al.). Schomerus et al.'s (2009) study, as mentioned earlier, has shown that personal self-stigma has more impact on a person with depression than anticipated stigma from others. Stevelink et al. (2012) also rated the content validity of the DSSS negatively because depression patients were not involved during item generation or selection.

In contrast to the DSSS, the scale content of Barney et al.'s SSDS (2010) was largely developed from views expressed by individuals with direct or indirect experience of depression. The SSDS's 16 Likert items were drawn from focus group discussions followed by testing and refinement over three surveys. The first survey involved 408 undergraduate psychology students and the second involved 330 members of the online network of Australia's national depression initiative *beyondblue* (86% reported they had personally experienced depression, the others were carers or primary support providers of people with depression). The final scale validation survey involved 1,312 members of the public who were asked how they would perceive themselves if they had depression. Items were phrased using wording expressed by the focus group participants (e.g., I would feel ashamed") and responses measured on a five-point scale ranging from 1 (*strongly agree*) to 5 (*strongly disagree*). Barney et al. provided a vignette describing a male diagnosed with DSM-IV major depression to ensure a common understanding of what depression entailed. The items showed consistent results in all three surveys, suggesting that the scale has good construct validity. Factor analysis identified four factors of shame, self-blame, help-seeking inhibition, and social inadequacy. In addition, multi-group modelling and comparison of regression weights showed that the SSDS performed dependably in participants with all levels of depressive symptoms. Another benefit of the SSDS is that former focuses only on self-stigma of depression and not perceived stigma from others, unlike the DSSS.

However, Stevelink et al. (2012) gave the SSDS a negative rating for reliability because intraclass correlation coefficients (ranging from .49 to .63) were below the review's .70 quality criterion. While the SSDS's limited reliability is admittedly a constraint, I chose to use it for the reasons detailed in the previous paragraph and also because it was developed in an Australian context (Barney et al., 2010). To counter the limitations of the SSDS, I combined items from it with the ISMI (modified to be relevant to depression). This allowed me to take advantage of the strengths from both scales for the self-stigma portion of my thesis (more details in Chapter 7).

The availability of psychometrically sound self-stigma scales enables researchers to determine the nature and degree of depression self-stigma (an important component of my thesis), to appropriately design interventions to target aspects of self-stigma, and to evaluate whether such programmes are effective (Barney et al., 2010). Rüsch, Angermeyer, and Corrigan (2005) suggested reducing the mental illness self-stigma by normalising symptoms and communicating about the neurobiological basis of mental disorders. Similarly, Corrigan (2016) recommends normalising mental illness as one of the two ways to resolve mental illness stigma. He gave the example of how *beyondblue* released public service announcements and materials that showed depression as being normal. At the same time, the campaign explained what constitutes depression interventions, portraying these as similar to other medical treatments. In other words, depression is simply seen as just another medical condition.

However, Schnittker (2008) argued that the rise of genetic frameworks for mental illness fosters an essentialist perspective of mental conditions as stable and unyielding, even with treatment. Consequently, there is less tolerance. Similarly, Thachuk (2011) criticised the use of biomedical models to challenge stigma because likening mental illnesses to physical illnesses reinforces the idea that those with mental illness are fundamentally different. It also entrenches misperceptions that people with mental conditions are inherently more violent and promotes over-reliance on diagnostic labelling and pharmaceutical treatments. This connection between explanatory models and stigma was previously mentioned in Chapter 3. Researchers like Dietrich et al. (2004) have called for a reconsideration of the assumptions behind anti-stigma initiatives that frame of mental illness as biomedical conditions. After conducting representative interview surveys in Germany, Russia, and Mongolia, the authors concluded that publicising biomedical causal models might not reduce the desire for social distance towards people with mental illness.

In the case of depression, the biomedical causation model has not been shown to conclusively reduce certain aspects of self-stigma such as a sense of helplessness (Cunningham, Sirey, & Bruce, 2007). Using discourse analysis, Lafrance (2007) showed that women constructed their depression as a medical condition to try and validate their suffering. However, Lafrance noted an uneasy fit between the women's subjective perception of depression and the objective discipline of biomedicine, which causes delegitimisation of the women's experience. My thesis aims to further investigate the link between depression explanatory models and self-stigma.

Depression Explanatory Model and Self-Stigma

Numerous initiatives to combat mental illness stigma, for example campaigns launched by the US National Alliance for Mental Illness, intentionally and clearly depict mental disorders as medical conditions (Angermeyer, Holzinger, Carta, & Schomerus, 2011). Biogenetic causal models of mental illness are assumed to be destigmatising for two reasons said Angermeyer et al. Firstly, assigning biological or genetic factors as the cause of mental illness should shift blame away from the affected person since the biogenetic factors are beyond the person's control. Secondly, if individuals are held less responsible for their condition, they should logically undergo less social rejection. Longitudinal studies that track public attitudes towards mental illness and people with mental illness do show an overall increase in biomedical attributions in the past 20 years in the US (Schnittker, 2008), Australia (Jorm, Christensen, & Griffiths, 2005), and Germany (Angermeyer, Holzinger, & Matschinger, 2009). However, Angermeyer et al.'s (2011) systematic review of 33 population studies found that biogenetic causal models for mental illness were not linked to more tolerance. Also, they found that the notion of personal responsibility for mental illness was not associated with rejection in most of the studies reviewed. Instead, it was stereotypes of unpredictability and dangerousness that influenced the public's perception of people with mental illness, while the issue of responsibility was less salient. In light of this, Angermeyer et al. concluded that promoting biomedical causal models is an unsuitable strategy for combating rejection and stigma towards people with mental illness.

The findings from Angermeyer et al. (2011) were corroborated by a meta-analysis of 28 experimental studies on how biogenetic explanations affect stigma (Kvaale, Haslam, & Gottdiener, 2013). Kvaale and colleagues analysed whether biomedical explanations affect blame (perceptions of responsibility and feelings of anger), perceived dangerousness (perceptions of unpredictability and feelings of fear), desire for social distance (unwillingness to enter into a social relationship), and prognostic pessimism (perceptions of low treatability and long illness). Kvaale et al. found that biomedical explanations reduce blame but increase pessimism. Biomedical explanations were also found to increase endorsement of the stereotype that people with mental illness are dangerous, although the authors said this could be due to publication bias. As for social distance, biomedical explanations did not seem to have an effect. Kvaale et al. concluded that promoting biomedical explanations to reduce blame could have the unintended effect of causing patients to be pessimistic and more prone to self-fulfilling prophecies that could hinder recovery from mental illness. Deacon & Baird (2009) have similarly noted the link between the chemical imbalance explanation for depression and pessimism that further disempowers patients. In other words, biomedical portrayals of depression may not help to combat mental illness stigma.

Some researchers have noted that the biomedical explanations for depression can be useful in reducing certain types of stigma, including facets of self-stigma. For example, as mentioned in Chapter 3, Schreiber and Hartrick (2002) argued that their interviewees had adopted biologically oriented perspectives of depression in an attempt to combat feelings of shame and fear associated with being depressed. A biomedical view of depression is supposed to dispel notions of bad character and personal responsibility. This philosophy was encapsulated in the public education campaign by the National Association for Research on Schizophrenia and Affective Disorders, which had the slogan “depression is a flaw in chemistry, not character” (Goldman et al., 1999, p. 577). However, Schreiber and Hartrick found that biomedical explanations reinforce anticipated stigma from others.

Other researchers have pointed out that the downside of diminishing one’s responsibility for making psychosocial changes could outweigh the positive aspects (e.g., Cunningham et al., 2007; Gammell & Stoppard, 1999). Gammell and Stoppard’s study involved nine women diagnosed with depression and was published almost two decades ago, making it potentially less relevant today. Cunningham et al.’s study cast doubts on whether emphasising the biomedical basis for depression does decrease self-stigma. Their research among 18 attendees of a mood disorders support group in Ireland concluded that the biomedical model alleviates patients’ sense of responsibility and thus reduced their guilt. However, patient beliefs of illness uncontrollability may undermine their sense of agency, encourage depressive attributional styles, and affect treatment outcomes. The small number of participants, already participating in a general mood disorders support group, limits the generalisability of Cunningham et al.’s findings to individuals with depression. Thus my study aims to gather evidence of any association between depression explanatory models and self-stigma. Specifically I test the hypothesis that patients who more strongly endorse biomedical causation for depression would experience lower self-stigma at Time 1 than patients who less strongly endorse biomedical causation.

In this chapter I have explained self-stigma in relation to depression. By highlighting the role of psychosocial factors, doctors can offer patients a greater sense of control that they can reduce the impact of the illness, as well as mitigate the effects of stigma. Hayward and Bright (1997, p. 352) recommended the sharing of "a holistic conception of mental illness, incorporating both psychosocial and biological models". They further recommend that patients and doctors discuss how to have a healthy lifestyle to prevent relapse and to help lower stress. The key is to empower the patient by encouraging their sense of self-efficacy and personal worth. To accomplish this, communication effectiveness between patient and GP is crucial. The next chapter will examine communication accommodation theory (CAT: Gallois et al., 2005; Giles, 1973) and the role of communication in my thesis.

Chapter 5: Communication Effectiveness

In the previous chapter, I have explained how self-stigma can affect individuals with depression. Now I elaborate on another key aspect affecting depression management in primary care – the communication effectiveness between general practitioners (GPs) and their patients. I define communication as effective when both patients and doctors are satisfied with their interactions and wish to continue their therapeutic relationship. This arises from both sides achieving their goals, which may differ from patient to patient (Deledda, Moretti, Rimondini, & Zimmermann, 2013), and from doctor to doctor (Veldhuijzen et al., 2013). Effective communication also reflects the presence of mutual elements such as rapport, respect, and trust. I begin by giving a brief overview of how health communication has grown in importance over the years, especially in regards to quality of patient care. I review studies that show the link between doctor-patient communication and treatment outcomes, particularly in managing depression. This is followed by a description of communication accommodation theory (CAT: Gallois et al., 2005; Giles, 1973), which looks at interpersonal communication as an intergroup phenomenon. I highlight the lack of research into the association between CAT and depression explanatory models (EMs), which I address by examining the relationship between EMs of depression patients and their GPs and patient-GP communication effectiveness.

Health Communication

Health communication first developed as a field of study in the late 1960s (Thompson, Cusella, & Southwell, 2014) with the work of paediatrician Barbara Korsch and colleagues (e.g., Korsch, Gozzi, & Francis, 1968), who focused on the interaction between healthcare providers and patients. Most researchers have taken an interpersonal approach to health communication, as seen in the early issues of the journal *Health Communication*. In the journal's inaugural issue, Korsch (1989) wrote of patient dissatisfaction due to failures in patient-doctor communication, and doctors who inaccurately perceived that their communication with patients was effective. Furthermore, doctors' priorities were affected by increased medical technology, which worsened the communication problems. Korsch lamented "[a]ll too often, a penicillin injection was substituted for reassurance, and laboratory tests were employed when careful interviewing for medical history might have been preferable" (p. 5). Later, emphasis shifted to the communication in health campaigns, incorporating principles from sociology and public health to examine the impact of the campaigns on their target audience (Thompson et al., 2014). Nevertheless, provider-patient interactions continue to be studied extensively today, especially by health practitioners whose approach is often atheoretical (Kim, Park, Yoo, & Shen, 2010; Thompson & Zorn, 2010).

Early researchers such as Pendleton (1985) and Beisecker (1990) investigated the factors that influence the communication relationship between patients and providers. Pendleton cited studies showing communication failures from both sides of clinical encounters. Patients felt unable to freely express their worries and desires (Fremon, Negrete, Davis, & Korsch, 1971); feared they could not understand their doctor (Fitton & Acheson, 1979); and had health beliefs that were overlooked by their doctors (Tuckett, 1982). The health beliefs referred to in Tuckett's study were arguably explanatory models (refer to Chapter 3), supporting the importance of exploring this further in my thesis. As for doctors themselves, they acknowledged communication challenges in about 25% of their consultations (Pendleton, Brouwer, & Jaspars, 1983). This could be a vast underestimation because doctors tend to overestimate their communication skills (Ha & Longnecker, 2010) and often perceive their communication to be sufficient or even excellent while their patients are dissatisfied (Stewart, 1995).

Beisecker (1990) examined power in doctor-patient relationships that were becoming, and I would argue continue to be, increasingly salient with the growing awareness of patient consumerism and patients' rights. Beisecker's definition of power incorporated the ability of individuals in a social relationship to exert their will despite opposition, first defined by Weber (1947), and the capacity to bring about desirable changes in the behaviour of others while preventing undesirable modification of their own behaviour by others, as defined by Tawney (1931). Doctors have power over their patients because they have high status and because they supply services that patients need (Blau, 1964; Watson, Hewett, Gallois, 2012). Beisecker noted that patients could be empowered during consultations if doctors allowed a transfer of power.

In a study involving rehabilitation medicine patients, Beisecker and Beisecker (1990) showed that contextual variables (such as consultation duration and diagnosis) influence the role patients play more than sociodemographic factors and patient attitudes. For example, patient desire for information and participation in decisions predicted patient information-seeking behaviour only in consultations lasting at least 19 minutes. This indicated that longer interactions were needed for patient attitudes to be expressed as behaviour, which is important considering primary care settings that are often time-scarce. On the other hand, Lemon and Smith's (2014) review found that the length of a consultation does not determine patient satisfaction but rather the extent in which doctors discuss the psychosocial factors affecting their patients.

The topic of power and status differences between patients and their healthcare providers is still extensively researched today (e.g., Baker, Gallois, Drieger, & Santesso, 2011; Baker & Watson, 2015; Watson, Jones, & Hewett, 2016) because it impacts on doctor-patient communication and the strength of their therapeutic alliance. In recent decades, patients' access to medical information on the Internet has empowered patients, thus affecting the dynamic of their interactions with doctors (Mittman & Cain, 2001). Good patient-doctor communication and strong therapeutic alliance are both correlated to positive treatment outcomes (Street, 2003; Street, Makoul, Arora, & Epstein, 2009). In particular, meta-studies (e.g., Horvath, Del Re, Fluckiger, & Symonds, 2011; Sharf, Primavera, & Diener, 2010) have reiterated the importance of the working relationship between patients and their doctors in determining the effectiveness of mental health treatments. In these studies, enduring therapeutic alliances were significantly linked to lowered risk of treatment discontinuation and improvement in post-intervention symptoms.

One study that demonstrated the link between patient-centred communication and beneficial outcomes in the context of primary care, the context of my thesis, is Stewart et al.'s (2000) observational cohort study. The authors audiotaped clinic visits of 39 randomly chosen doctors and 315 of their adult patients who had at least one recurring health issue. The recordings were scored for patient-centred communication and patients were also asked for how patient-centred they perceived their visit to be. The study's primary health outcome was the extent of patient recovery from discomfort and concerns, measured using patients' self-report on visual analogue scales on symptom severity before the GP visit and two months after. The secondary health outcome was patient health status as measured on the Medical Outcomes Study Short Form-36. Through chart review the study also obtained variables for medical care utilisation in the two months following the initial visit. Stewart et al. found a significant correlation between patient-centred communication and patients' perception of finding common ground with their doctors. Furthermore, high total scores on patient-centred communication and high sub-scores on finding common ground were correlated with better patient recovery, improved emotional health after two months, and increased efficiency of care with fewer diagnostic tests and referrals.

Good doctor-patient communication is not only beneficial in primary care settings. Review studies into specialist treatment of medical conditions such as cancer (Arora, 2003), hypertension (Harmon, Lefante, & Krousel-Wood, 2006), and urinary tract infection (Platt & Keating, 2007) also show the importance of effective doctor-patient communication. Effective communication by the health professional can help manage patients' emotions, promote understanding of medical knowledge, and improve recognition of patient requirements, views, and expectations. Patients who highly rate their communication with their doctor are more likely to experience satisfaction with their care, share salient details for a correct diagnosis, act on advice, and comply with prescribed treatment.

However, many studies are often framed in terms of what doctors should do. For example, the measure of patient-centeredness that researchers used in Stewart et al.'s (2000) study were all focused on doctor behaviour. The first dimension was the disease and illness experience (doctors score highly by discussing their patients' symptoms, feelings, the ability to function, and expectations). The second dimension was on understanding the whole person (doctors score highly by asking about one's life cycle, personality, and family). The third was on finding common ground (doctors comprehensibly explaining the problem and treatment plan, answering questions, and making decisions with the patient). The emphasis on doctors being responsible has been the case since the 1980s as Thompson (1984) also noted an apparent bias on the part of researchers, who seem to fault healthcare providers when interaction problems arise, instead of looking at both interactants.

Similarly, despite mentioning that integrated health systems now have a more holistic approach towards patients, Thomas (2006) appeared more focused on helping health professionals get their message across since "[they] now know what to tell people in most instances" (p. vi). However, I would argue that provider-patient communication goes beyond doctors knowing *what* to say to their patients. It also goes beyond them knowing *how* to say what they want to say. Rather, my approach to GP-patient communication is to see it as a dynamic two-way process (e.g., Baker & Watson, 2015; Epstein, 2006; Street, Krupat, Bell, Kravitz, & Haidet, 2003), with perspectives and communication behaviour of both parties being equally important. I agree with Platt and Keating (2007) who said education of patients and doctors, and their concordance about disease reliably leads to improved treatment compliance and reduced rates of recurrence. They argued for improving communication in consultations by encouraging appropriate two-way sharing of medical and patient information. This emphasis on two-way sharing is a crucial aspect of communication accommodation theory, which is discussed later.

In sum, research points to provider-patient communication as a key factor in helping patients to manage their health problems. If done effectively, communication contributes to improved welfare, hence Thompson's (1984) reference to communication as the invisible helping hand in the health and social service professions. At the same time, ineffective communication can lead to wrong diagnoses, issues with treatment adherence, and intercultural misinterpretations (Ley, 1983; Thomas, 2006). Thomas asserted that most problems faced in healthcare are due to communication failures, especially when interactants overlook subtleties and implicit exchanges that have profound implications both for patients (e.g., quality of care) and doctors (e.g., lawsuits). These different perceptions of what constitutes high-quality care can arise from doctors and patients having different EMs – the focus of this thesis.

Provider-patient communication in managing mental illness. Ronald M. Epstein (2006), a medical doctor and professor of family medicine and psychiatry, noted that patients, doctors, and researchers might seek different solutions because they hold different views about communication. For example, communication researchers may focus on the specific mechanisms of communication, but patients may just want to feel understood. Accordingly, patients focus on whether they experience trust, a suitable level of autonomy, care, and doctor competence in their therapeutic relationships. From his personal observations as a patient and other examples in literature, Epstein argued for communication research to not only study clinician behaviours and patient perceptions of care, but also focus on additional aspects of provider-patient communication. One of these aspects involves what patients notice, desire, and need, and how their views differ from those of doctors. Another aspect is the context of the clinical encounter, including factors such as illness type and severity. These aspects can be addressed by eliciting explanatory models (EMs). Hence my thesis is interested in the intersection between depression EMs and GP-patient communication.

As mentioned in Chapter 2, depression and other mental disorders have become increasingly constructed as biomedical diseases (Pescosolido et al., 2010). This influences treatment choices by doctors (Ahn, Proctor, & Flanagan, 2009) and symptom perceptions of patients (Lebowitz, 2014; Lebowitz, Ahn, & Nolen-Hoeksema, 2013). Lebowitz, Ahn, and Oltman (2015) studied how biological conceptualisations influence people’s perceptions of doctors. They asked 606 participants to imagine that they were seeking treatment for a loved one with mental illness, while another 586 participants were asked to imagine they were seeking treatment for themselves. Both sets of participants were recruited through Mechanical Turk (Buhrmester, Kwang, & Gosling, 2011) and given first-person descriptions of two mental-health clinicians. A sample statement of the biologically oriented doctor’s was “I believe that mental disorders are brain diseases and that it is crucial to understand the genetic basis of each patient’s symptoms” (Lebowitz et al., 2015, p. 670). A sample statement for the psychosocial doctor was “I believe that mental illnesses are disorders of thoughts and emotions.” Results from both participant groups showed agreement on how subjects viewed doctors depending on the type of EM they employed. Biologically oriented doctors were seen as more competent and effective when the illness in question was perceived to be biomedical in nature. Other than that, there was no significant difference in perceived competence of doctors who were biologically oriented. In fact, such doctors were deemed less effective when the illness was perceived to be psychosocial in nature. Interestingly, all participants perceived biologically oriented doctors as significantly less warm on average than doctors who were psychosocially oriented. Lebowitz et al.'s findings have important clinical implications because perceptions of warmth and competence are vital elements for the therapeutic alliance between patients and their healthcare provider (Ackerman & Hilsenroth, 2003), especially in mental health management.

Another aspect of the therapeutic alliance is the influence doctors and patients exert upon clinical decisions. This can arguably be seen as a question of power, which was discussed earlier in this chapter. Karasz et al. (2012) examined conversational influences on the decision-making of doctors with respect to depression treatment, in what they considered to be the first study to analyse GP-patient communication about the nature of depression. The study aimed to investigate the low rate of adherence by primary care doctors to introduced care guidelines that emphasised pharmacotherapy. Karasz et al. cited previous studies (e.g., Nutting et al.; Peveler & Kendrick) where both patients and doctors considered medication to be ineffective for depression if it was psychosocial in nature. In Karasz et al.'s study, patients were unwilling to take antidepressants and doctors appeared to base their treatment choices on common sense or clinical experience, using informal rules instead of the published guidelines. Using purposive sampling, 30 transcripts of primary care consultations about depression or distress were selected from datasets collected from the UK, the US, and the Netherlands. The consultations were chosen based on whether the doctors offered medication, psychotherapy, or no formal intervention.

Using qualitative analysis strategies, Karasz et al. found that patients communicated their "conceptual representations of distress" (2012, p. e55) at the start of each clinic visit. Patient narratives used when communicating their depression concepts were either centred on symptoms, on life circumstances, or had a mix of both. Karasz and colleagues showed that the doctors' decisions were strongly associated with the patients' narratives. Doctors almost always suggested medication to patients with symptom narratives, while very rarely doing so for patients with situation narratives. As for patients with mixed narratives, doctors offered medication to about half of them. Furthermore, doctors did not try to persuade patients to accept a biomedical view of depression, nor did they try to persuade them about medication. Instead, doctors were highly responsive to even subtle cues of patient beliefs. Karasz et al. observed that offers of medicine that were ultimately declined by patients had been made in tentative terms, which suggested that doctors anticipated patient preferences and considered these to be legitimate.

Karasz et al.'s (2012) conclusion that patient agenda drives treatment decisions in primary care consultations for depression, and that doctors were attuned to their patients' views and preferences, differed markedly from earlier findings by Schwenk, Evans, Laden, and Lewis (2004). Schwenk et al.'s national survey study involved primary care patients suffering from chronic recurrent depression in the US. The study indicated that communication barriers do exist between patients and their doctors. From an adult sample who completed a structured phone interview, Schwenk et al. found that the majority of patients were satisfied with their medical care and felt sufficiently informed about their treatment (all the participants were taking a single antidepressant that had been prescribed by a GP). However slightly more than half of the 1,001 patients (57.8%) said their GP did not ask about their preferences or willingness to live with certain side effects before prescribing medication. Of the 466 patients who experienced side effects, half of them (55.4%) stopped taking the antidepressant because of side effects. In addition, 75.1% of the 1,001 patients still reported that they had mild or moderate depression, but they did not discuss unresolved symptoms with their GPs. Schwenk et al. argued that the many different barriers to full recovery from depression could be due to insufficient communication between patients and their GPs. The authors noted that patients want more active communication about medication side effects, more collaborative and patient-centred communication and treatment planning, and a mutual dedication to achieve wellness and complete resolution of symptoms.

It is possible that the findings differed because Karasz et al. (2012) and Schwenk et al. (2004) were essentially examining different aspects of doctor-patient communication. Karasz and colleagues looked at the decision-making process of doctors and patients with respect to treatment options; Schwenk et al. looked at how comprehensive doctors are or appear to be when giving information about medication. Nevertheless, results of the studies follow the trend of moving away from the biomedical approach to provider-patient communication towards a more patient-centred approach trend. Researchers have noted this trend since the late 1990s (e.g., Mola, De Bonis, & Giancane, 2008; Sharf & Street, 1997; Stewart, 2001; Watson & Gallois, 1998). Epstein et al. (2005) said the goal of patient-centred communication (PCC) is two-fold: helping healthcare professionals to provide care that is aligned with patient values, requirements, and desires while enabling patients to actively contribute and engage in making decisions about their health and treatment. The authors proposed four communication domains to consider when evaluating PCC in consultations: patient perspective, psychosocial context, shared understanding, and shared power/responsibility. These domains are highly relevant to my thesis because they can be accessed by eliciting patient EMs. Therefore, it is necessary to briefly review recent studies examining how PCC contributes to effective depression management.

Patient-centred communication in depression treatment. One recent study on the role of PCC in managing depression was by Roter et al. (2014). The study used 307 analogue patients (APs) to uncover the effects of doctor gender and communication style on patients' verbal communication about depression management. APs can be either patients or healthy participants who are asked to rate videotaped clinical consultations as if they were the patient in the video. A systematic review of the validity of using APs in doctor-patient communication research supported their use as proxies for clinical patients (Van Vliet et al., 2012). AP perceptions of communication overlap with those of clinical patients, and AP evaluations of communication equal that of clinical patients, while overcoming ceiling effects. In Roter et al.'s study, the APs were participants aged above 21 who were not currently being treated for depression. The video-consultations were computer simulated with actors as doctors whose gender and communication style (high patient-centred (HPC) and low patient-centred LPC) were manipulated as the independent variables. The dependent variables were the APs' verbal responsiveness and care ratings. The APs (56% female; 70% African-American) were randomly assigned to conditions and told to reply verbally to the doctor's depression-related questions. The APs also had to specify a willingness to persist with treatment. The study found that both male and female APs talked significantly more overall and communicated with significantly more psychosocial and emotional content to HPC gender discordant doctors (all $p < .05$). Furthermore, the APs were more willing to continue treatment with gender-discordant HPC doctors ($p < .05$) but there was no effect on the LPC condition. The implication of this study was that high patient-centeredness promotes active patient involvement in depression management particularly in gender discordant pairs of patients and doctors.

Another study involving APs showed that communication style has a greater influence on patients' comfort when disclosing depression, compared to race concordance between patients and doctors (Adams et al., 2015). The study involved 160 African-Caribbean and 160 white British APs, categorised by gender and depression history (half of the sample had previously received depression while the other half had not). The APs participated in simulated consultations with videotaped doctors. The independent variables for the doctors were race (black or white), gender, and communication style (HPC or LPC). The APs rated features of the doctors' communication (e.g., effectiveness of doctors' non-verbal skills) and their comfort in sharing depression symptoms. The study found that race concordance did not affect African-Caribbeans' comfort over depression disclosure. Instead, it was whether the doctor exhibited an HPC communication style that made the APs significantly more positive about their interactions with the doctors, and more comfortable, both overall and when discussing emotions (all $p < .05$). Interestingly, the APs were more certain about considering psychotherapy when a doctor had HPC communication, but were less certain about considering antidepressants. Adams et al. argued that changing the communication style of doctors to be more patient-centred could help improve diagnostic accuracy and reduce treatment disparities.

As reflected in the studies mentioned above, the realisation that communication is central in medical encounters has led to a substantial increase in the amount and quality of healthcare communication research in the last decades (Freimuth, Massett, & Meltzer, 2006; Kim et al., 2010). We now better understand the importance of good patient-provider communication and the communication factors that contribute to a strong therapeutic alliance. Such factors include the attributes of doctors (e.g., how patient-centred their communication style is) and patients (e.g., how proactive they are during consultations), as well as situational characteristics (e.g., diagnosis).

However, as mentioned previously, there has been long-standing criticism that most health communication research, including provider-patient communication, has been atheoretical to date (see Beck et al., 2004; Cameron, De Haes, & Visser, 2009; Cegala, McGee, & McNeilis, 1996; Kim et al., 2010; Thompson & Zorn, 2010). Beck et al.'s review found that 75% of studies did not have theoretical frameworks. Kim et al.'s later review of publications in the journal Health Communication showed that the proportion fell in the intervening years. Nevertheless, the authors still found that about a third (32.3%) of the studies published between 2007 and 2010 were atheoretical. This was not due to a lack of theories because Kim et al. were able to identify over 104 different theories in the first 100 issues of the journal since it started publishing in 1989. Rather, it pointed to a lack of theory application to guide research. This could have been due to studies being undertaken by health practitioners instead of social scientists. Cameron et al. argued that using theories to guide research can result in greater understanding and better application of the findings. The theory that I invoke for my thesis is communication accommodation theory (CAT: Gallois et al., 2005; Giles, 1973). In the next section, I describe CAT and why it is appropriate for studying perceptions of communication effectiveness from the perspective of depression patients and their

GPs.

Communication Accommodation Theory

CAT has its origins as Speech Accommodation Theory (SAT), an interpersonal communication model that Giles and colleagues developed to explain speech-style modifications (Giles, 1973; Giles, Taylor, & Bourhis, 1973; Soliz & Giles, 2014). Before SAT, linguists had studied how speech is affected by context and the role and identities of the interactants. However, SAT's contribution was to theorise that context does not determine modifications in speech, but instead, the modifications reflect the attitude of the speakers towards each other. Giles theorised that people converge, that is, make their speech more like their conversation partner, to show liking, admiration or to gain approval. People diverge, that is, make their speech more distinct from their conversation partner, for the opposite reasons. Maintenance, that is, where speech style is unchanged, is another way to show dislike, although to a lesser degree than divergence. SAT posits that people are motivated to accommodate because others correspondingly attribute the same motives of like or dislike when they encounter convergence, divergence, or maintenance.

With subsequent elaborations and revisions that extended the theory, it expanded from speech to communication accommodation (Gallois & Giles, 2015). CAT proposes that interpersonal communication in many instances occurs at an intergroup rather than interpersonal level. Intergroup refers to interactions between individuals who see the group membership of their speech partner as salient. Individuals have many social memberships such as different professions or cultures, and social identity theory (SIT: Tajfel & Turner, 1986) predicts that group memberships affect social behaviour. Specifically, we favour in-group members and discriminate against out-group members when salient. SIT focused on behaviours, whereas CAT explains how our language to in- and out-group members can be seen as linguistic markers of social identity. For example, the members of two rival football teams who meet at a match will see their social identity of team member as salient and will use language that shows their group membership.

One of the main socio-psychological processes of CAT is causal attribution. This is when a person interprets another's behaviour and makes a judgment about the other, based on the motivations that the person perceives as the reason for the other's behaviour (Simard, Taylor, & Giles, 1976). Causal attribution adds another layer to the accommodative strategies of convergence and divergence. Although convergence is generally perceived positively, it can be influenced by the listener's attributions of the speaker's motive, that is, the speaker's reason for accommodating. Giles and Smith (1979) illustrated this with an experiment involving French and English-speaking

Canadians. When participants were told that individuals from the different group willingly converged to decrease cultural barriers, they evaluated the behaviour more positively than when told that situational pressures forced convergence.

As an intergroup theory of interpersonal communication, CAT uniquely offers a different perspective from other models of language and social interaction, which tend to focus mainly on the interpersonal aspects of communication (Gallois & Giles, 2015). An intergroup approach takes into account how communication behaviour is influenced by the power differentials that exist between groups. One area that CAT has been applied in is health communication (Nussbaum & Fisher, 2009; Watson & Gallois, 1999), particularly for patient-doctor communication (Coupland, Coupland, Giles, & Henwood, 1988; Street, 2001; Watson & Gallois, 1999). Group memberships are highly salient in doctor-patient interactions because health providers and patients interact as group members and as individuals (Baker & Watson, 2015; Watson & Gallois, 2007). In other words, the patient focuses on the doctor as a member of the doctor group rather than as an individual with likes and dislikes. In the same way, the doctor most often focuses on the patient as a member of the patient group instead of as an individual. While this is not problematic per se, it can cause problems if doctors and patients only interact at the intergroup level. This is because therapist attributes and skills that positively impact the therapeutic alliance, such as being warm and interested, facilitating affect expression, and attending to patient experience (Ackerman & Hilsenroth, 2003), are based on interpersonal interactions. Having summarised the premises that underpin CAT, I now extend my theoretical overview of CAT by linking it to depression research.

Communication accommodation theory strategies and depression. Over the years, communication researchers have highlighted several strategies, which depending on how they are used, can result in either an accommodative or non-accommodative stance (Giles, Gasiorek, & Soliz, 2015). SAT’s focus was on the strategy of approximation, which concerns language production (Watson et al., 2012). Approximation is concerned with the extent to which interactants alter their speech behaviours (such as speed, pitch, and accent) to converge towards their speech partner (linguistic convergence), or to exaggerate linguistic differences (divergence, maintenance), as explained earlier. Approximation will not be investigated in this thesis because there are no recorded interactions between patients and health professionals. In 1988, Coupland et al. expanded CAT by introducing three new communication strategies of interpretability, discourse management, and interpersonal control. Later, the strategy of emotional expression was added (Watson & Gallois, 1998). I briefly explain each strategy with examples relevant to GP-patient communication for depression management.

Interpretability is concerned with the judgement of a speech partner's communication competence (Watson et al., 2012). An individual can be accommodative and use words that they think their conversation partner can understand clearly, or be non-accommodative by using words that the other will not understand. Non-accommodation can be deliberate or unintentional but leads to one speech partner using, for example, medical jargon instead of simplistic language. For example, a doctor may explain the mechanism of neurotransmitters and antidepressants using medical jargon that the patient does not comprehend. The doctor has misjudged the competence level of the patient, which then affects comprehension, decreases patient-doctor trust, and reduces treatment adherence. This was demonstrated in a study by Bull et al. (2002) where 72% of doctor participants said they usually tell patients to use antidepressants for at least half a year, but only 34% of patients recalled being told that. The discrepancy could have been due to interpretability issues.

Discourse management is not about the content but about the process of communication, that is, how one manages a conversation. The strategy of discourse management is reflected in behaviour such as minimal encouragers, silence to prompt someone to speak, and minor interruptions ("tell me more"), which allows a person to engage in the conversation. Watson and Gallois (2004) found that discourse management is an important strategy in managing effective communication. In depression, good discourse management could include joint topic selection by the doctor and the patient (e.g., there is time to discuss mainstream as well as alternative forms of therapy).

Interpersonal control is concerned with role relations in the communication. The patient may be constrained to portray a passive role, while the doctor holds a dominant role, or the patient may move towards a more equitable relationship. Behaviours that reflect keeping the patient passive and the doctor in control are when doctor changes topics, interrupts patient to stop them speaking, terminates the consultation, and does not allow the patient to ask questions.

Finally, emotional expression is about meeting discerned or expressed emotional or relational needs of both interactants. Strategies should enable a depressed individual to share their fears and worries about the condition, with the doctor extending appropriate empathy, reassurance, and understanding in their response. For healthcare professionals, emotional expression entails appropriate management of their own affect (Watson & Gallois, 1999).

Studies by Watson and Gallois (1998, 1999, 2002) found that strategies of interpersonal control, discourse management, and emotional expression strongly predicted patient satisfaction. Patients' ratings of consultations were highest when they perceived that they had some control over the interaction (e.g., they can confirm understanding and question decisions), when there was dynamic engagement, and when they felt reassurance from their healthcare providers. Conversely, Lagacé, Tanguay, Lavallée, Laplante, and Robichaud (2012) found that older patients in aged care had lower perceptions of quality of life when healthcare providers used patronising speech, exerted interpersonal control, and were non-accommodative.

Communication accommodation in primary care. CAT is an ideal theory to use in the context of primary care depression management where patients make multiple visits to the same GP over the course of their illness. Owing to the Better Access initiative in Australia (Australian Department of Health, 2012), a sustained GP-patient relationship is common even for depression patients who consult specialists. This is because Better Access requires patients to obtain GP referrals and reviews for fully subsidised individual consultations and group sessions each year (up to 10 of each).

CAT-based research points to the benefits of medical practitioners facilitating active participation of patients during consultations, whereby patients perceive the clinical encounter as more interpersonally important and constructive (Street, 2003; Watson & Gallois, 2004). Street found that patient engagement in clinical encounters helps to improve patient satisfaction and treatment compliance. Watson and Gallois showed that when doctors intentionally foster rapport with patients through being reassuring and caring, patients also feel more positive about the consultations, viewing them as more beneficial compared to when rapport building is absent.

However, it is insufficient to look at doctors' behaviour alone because causal attribution, as explained previously, also plays a part. The reasons patients attribute to a doctor's accommodative behaviour can influence a patient's response. For example, a GP may take time to carefully explain how antidepressants work because they wish to help the patient make an informed decision about medication. The patient may attribute the doctor's behaviour as an attempt to convince them to agree to antidepressants. Depending on whether the patient is convinced of the biomedical nature of the depression, the GP's communication about antidepressants could be effective or ineffective in building the therapeutic alliance.

This is perhaps why Giles, Coupland, and Coupland (1991, p. 2) said that communication accommodation involves taking into account "constellations of underlying beliefs". In the context of primary care depression management, these beliefs would include the depression EMs of GPs and their patients. More recently, Baker and Watson (2015) said CAT is valuable because it takes into account how participants (doctors and patients) bring their experiences, perspectives, priorities, and beliefs into the intergroup interaction, that is, the clinical encounter. The participants' social background can thus influence them to view the interaction in intergroup terms.

In the same way the EMs of depression patients and GPs also impact on their perception of their interaction. To date there are no studies linking depression EMs and communication accommodation. The few studies that examine communication and depression management have mostly been mentioned in this chapter (e.g., Adams et al., 2015; Karasz et al. 2012), but they neither featured EMs or CAT.

There have been two general (not depression-focused) articles linking communication and explanatory models. The first is Helman's (1985) pilot study, which was discussed in Chapter 3. As it only involved 42 patients and 18 practitioners, it had limited generalisability. Furthermore, CAT was not featured at the time of Helman's study. The amount of agreement between the patient's EM, the doctor's EM, and the doctor's perception of the patient's EM, was used as the measure of communication success and predictor of future communication success. Such a measurement for successful communication is not only crude, but could be inaccurate because a patient's EM could overlap with their doctor's EM purely by chance, and not because of effective communication. In contrast, my study uses a CAT-based survey to measure communication effectiveness, representing a step forward in understanding the link between communication accommodation and depression EMs.

The second article is Bhui and Bhugra's (2004) discussion on the communication complications that arise between people from different cultural and linguistic groups. The authors noted that mental healthcare providers often differ from their patients in terms of their cultural background and illness EMs. As a result, doctors are challenged when handling consultations involving illness EMs from another culture. Anecdotal evidence pointed to the need for mental healthcare providers in multicultural settings to prioritise the reconciliation of different EMs during consultations. Even if EMs differ between patient and doctor, doctors should work on persuading patients that agreeing to disagree is acceptable. Reaching such a compromise quickly requires doctors to avoid being antagonistic and conflictual during the consultation and allowing patients to maintain their own explanations. While Bhui and Bhugra did not mention CAT in their article, I argue that CAT strategies can aid GPs and patients to negotiate these challenging discussions with mutual respect.

In this chapter I described how CAT recognises the significance of shared perspectives (Jones & Watson, 2012), which makes it particularly suited to my study on depression EMs of primary care patients and GPs. If certain EM dimensions are shown to have associations with high scores on the CAT-based survey, it would demonstrate the value of acknowledging patients EMs in improving communication accommodation.

Over the past four chapters, I have reviewed the literature that addresses the fundamental concepts of depression, explanatory models, mental illness stigma, and doctor-patient communication. The next chapter will explain the methodology of my studies to address the RQs outlined in Chapter 1.

Chapter 6: Rationale for Methodology

In this chapter, I outline the rationale underpinning my research methods. I begin by discussing epistemology, that is, the theory of knowledge (Crotty, 1998), which provides the philosophical groundwork for deciding what types of knowledge are possible. This information in turn impacts on how one gathers the desired knowledge. While there is a range of epistemological stances, Crotty's explanation of objectivism and constructionism provides the framework to situate my research.

Objectivism posits that there is objective truth that can be discovered with precision and certainty. Under this epistemology it is vital to have a controlled and structured approach to research, which involves identifying a clear research topic and constructing appropriate hypotheses that can be empirically measured. Inferential statistics is central to objectivist research because the aim is to have generalisable findings from the study sample that can be applied to a larger population. Researchers report probabilities that their findings are accurate, not absolute certainties (Gray, 2014). Quantitative methods such as questionnaires are a common tool under objectivism.

Constructivism, on the other hand, focuses on the meaning that arises from social engagement between objects and subjects – there is no one objective truth. Knowledge acquired about any reality consists of socially constructed perceptions or interpretations rather than objectively determined facts (Carson, Gilmore, Perry, & Gronhaug, 2001; Crotty, 1998). It is sometimes referred to as interpretivism. Instead of a scientific hypothesis-testing framework, constructivism favours a more personal and flexible approach that can better capture the motives, meanings and reasons behind human interactions (Black, 2006). Non-experimental methods are generally preferred where the research setting or subjects are not manipulated and the data are not placed in predetermined categories. Under constructivism the researcher collaborates with the informant(s) to develop new understanding. This emergent approach aligns with the belief that humans are adaptive and that one cannot gain prior insight to context-bound social realities (Hudson & Ozanne, 1988). Qualitative methods such as interviews are a common tool in constructivism.

I positioned my research along the spectrum between objectivism and constructivism because I believe that there are benefits to both approaches. Also, the subject matter that I am interested in can be examined through both paradigms. As explained in the literature review chapters, I am researching explanatory models and how they potentially correlate with levels of patient self-stigma, GP-patient communication effectiveness and depression outcomes. These are subjective mental constructs that can be inferred quantitatively from self-reporting measurements (e.g., attitude scales) and also qualitatively from what people say in interviews (Guthrie, 2010).

Social science methodology has evolved and research is no longer dichotomised between the extremes of objectivism or constructivism. Instead the use of mixed methods has gained importance in social science research, where both quantitative and qualitative methods are used to counteract their respective limitations (Guthrie, 2010). Another reason for using mixed methods is that it can give rise to multiple perspectives that will help to form a rich and deep picture of the issue being studied (Neuman, 2014). Further, the use of mixed methods offers the possibility of obtaining complementary findings that increase the validity and credibility of the results. This is known as triangulation (Webb, Campbell, Schwartz, & Sechrest, 2000), or convergence. My thesis used mainly questionnaire surveys, but I also conducted interviews with a small number of patients and GPs to gain further insight into the role of EMs. I now discuss both methods, along with their strengths and weaknesses.

The Survey Method

Groves et al. (2009, p. 2) defined the survey as “a systematic method for gathering information from (a sample of) entities for the purposes of constructing quantitative descriptors of the attributes of the larger population of which the entities are members”. Surveys have long been used in the social sciences, as it is useful for understanding patterns in large groups and for describing people’s general attitudes. Longitudinal surveys show how people’s views change over time. The use of questionnaires is one of the main research techniques in the survey method (Guthrie, 2010). A good questionnaire must have the following characteristics: discrimination, validity and reliability (Field, 2003).

Discrimination means that people who differ in their attitudes about any particular questionnaire topic will end up with different questionnaire scores. It requires selecting items that are not confounded by other factors. Also, the degree of difference between respondents should be proportionate to the difference in scores from the questionnaire. For example, if two participants on a stigma survey score 15 and 30 respectively, the survey has good discrimination if the second participant does actually experience twice as much self-stigma than the first. Discrimination is the basis for validity and reliability, which I will explain next.

Validity. Validity refers to how accurately a questionnaire measures what it sets out to measure (Babbie, 2015). The first aspect of validity examines the appropriateness of the content of the instrument, which is in turn made up of face validity, construct validity, and content validity.

There is some variation in the use of these validity terms, but for the purpose of this thesis, face validity is whether the instrument appears to measure what it claims to measure. When rating the face validity of an instrument it is important to choose the right individuals who have the relevant expertise. Evaluating readability, feasibility, clarity of wording, layout and style of the questionnaire is also a part of assessing face validity (Acharya, Thomas, & Cann, 2016). Babbie defined construct validity as the extent to which an instrument relates to other probable variables within a theoretical scheme of associations. For example, the construct validity of an intelligence test would depend on a theory of intelligence. Construct validity is determined using factor analysis and other correlational methods. Finally, content validity is how comprehensively a measure covers the range of meanings found in a concept. For example, a measure for prejudice needs to consider different types of prejudice arising from gender, race, age, and so forth

The second broad aspect of validity examines how an instrument relates to other measures and is known as criterion validity. It is made up of concurrent validity and predictive validity (Nelson & Fife, 2013). Concurrent validity is the extent to which an instrument corresponds to an external criterion that is presently known to be valid. For example, a new personality test might be compared with an older test that is already known to have high validity. Predictive validity is the ability of an instrument to forecast an outcome. So if the aforementioned personality test makes the prediction that certain personalities will be more likely to have certain job preferences, a survey of job preferences will confirm or disprove its predictive validity.

Reliability. It is not enough for a questionnaire to be valid – it must also be reliable. Reliability is the ability of a questionnaire to produce similar results repeatedly under similar conditions (Babbie, 2015). It can be measured by estimating correlation coefficients. Reliability can be measured in terms of stability, equivalence and internal consistency (Bolarinwa, 2015). The test-retest method is used to assess stability, to ensure that same results are obtained when the same participant completes the questionnaire twice at different times. However, it is only useful for stable traits and the researcher needs to be aware of the practice effect and the effect of memory if the interval between tests is too short. To assess equivalence, the inter-rater method is used. This is where two or more observers independently study a single phenomenon at the same time using the same instrument, and the percentage of agreement between them is calculated. To assess internal consistency, the split-half method is used to determine whether all sub-parts of a scale or instrument measures the same characteristic (homogeneity). The content of the questionnaire is divided into two and the scores of the first half are correlated with scores of the other half. Large correlations indicate high reliability. However, there are a few ways to divide a data set and the results could be due to the way the set was split. To overcome this, Cronbach (1951) proposed splitting the data in every way possible and computing the correlation for each split. The average of these values is known as Cronbach's alpha (Fonseca, Costa, Lencastre, & Tavares, 2013) and is the most common measure of scale reliability (Peterson, 1994). Nunnally and Bernstein (1994) said an alpha of .70 reflects modest reliability and is acceptable in the early stages of research, a recommendation that applies to the exploratory nature of my thesis.

Question types. A questionnaire is basically a standardised set of questions, made up of a mix of open-response or closed-response questions. Good open-response questions are high on validity because participants use their own words to give comprehensive answers. Reliability can be a problem if different researchers get different answers, even though the question is the same (Guthrie, 2010). On the other hand, closed-response questions are less valid because respondents are restricted in their choice of pre-determined answers, but they are more reliable. Adding the other category and a simple probe for further explanation (e.g., "Why?" or "Please explain") can increase the validity of fixed-choice questions.

Guthrie (2010) listed the different types of responses to closed questions by complexity – starting with the most basic, which are categorical responses where participants usually choose one response such as yes or no. This is followed by checklist responses (participants are given more categories and they can choose more than one). Ranked responses and scaled responses are next. In the latter, respondents choose from options that correspond from one extreme to another (also known as the semantic differential such as high-low or good-bad), with scores in-between for the variable in question. Scaled responses can measure attitudes and opinions by combining the results on a number of questions. This is because people are not uni-dimensional in their thinking, so it is not uncommon to have 10 questions to capture a single attitude.

One of the most common scaled response formats used in attitude research involves Likert items. First introduced by Rensis Likert in 1932, they are relatively easy to develop and administer (Narli, 2010). However, there are debates about what type of analysis is appropriate for data produced by Likert items – some argue that Likert items produce data that should be analysed as ordinal data (Jakobsson, 2004; Jamieson, 2004). Clason and Dormody (1994) argued that using methods focused on location parameters (i.e., mean and standard deviation) would oversimplify the analysis, leading to inferential errors. Others suggest complex solutions to the perceived problem that the intervals between the options for a Likert item are irregular and arbitrary (Albaum, 1997;

Hodge & Gillespie, 2003). The more practical academics advocate combining multiple Likert items to form scales or indexes (e.g., Carifio & Perla, 2008), instead of analysing individual items. Brown (2011) cited researchers who have demonstrated that Likert scales can be effectively analysed as interval data as long as certain conditions are met. These include having at least five options to cover the range of responses (e.g., strongly agree, agree, neutral, disagree, strongly disagree) and checking the Likert scales for validity and reliability (Allen & Seaman, 2007).

Where it may not be appropriate or feasible to have multiple Likert items to form scales, the opinion of researchers is again divided. In such cases, Clason and Dormody (1994) argued that non-parametric methods are preferable to parametric methods. For comparing between two groups, the non-parametric Mann-Whitney's test is the equivalent of the parametric *t*-test. Over the years, many researchers have concluded that the Mann-Whitney's test is less powerful than the *t*-test for data that has normal distributed and equal variances (e.g., Baker, Hardyck, & Petrinovich, 1966; Heeren & D'Agostino, 1987; Rasch & Guiard, 2004). However, these studies have often examined relatively simple and continuous distributions and not the complex and truncated distributions of Likert-item data, with its distinct characteristics of discrete variables, tied numbers and limited range (De Winter & Dodou, 2010). De Winter and Dodou found that the Type I and II error rates of the *t*-test and Mann-Whitney's test were mostly comparable for five-point Likert items. They defined 14 population distributions in their study, drew pairs of samples from the populations, and conducted the *t*-test and the Mann-Whitney's test with these pairs. Results showed that the two tests had equivalent power for most of the pairs, except for the pairs from the population with skewed/peaked distribution (Mann-Whitney's test was more powerful) and the pairs from the population with a multimodal distribution (*t*-test was more powerful). In addition, the Type I error rate of both methods never exceeded 3% above the nominal rate of 5%, even when sample sizes were highly unequal.

Designing questionnaires. It takes exacting preparation to develop and trial a good questionnaire so researchers often use or adapt existing questionnaires that have already been tested for validity and reliability. Using available scales is also effective and efficient because the same measures can be used in different studies, which results in consistency and the opportunity to compare findings from different studies. Questions must be clear and each question must relate to the research problem. Therefore, questionnaires are pilot-tested to improve question clarity and reduce unnecessary questions (Guthrie, 2010). Questionnaire length is another factor to consider, especially for self-completion questionnaires. Long questionnaires with many questions run the risk of not being completed, though the threshold for what constitutes a long questionnaire depends on the motivation of the participants. Since my target population of people with depression may be low in energy and have difficulty concentrating, this is an important factor in my questionnaire research design.

Implementing questionnaires. Guthrie (2010) listed several ways to implement questionnaire surveys. They can broadly be grouped into two broad categories, which I have termed in-person methods and mediated methods. In-person methods range from the random soliciting of responses on the street to the more targeted approach of going to where respondents are likely to be. I decided against random soliciting because my target population is people with depression, who are very unlikely to admit their condition to a stranger approaching them. I also did not wait at GP clinics to recruit participants, as this would not have been an effective use of time. Clinic practice managers are also unlikely to allow researchers to approach patients in the waiting areas, as that may be seen as harassment. I attempted a group administration of my questionnaire because it was more efficient than approaching participants individually. However, I was unsuccessful in getting access to groups of people who were depressed (e.g., one gatekeeper of a depression support group turned down my request to talk about my research at their gatherings).

Mediated methods involve distributing questionnaires through the mail, phone or the Internet. As postal addresses of people with depression were unavailable, this method was not possible. I did not use the phone method to randomly find people with depression because firstly, many people do not list their contacts in the phone directory. Secondly, cold calling is likely to lead to rejection and high non-response rates due to the high probability of the researcher being mistaken for a telemarketer. Instead, I chose a web-based questionnaire for my study because it afforded the anonymity that would encourage people with depression to participate. Participants were invited to take part through flyers and social networks; and for those without online access, they could request for mailed hard copies of the questionnaire. This mixed-mode administration raises the issue of sampling, which is an important consideration in survey methodology.

Sampling considerations. Traditionally, questionnaires are distributed in person or through the mail – both of which allow for a response rate to be calculated. A high non-response rate affects the validity of the data by introducing response and non-response biases. The increased use of web-based questionnaires has necessitated convenience-sampling methods where response rates cannot be calculated. This is because it is not possible to know the reach of a poster or flyer, so I could not determine the baseline for response rates. Instead, I assessed the representativeness of my sample by looking at the spread of participants in terms of gender, age, economic status, educational background and locality (where participant provided the name of suburb they were living in). Using non-randomly obtained convenience samples means that results will have limited generalisability but this is acceptable given the exploratory nature of the studies in this thesis. The mixed-mode administration also led to considerations over combining online questionnaire data with paper questionnaire data.

Comparing online questionnaires with paper versions. Sax, Gilmartin, Lee and Hagedorn (2003) analysed the response rates, non-response bias, and response bias across two groups of community college students – those who received a follow-up questionnaire of their college experiences via email and those who received it by standard mail. Sax and colleagues chose to research community college students because they are known for being difficult to locate and have historically had below-average participation rates. My method for reaching my participants was different from Sax and colleagues because I did not have participants' e-mail prior to the survey. I instead distributed flyers and posters with the questionnaire link to GP clinics and other locales, and I used social media – see Chapter 7). However, my target population was similarly difficult to locate with probable low participation rates due to the stigma surrounding depression (see Chapter 4).

Sax et al. (2003) found that response rates for the online questionnaire group were double that of the paper questionnaire group. In both online and paper questionnaires, women had higher response rates, a finding that is consistent with other similar studies. They found that gender gap in response rates is narrower in email administration. Despite these differences, they concluded that email and paper administration of questionnaires yielded similar enough findings that the results from the two methods can be aggregated.

Benefits and limitations of questionnaires. The following discussion of the pros and cons of questionnaires is based on a university introductory module to research ("The advantages and disadvantages of questionnaires", n. d.). Practicality is a key advantage of questionnaires. Large amounts of data can be collected from a high number of participants within a short period of time, for relatively low cost. This is especially true of online questionnaires that are hosted on university servers for free. Findings from questionnaires can be quickly collated and analysed using statistical programs. When data has been quantified, it can be used to compare with other research studies that employ questionnaires. Analysing questionnaire data can be arguably more objective and scientific than other forms of research such as interviews. Questionnaires also enable change to be measured easily.

The disadvantages of questionnaires include not being able to ascertain how truthful participant responses are. The researcher also has no control over how much effort respondents put into answering the questionnaire – the latter could read questions wrongly or be forgetful. There is also unacknowledged subjectivity in questionnaires that occurs when people read differently into each question and reply based on their own understanding of the question. This would compromise the validity of the aggregated results. As discussed earlier, issues of validity should be addressed when designing questionnaires, for example by having a piloting process to clarify questions.

Questionnaire topics and the options available for answering the questions are usually pre-determined by the researchers, thereby limiting the potential scope of participant responses. For example, one of the dimensions in the CONNECT instrument (mentioned in Chapter 3) is biomedical cause, where “a higher score indicates a more biomedical perception of cause” (Haidet et al., 2008, p. 237). The instrument will not be able to capture the difference between a participant who thinks evil spirits are the cause of their illness and a participant who attributes their illness to work stress because their score for this dimension would be similar. Other researchers may choose to address this issue but the point remains that questionnaires will inevitably reflect what the researcher deems important. Researcher assumptions are therefore imposed upon respondents (Galasiński & Kozlowska, 2010). One of the ways to counter the weaknesses of questionnaires is to incorporate the use of qualitative interviews, which I will now elaborate on.

The Interview Method

An interview is essentially a conversation with a purpose that is determined by the interviewer (Kvale, 2007). It is an important research method to uncover how people make sense of their experiences as described in their own words. A common type of research interview is the semi-structured qualitative interview, defined as “an interview with the purpose of obtaining descriptions of the life world of the interviewee with respect to interpreting the meaning of the described phenomena” (Kvale, p. 8). A semi-structured interview has a sequence of topics to be covered as well as some predetermined questions but it is flexible to change depending on what the interviewee discloses. I will now discuss the benefits and limitations of the semi-structured interview method.

Benefits and limitations of semi-structured interviews. Semi-structured interviews can benefit both the researcher and the subject. Firstly, interviewers can prepare questions ahead of time. The use of similar questions results in reliable and comparable qualitative data. Information from semi-structured interviews can help to answer the higher-order “why” and “how” questions. Secondly, on the part of the interviewee, they have more freedom to express themselves in a semi-structured interview as the questions act as a guideline instead of a rigid framework. The subject can ask questions of the researcher, thereby facilitating two-way communication.

However, interviews are time-consuming and resource intensive. It is costly to transcribe interview recordings, and extensive effort is needed to code and analyse interview data properly. This is why the number of interview subjects is usually low, numbering in the tens compared to the hundreds or even thousands of questionnaire survey participants. The interviewer is the research instrument and must be skilled in interviewing as well as in analysing the data without construing too much.

During the interview the researcher has to be prepared to deal with any situation that may arise. Part of the preparation involves piloting the interview questions to ensure that they are clear and not leading. Another limitation of interviews is the interviewer effect. This is where interviewees differentiate their responses (how much they are willing to disclose and how honest they are) depending on their perception of the interviewer (Denscombe, 2010). Similarly, the researcher could also be influenced by their perception of the subject. One of the ways to mitigate this problem is to have telephone interviews.

Telephone versus in-person interviews. Traditionally, interviews involved one interviewer meeting face-to-face with a single interviewee. Advances in technology now enable interviews to be conducted through the telephone or the Internet, using Voice over Internet Protocol video calls (e.g., Skype). The risk of the interview being influenced by the visual perceptions of the researcher and/or the subject is reduced in a telephone interview. As both the in-person and telephone interview methods are used in this study (details in Chapter 7), I will now discuss how they differ.

Telephone interviews help to save on cost by reducing travel and they enable researchers to interview subjects regardless of their geographical location (Novick, 2008). Conducting phone interviews is also safer for the interviewer. Despite these advantages, telephone interviews are used less frequently compared to in-person interviews in qualitative research (Opdenakker, 2006). This is due to the perception that telephone interviews are not as ideal as meeting face-to-face because visual cues are absent. The lack of visual cues is said to result in the loss of contextual information from non-verbal communication, the failure to build rapport or to probe, and the misunderstanding of responses.

However, Novick (2008) argues that the absence of information from non-verbal communication in telephone interviews may not be that significant a loss because such non-verbal data can be easily misinterpreted (Sturges & Hanrahan, 2004). Often such data would be excluded in analysis methods that depend on transcripts, making them not dissimilar from in-person interview transcripts. Also, there are ways of compensating for the lack of visual cues such as by noting intonation (Opdenakker, 2006), sighs and pauses (Sturges & Hanrahan). The problem of rapport can be overcome by having an informal chat before starting the interview, and choosing words and intonation carefully to reflect an empathic and non-judgmental response to what interviewees share. Respondents have been known to be uninhibited on the telephone and willing to share sensitive information freely, which is why there is growing evidence that qualitative telephone interviews yield high-quality, in-depth data (Sturges & Hanrahan; Sweet, 2002). Telephone interviews foster greater privacy and anonymity, and reduce social pressure than in-person interviews because subjects are allowed to speak from the comfort of their own space (McCoyd & Kerson, 2006). Nevertheless, regardless of what interview mode is used, there are power and emotional dynamics to consider, which I will discuss next.

Power and emotional dynamics of interviews. Power initially resides with the interviewer, because the interviewer decides whom to interview and what to interview about (Kvale, 2007). However, once the interview begins, the balance of power becomes more complex as power shifts dynamically between the researcher and the subject. Hoffman (2007) calls it “the interview dance” because the interview’s progress is determined by both the interviewer questions and the interviewee’s responses. It is important to consider social divisions and hierarchies involving socio-economic status, gender, ethnicity, age, and other factors. This is especially true when interviewing marginalised or stigmatised groups such as people with mental illness – care must be taken to prevent further disempowerment of the subjects during the interview.

Care must also be taken with respect to the emotional aspects of interviews. Both researchers and subjects may become emotionally engaged through the shared interview experience. The subject may speak of emotionally charged events or beliefs while the researcher may feel the emotional strain of creating rapport. The latter experience is further complicated by the dilemma of how much emotion to share: too much and the researcher ends up silencing the subject; too little and the researcher appears unresponsive or unable to understand the subject’s experience (Hoffman, 2007). In conclusion, power and emotions in qualitative interviews will inevitably affect the research process, but researchers argue that reflecting on these dynamics will enhance the process of knowledge generation and provide a deeper understanding of the research topic (Hoffman; Schwalbe & Wolkomir, 2002).

In this chapter, I provided justification and explanation for the methodological approaches used in my research. The next chapter will elaborate on the specific details of how I collected and analysed my data.

Chapter 7: Methods

This chapter describes the research setting of my thesis topic and the process of participant recruitment. I explain the quantitative instruments used to survey participants about depression explanatory models (EMs), self-stigma, patient-GP communication effectiveness and treatment outcomes, as well as the qualitative questions guideline used to interview patients and GPs. An outline of the statistical analysis used to analyse the data collected between March 2014 and January 2016 concludes the chapter.

Research Context

My research involved participants residing in Australia primarily because I am based in Brisbane and can more easily access such participants for surveys and interviews. In addition, there are advantages for researching depression EMs here. Firstly, there is a general awareness about mental illness in Australia that has been cultivated from previous mental health campaigns over the years; for example, *beyondblue* started in 2000 (Highet, Luscombe, Davenport, Burns, & Hickie, 2006). As such, one can expect greater depth in discussing depression perspectives or EMs. Secondly, Australia as a multicultural nation provides a context to show whether participants from different cultures reflect differences in their depression EMs. As explained previously, EMs can be influenced by culture (see Chapter 3). As of June 2016, 28.5% or 6.9 million of Australia's estimated resident population were born overseas (ABS, 2017a). Australians identify with 270 ancestries and speak over 260 languages, with four million speaking a language other than English (Australian Department of Social Services, 2011).

Participants

There were two different groups of survey participants – one group consisted of individuals who had sought primary care treatment for depression or depressive symptoms (Time 1 $n = 238$; Time 2 $n = 129$) while the other comprised GPs whose patients were study participants who consented to their doctors joining the study ($n = 29$). The terms depression and depressive symptoms were used in the survey instruments without further qualification. This was deliberate because it was assumed that there would be multiple constructs of depression (see Chapter 2) so this thesis avoids narrow definitions of the condition. I collected longitudinal data from the first group by asking the participants to complete two surveys spaced six months apart (henceforth known as Patient Survey 1 and Patient Survey 2). Patient Survey 1 participants were recruited between March

2014 and June 2015, and Patient Survey 2 participants were completed between September 2014 and January 2016. Most of the respondents completed the surveys online; hard copy responses came from less than 5% of participants for both surveys. The GP participants were surveyed once. Both patient and GP participants were convenience samples. The criteria and method of recruitment for the two types of participants will be explained later.

Participant characteristics. Data screening of participants who did not meet the inclusion criteria (or who took the survey twice) removed a total of 24 cases (9.2%) from Time 1, leaving 238 participants. For Time 2, seven cases were removed (5.1%), leaving 129 participants. The demographic information for the two samples is outlined in Table 1. Demographic information is missing for some Time 1 participants (depending on the variable the proportion ranged between 4.2% and 7.6%).

The relative proportion of participant characteristics was comparable between both timeframes, that is, the ratio of female to male participants was about 3:1 at both Time 1 and Time 2. The higher proportion of women reflects the gender difference in depression as mentioned in Chapter 2. Participants were generally diverse in terms of age and average household income level. The income categories were based on the 2011-2012 ABS Survey of Income and Housing (ABS, 2013a). Slightly more than half the participants were employed, in line with a previous study that found depression rates in the employed to be similar to those who are unemployed (Mihai, Ricean, & Voidazan, 2014). Roughly a third of the participants were born overseas, which is comparable to the latest national estimates (ABS, 2017a). However, the proportion of White Caucasian/European participants was twice that of the national sample (36%; ABS, 2017b). This reflects greater willingness of such participants to seek mental health treatment compared to other ethnic groups. This has been noted by Herman et al. (2011) in a study of college students, which found that European American students were 3.7 times more likely to have received treatment for depressive symptoms than Asian American, Native Hawaiian, and Pacific Islander students. Finally, the proportion of participants with a Bachelor's degree or higher in this study was double that of the national sample (30% in 2012; ABS, 2013b). This is because recruitment efforts began and was sustained at the University of Queensland. The difference between the patient sample and the national sample impacts upon the former's representativeness and thereby limits the generalisability of findings. However, this is acceptable given the exploratory nature of this thesis' studies.

Table 1

Characteristics of Participant Patients in Absolute Numbers and Percentages

Characteristics	Time 1 number (%)	Time 2 number (%) $n_2 = 129$
Gender ($n_1 = 228$)		
Male	57 (25.0)	31 (24.0)
Female	171 (75.0)	98 (76.0)
Age in years ($n_1 = 228$)		
18-24	76 (33.3)	35 (27.1)
25-39	96 (42.1)	56 (43.4)
40-54	41 (18.0)	34 (26.4)
55-65	15 (6.6)	4 (3.1)
Country of birth ($n_1 = 225$)		
Australia	152 (67.6)	93 (72.1)
Overseas	73 (32.4)	36 (27.9)
Culture/Ethnicity ($n_1 = 224$)		
Aboriginal and/or Torres St Islander	5 (2.2)	2 (1.6)
Asian	37 (16.5)	14 (10.9)
White Caucasian/European	163 (71.5)	102 (79.1)
Other	19 (8.3)	11 (8.5)
Education ($n_1 = 220$)		
Bachelor degree or higher	138 (62.7)	87 (67.4)
Certificate/Diploma/Advanced Diploma	46 (20.9)	21 (16.3)
Up to Year 12 or equivalent	36 (16.4)	21 (16.3)
Employment ($n_1 = 224$)		
Yes	137 (61.2)	86 (66.7)
No	87 (38.8)	43 (33.3)
Average household income ($n_1 = 227$)		
High income[a]	43 (18.9)	22 (17.1)
Middle income[b]	79 (34.8)	47 (36.4)
Low income[c]	73 (32.2)	46 (35.7)
Don’t know/Prefer not to disclose	32 (14.1)	14 (10.9)

Note. The categories African, Latin American, and Middle Eastern were subsumed under Other. Participants who specified other culture/ethnicity were placed in applicable categories or Other if none were suitable, along with those who were unsure. [a]Income after tax averages $1,800/week or $93,600/year. [b]Income after tax averages $780/week or $40,500/year. [c]Income after tax averages $450/week or $23,400/year.

Table 2 summarises participants' health-related characteristics at Time 1, for example, onset of depression/depressive symptoms and whether they have other conditions (co-morbidity). It also shows how long participants experienced depression symptoms before they recognised the symptoms to be depression (unrecognised depression), and the period of time between realising they have depression and consulting a healthcare professional for their condition (untreated depression). As these were free-text questions, some participants responded very generally; for example, some said they had experienced depression all their lives but did not give a specific time frame. These constituted the missing data (depending on the variable the proportion between 7.6% and 15.5%).

The mean onset of depression was 10 years ago (range = 0.039-45, *SD* = 9.3) for the 208 participants who answered this question (Table 2). In addition, the largest group of participants were those who had long-standing depression (>10 years), accounting for 36%. The mean length of unrecognised depression was 3.90 years (range = 0-30, *SD* = 4.95) based on 197 participants; and the mode was a year or less (44.7%). For length of untreated depression, the mean was 2.6 years (range = 0-25, *SD* = 4.2) based on 192 participants; mode was also a year or less (65.7%).

As for psychiatric co-morbidity, Table 2 shows categories of conditions that afflicted more than 10% of the participants at Time 1. The categories are based on the DSM-5 (APA, 2013), which listed obsessive-compulsive disorder separately from anxiety disorder. This was a change from the DSM-IV. For a complete list of co-morbid conditions, both psychiatric and physical, refer to Appendix B. Slightly more than half of the participants rated their physical health at the time of both patient surveys as fair. Almost one-third of them felt that their physical health had a moderate impact on quality of life, while another third felt the impact was only slight.

Health characteristics of participants in both timeframes were mostly similar except for the proportion of participants who had been formally diagnosed with depression. A chi-square goodness-of-fit test showed that the percentage at Time 2 (86%) was significantly higher than at Time 1 (76.8%), $\chi^2(1, N = 129) = 6.19, p = .013$, with small-to-medium effect size (Cohen's $w = 0.22$). The test also showed that percentage of psychiatric co-morbidity at Time 2 (14%) was not statistically significantly different than at Time 1 (18.1%), $\chi^2(1, N = 129) = 1.50, p = .22$, with small effect size (Cohen's $w = 0.11$). Anxiety, phobia, and panic disorders were the most common, affecting about four-fifths of participants who had specified a co-morbid condition at both timeframes. Some of the other conditions mentioned were post-traumatic stress disorder, borderline personality disorder, and obsessive-compulsive disorder. Some participants had more than one co-morbid condition.

Table 2

Health Characteristics of Participant Patients in Absolute Numbers and Percentages at Time 1

Characteristic (n_1)	Number (%)
Diagnosed with depression (220[a])	169 (76.8)
Earliest depression onset (214[a])	
≤ 1 year ago	39 (18.2)
> 1 and ≤ 5 years ago	48 (22.4)
> 5 and ≤ 10 years ago	50 (23.4)
> 10 years	77 (36.0)
Unrecognised depression (206[a])	
≤ 1 year ago	92 (44.7)
> 1 and ≤ 5 years ago	59 (28.6)
> 5 and ≤ 10 years ago	38 (18.4)
> 10 years	17 (8.3)
Untreated depression (201[a])	
≤ 1 year ago	132 (65.7)
> 1 and ≤ 5 years ago	42 (20.9)
> 5 and ≤ 10 years ago	16 (8.0)
> 10 years	11 (5.5)
Co-morbidity with mental illness[b] (238)	43 (18.1)
Current physical health (219[a])	
Poor	45 (20.5)
Fair	111 (50.7)
Good	63 (28.8)
Impact on quality of life (218[a])	
Severely	34 (15.6)
Moderately	69 (31.7)
Slightly	69 (31.7)
Not at all	46 (21.1)

Note. Participants were not asked about depression onset or length of unrecognised or untreated depression at Time 2. [a]Variation in *n* due to missing/unspecific responses. [b]Where participants specified at least one condition, or symptoms indicative of a condition, besides depression.

Materials

Quantitative instruments.

EM Instrument. The original CONNECT instrument consists of 19 statements covering six dimensions of illness EMs (Haidet et al., 2008). Four of the original six dimensions were renamed for greater precision (e.g., internal locus of control is more accurate than patient fault). The conceptual definitions of all six dimensions were retained (pp. 237-238), with the exception of the causation dimension, which was expanded to include psychosocial aetiology for depression. I also added a seventh dimension of attitudes towards antidepressants after a suggestion by a participant in the piloting process (described later).

The seven dimensions in my modified CONNECT instrument for depression and their definitions were as follows:

1. Causation: A person's perception of the degree to which the depression is perceived to be of a biomedical nature (i.e., following a disease-oriented model of illness) or a psychosocial nature. A higher score for the respective statements indicates a more biomedical/psychosocial perception of the cause.
2. Internal locus of control: The degree to which the person perceives that his or her own actions contributed to the depression. A higher score indicates a perception of greater responsibility for one's illness.
3. Patient agency: A person's perception of their ability to improve the depression (i.e., decrease frequency of symptoms). A higher score indicates a perception of greater agency.
4. Effectiveness of non-medical therapies subscale: A person's perception of the effectiveness of non-medical therapies. A higher score indicates a perception of the greater effectiveness of non-medical, or alternative/complementary therapies.
5. Significance subscale: A person's perception of the significance depression has in their life. A higher score indicates a perception of the more central meaning of illness to one's overall life.

6. Preference for partnership: The extent to which the patient wants to be partners with their GP in managing the depression. Partnership implies patient and GP both sharing in decision-making and information exchange. A higher score indicates a greater desire to have a partnership with one's GP.
7. Beliefs about antidepressant: Participants' perception of aspects of antidepressant use such as the necessity of antidepressants, problems arising from harmful side effects, and over-prescription by doctors.

My modified CONNECT instrument for patients had 16 items. An example of a statement was "I have depression because something in my body is not functioning efficiently (e.g., brain chemistry imbalance)." Participants indicated agreement using a 6-point Likert-type scale ranging from 1 (*strongly disagree*) to 6 (*strongly agree*). The full list of statements is in Appendix C1. Most of the items were analysed as individual items due to low Cronbach alphas for the dimension subscales. The exception was belief in non-medical therapies and belief in the significance of depression. Initial Cronbach α for the subscales of belief in non-medical therapies and belief in the significance of depression were .58 and .57 respectively. The ordinal for the two subscales showed a negligible improvement (.62 and .63 respectively). The inter-item correlation values for the non-medical therapies subscale were all between .20 and .40 except for the alternative providers and complementary medicine pair. When the alternative providers item was excluded from the analysis, it increased α marginally to .59, but doing the same for the complementary medicine item decreased α. Consequently, belief in alternative providers was analysed as an individual item in subsequent analyses. Similarly, all inter-correlations involving the item on positive outcomes from depression in the significance of depression subscale were below .2, so the item was excluded from the subscale, resulting in a revised α of .72. In the interest of keeping the analysis consistent for RQ1a, all 16 items measuring patient's depression EM beliefs were analysed as individual variables first. The subscales for non-medical therapies and significance of depression were only analysed if no significant results were found for the individual variables.

Patients were also asked whether they thought their GP shared their views about depression causation; internal locus of control; sense of agency; non-medical therapies; and significance of depression. For the sake of brevity each dimension was represented by a single question; for example, for the internal locus of control dimension the question was "Do you think your GP views your depression in the same way as you do, in terms of how much your actions contributed to your condition?." Patients could respond with yes; no; or do not know/unsure.

In surveying the GPs I aimed to uncover their EM for their patients' depression, that is, how would they explain this patient's depression. Therefore the 16 statements in the modified CONNECT instrument were phrased accordingly to mirror the items on the patient EM version of the instrument. A sample statement in the GP EM instrument was "This patient has depression because something in his/her body is not functioning efficiently." I also asked GP participants for their perception of how their patients viewed their depression. A sample statement on GP-perceived patient EM was "I think this patient thinks he/she has depression because something in his/her body is not functioning efficiently." Participants indicated agreement using a 6-point Likert-type scale ranging from 1 (*strongly disagree*) to 6 (*strongly agree*). The complete list of items asking for GP EMs and GP-perceived patient EM is in Appendices C2 and C3.

Self-stigma instrument. The self-stigma instrument was only for patients with depression. I combined the 16-item Self-Stigma of Depression Scale (Barney et al., 2010) with a modified version of the 29-item Internalised Stigma of Mental Illness scale by Ritsher et al. (2003), which used the term *depression* in place of *mental illness*. Where items overlapped between the two scales, I chose the wording of items in the ISMI scale because it had higher psychometric ratings (Stevelink et al., 2012). After the piloting process, 22 statements were either removed or combined with other statements. The items were thematically grouped a priori into six dimensions, four of which were from the ISMI scale. The other two subscales of help-seeking inhibition and self-blame were from Barney et al.'s survey. The subscales definitions, based on the work of Ritsher et al. and Barney et al., were as follows:

1. Alienation: The degree to which respondents feel they are less than full members of society
2. Stereotype endorsement: The degree to which respondents concur with common stereotypes about people with depression
3. Discrimination experience: Respondents' perception of the way that they currently tend to be treated by others
4. Social withdrawal: The degree to which respondents avoid interacting due to their illness
5. Self-blame: The degree to which respondents think it is their fault that they are not coping – whether functionally or relationally
6. Help-seeking inhibition: The degree to which respondents experience internal barriers over seeking professional help for their depression

In order to lower the chances of participants feeling overwhelmed by negativity in thinking about self-stigma, some statements were rephrased to make them positive. For example, the statement “I feel embarrassed about seeking professional help for depression” became “I do not feel embarrassed about seeking professional help for depression.” Ritsher et al. (2003) had noted respondents’ spontaneous comments that they were glad to see positive statements among the negative ones in the ISMI scale. The resulting instrument, which I refer to as the modified ISMI-depression scale, had 23 items, of which nine were reverse-coded. These nine items also served as a validity check. The full list is in Appendix D. Participants indicated agreement using a 6-point Likert-type scale ranging from 1 (*strongly disagree*) to 6 (*strongly agree*).

In the study addressing RQ2, three of the six self-stigma subscales had acceptable Cronbach alphas according to Nunnally and Bernstein’s recommendation (1994) for exploratory research: alienation (α= .75), discrimination experience (α= .86), and social withdrawal (α= .79). An example of an alienation subscale statement was “I feel isolated and/or out of place in the world because I have depression.” An example of a statement in the discrimination experience subscale was “I feel ignored or taken less seriously by others because I have depression.” The social withdrawal subscale had statements like “I don’t talk about myself much because I don’t want to burden others with my depression.”

In addition to Nunnally and Bernstein’s (1994) guideline, I considered Hinton, Brownlow, McMurray, and Cozens (2004) who said Cronbach alphas of .50-.70 indicate moderate reliability. Therefore subscales in my studies that reported alphas between .50 and .70 were further analysed by calculating ordinal omega and inter-item correlation. Gadermann, Guhn, and Zumbo (2012) proposed the use of ordinal omega for Likert-type data with two to seven responses because internal consistency estimates for ordinal data can be inaccurate and underestimated if one assumes interval level data. For scales comprising less than 10 items, as is the case in my studies, Pallant (2013) recommended checking inter-item correlations, with a recommendation from Briggs and Cheek (1986) that these should range between .2 and .4.

The help-seeking inhibition subscale had an $\boldsymbol{\alpha}$ of .61 and an ordinal ω of .72. The inter-correlations between items ranged between .25 and .44. Taken together, this subscale showed sufficient internal reliability to be used in further analysis. A sample statement was “I would feel embarrassed if others knew I was seeking professional help for depression.” The stereotype endorsement subscale had a Cronbach alpha of .55 and an ordinal ω of .60. However, more than half of the inter-correlations between items (e.g., “Having depression means I am not suited for a long-term relationship or marriage") were below .20. Therefore the stereotype endorsement subscale was excluded from further analysis.

As for the self-blame subscale, it has only two items and for such scales, Eisinga, Grotenhuis, and Pelzer (2013) recommend using the Spearman-Brown coefficient to evaluate reliability. The Spearman-Brown coefficient was .18 for the self-blame subscale so it was excluded from further analysis as well.

Communication effectiveness instrument. I used the CAT strategies of inter-personal control, discourse management, emotional expression and interpretability that were described in Chapter 5. Based on these strategies, Watson and Gallois (1999) authored the original 16 statements to survey patients on their perceptions of their communication with their doctor. Their definitions were adapted to my research as follows:

1. Interpersonal control: The degree to which a participant is not constrained to in their role as a patient and felt that were viewed by the GP as an individual.
2. Discourse management: The degree to which a participant perceives mutual topic-sharing and appropriate levels of floor holding.
3. Emotional expression: For participants who are patients, it is the degree to which they think they are able to voice their concerns.
4. Interpretability: The degree to which the participant thinks that the interaction is clear and easily understood.

After a brief piloting process, I removed two statements "The GP controlled the consultation" and "The GP managed the consultation well" because they were vague. Statements that covered more specific aspects of the consultation, that is, "This GP gave me as much time as I needed to explain my feelings and situation" and "I felt I was able to express my needs clearly" were used instead. All participants indicated agreement using a 6-point Likert-type scale ranging from 1 (*strongly disagree*) to 6 (*strongly agree*). All four subscales showed acceptable reliability: interpersonal control (**α** = .85), discourse management (**α** = .94), emotional expression (**α** = .93) and interpretability (**α** = .88). The full list of items for the communication effectiveness instrument is in Appendix E.

Depression symptoms scale. The Clinically Useful Depression Outcomes Scale (CUDOS) by Zimmerman et al. (2008) was used to track patient symptoms of depression. Patients rated how well the 16 symptom items (e.g., My energy level was low) described them "during the past week, including today" using a Likert scale ranging from 0 (*not at all true*) or 0 days to 4 (*almost always *true*) or every day. They also indicated whether the symptoms interfered with their lives and to rate their quality of life in the past week (items 17 and 18). The authors of CUDOS have granted permission for the scale to be used by researchers for use in non-industry related studies, so I could incorporate it into my research. The piloting process resulted in a minor change: one item was shortened from “My appetite was poor and I didn’t feel like eating” to “My appetite was poor.” The full list of CUDOS items is in Appendix F. The total symptom severity score is the aggregated scores for the first 16 items. Responses to items 17 and 18 are not included in the total symptom severity score. Thus, the possible range of scores is 0 to 64. The severity cut-off scores are in Table 3.

Table 3

Cut-off Scores for Clinically Useful Depression Outcome Scale (CUDOS)

Depression Severity	CUDOS Score Range
Not depressed	0-10
Minimal Depression	11-20
Mild Depression	21-30
Moderate Depression	31-45
Severe Depression	>45

Piloting instruments. The patient instruments were briefly piloted with two individuals who had experienced depression and two postgraduate researchers who were familiar with designing questionnaires for research. The GP survey was designed with a completion time of about 10 to 15 minutes and piloted with two GPs who had more than 20 years of experience each. Based on their feedback, I rephrased statements that seemed vague, added examples to improve clarity, and removed repetitive statements where possible. The piloting process resulted in several dimensions of the modified depression CONNECT instrument having only single items. For the sake of consistency, all subsequent analysis examined the 16 items individually instead of having some of them combined as subscales.

Interview format

Patient interviews. The interviews were semi-structured with the aim of asking participants to elaborate on why their survey responses differed between both time periods. Changes between patients' answers to Patient Survey 1 and Patient Survey 2 were noted and 10 to 12 of the more pronounced changes were selected for discussion. For example, if a participant's response to a statement changed from strongly disagree in Patient Survey 1 to strongly agree in Patient Survey 2. I aimed for an average interview length of about 30 to 45 minutes and found this number of questions to be suitable.

GP interviews. The semi-structured doctor interviews aimed to understand to what extent EMs are discussed in primary care settings and why they are important. Topics covered by the interview guideline included: how much importance GPs accorded to knowing patient EMs and the process that GPs use to explore their patients' EM. The interview also asked whether differences between patient and GP EMs cause problems; and how accurate knowledge of patient EMs could play a part in alleviating patient self-stigma, improving GP-patient communication, and optimising recovery.

Procedure

Recruiting participants with depression. Participants were aged 18 to 65 and had consulted a GP for depression or depressive symptoms in the past three months. The minimum age limit of 18 was chosen to simplify the recruitment process as it precludes the need for informed written consent from parents or caregivers of participants.

The focus on primary care patients stems from literature that indicates that the majority of depression patients seek treatment with GPs. The rationale for the three-month time frame came from studies showing that the experience of depression is episodic, with estimates of episode length ranging between three and six months (Eaton et al., 2008; Spijker et al., 2002). The three-month exclusion criterion thus ensures that survey participants had a current on going or relatively recent past experience of depression. Individuals with possible pregnancy-related or bipolar-related depression were excluded as these depression subtypes were not the focus of this thesis.

I recruited participants through a number of methods: posting flyers on bulletin boards (sample flyer in Appendix G) in community spaces (e.g., libraries and shopping centres); distributing invitations through health organisations (e.g., Metro North Brisbane Medicare Local) and mental health support groups (e.g., Group 61 and Stepping Stones Clubhouse); disseminating flyers to general practice clinics and pharmacies. In addition, social media platforms such as Facebook were used. Through a web link provided on the recruitment flyer or social media post, participants could go online to answer the survey questions for each study. Alternatively, they could request a hard copy of the survey by texting my phone number, which was also on the recruitment flyer. If a hard copy was requested, I also sent a stamped addressed envelope for returning the completed survey. Participants could choose to receive a small token of appreciation for completing the first survey. The token was a silver sea turtle charm, with an accompanying note explaining that the turtle is linked to healing and good health in ancient cultures. After six months, participants were contacted for Patient Survey 2. The incentive for completing the second survey was a chance to win five lucky draw prizes of 90-minute mobile spa vouchers. I also sent reminder emails to improve the response rate; I sent up to three emails about Patient Survey 2 to participants who had done Survey 1. In addition to the patient surveys, I invited selected participants who had completed both patient surveys for one-on-one interviews. Of the 87 (67%, $n = 129$) who had indicated on the questionnaires that they were willing to be interviewed I selected 10 participants to reflect the diversity of gender and age bracket. I chose four males and six females, and at least one interviewee from each of the age ranges given in the survey (18-24, 25-39, 40-55, 56-65). The interviews were conducted between August 2015 and January 2016. One participant's interview was later excluded from analysis as she possibly had pregnancy-related depression. Participant information sheets for both patient questionnaires and interviews are in Appendices H1 and H2.

Recruiting GP participants. I invited all participants of Patient Survey 1 to nominate their GP for participation in the study so that I could obtain the GP's perspective of their patient's depression (e.g., what do they think caused their patient's condition?). In order to ensure that participants did not feel pressured to involve their GPs, I listed this optional question at the end of the survey. If participants agreed, they simply had to provide the name of their GP and practice address as well as their own name as per patient records at the completion of the survey. GPs who were nominated were invited to participate using e-mail invitations (see Appendix I), faxes, mailed hard copies of the GP survey, and where possible, in-person visits to the clinics (only within Brisbane). All correspondence with GPs, including the GP participant information sheet (see Appendix J) emphasised that GPs would not be asked to break patient confidentiality. This information helped to assuage some GPs' concern that the survey would ask for details about their patients.

A minimum number of 42 GPs was required for the study. I derived this minimum sample size from calculations using free-to-use software G*Power, with the expected effect size of .30 (medium according to Cohen, 1988) and power of at least .80. I could not find any depression EM studies that reported effect sizes so I based my estimate on a study on autism (effect sizes ranged from .18 to .32) by Carter (2008) and a study on schizophrenia (Ghane, Kolk, & Emmelkamp, 2010), which reported effect sizes of .56 to .90. The expected effect size of .30 used in my minimum sample size calculation was approximately the average of the lowest effect size reported in both studies. After the first six months of recruitment around 20 to 25 per cent of participants had nominated their GPs to take part in the study. I therefore decided on a corresponding target of at least 200 patients with depression. This number of patients would theoretically provide 50 GP contacts if the proportion of patients who consented to GP participation remained consistent.

I asked those who had declined to provide GP contacts for the rationale behind their decision; some said they did not want to trouble their GPs as they felt their doctors had busy schedules. However, even when patients gave their GP contacts, many doctors who were invited to join the study declined to participate. Numerous reminders via e-mail and visits in-person to clinics within Brisbane did not improve the response rate. Between March 2014 and October 2014 only eight GPs took the GP survey although invitations were sent out to 22 GPs. Therefore, in November 2014, a second strategy to recruit GPs was implemented that involved contacting GPs directly to ask them to nominate individuals for Patient Survey 1. This was to increase the proportion of patients who consented to their GP participating – the rationale being that participants of the patient survey would be more amenable to involve their GPs if it was their GP who personally told them about the study. This in turn increased the number of GPs successfully recruited into the study. Organisations such as the General Practice Training Queensland and Metro North Brisbane Medicare Local assisted me to reach out to GPs. GPs could either email their patients (this was done with the help of reception staff or the practice manager) with the web link for Patient Survey 1, or if they did not wish to use the link, they were provided with hard copy versions of the survey (with stamped addressed envelope for return).

In addition to the GP questionnaire survey, I interviewed 10 GPs on depression management in primary care. These were newly recruited and not drawn from the GPs who completed questionnaires. I recruited them through word of mouth and by disseminating a flyer about the interview via e-mail (see Appendix K). The GP interviews were conducted between April 2015 and January 2016. GP interview participant information sheet is in Appendix L. Interview consent form for both patients and GPs is in Appendix M.

Analysis

Data screening. Data from the quantitative survey was analysed using SPSS version 20. Using SPSS Frequencies, all variables were found to have values within the expected range. Participants who were either pregnant or had given birth in the past 12 months before completing either patient surveys were excluded, along with participants who mentioned having bipolar disorder. Participants who stated they did not see a GP or saw a health-care provider other than a GP were also excluded. Finally, for any participant who had taken the Patient Survey 1 twice, only their first set of data was retained for analysis.

Questionnaire data. Descriptive statistics were used to summarise participant characteristics and agreement with statements about depression. This answered RQ1a (What are depression EMs of primary care patients?). Cross-tabulation (with the chi-square test of independence) and correlation analysis was used to determine if participants who agreed with biomedical causation also agreed with psychosocial causation.

Since survey participants were recruited on the basis of their depression experience, their responses to questions about depression were not expected to be normally distributed. This is further reinforced by the use of a Likert scale. Therefore, non-parametric methods were used to compare how groups of participants responded to the EM survey items. The participants were grouped according to the following demographic characteristics: age, gender, education, ethnicity, and income.

As group comparisons are only meaningful if based on stable demographic information, cross-tabulations of Time 1 and Time 2 responses for the five key characteristics were visually checked to ensure that participants reported mostly the same information. This was followed by either Mann-Whitney's test for characteristics with two levels (e.g., gender) or Kruskal-Wallis (K-W) test (for characteristics with more than two levels (e.g., age). These two non-parametric tests evaluate whether the population medians on a dependent variable (DV) are the same across all levels of a factor (Green & Salkind, 2008) and are advisable when scores on the DV are ordinal (e.g., Likert scale responses) (Sedgwick, 2015). Post hoc procedures for significant K-W tests consisted of the Dunn's test (Dunn, 1964) with Bonferroni correction to evaluate pairwise differences while controlling for Type I error across tests. Effect-size r were calculated by dividing z by the square root of N (Pallant, 2013), to show the proportion of variability in the dependent variable ranks accounted for by the independent variable. Cohen's (1988) guidelines consider effect sizes of .10, .30, and .50 to be small, medium, and large, respectively. Participants were also grouped and compared according to these health characteristics: the presence of depression diagnosis, the length of depression experience, psychiatric co-morbidity, current physical health, and impact of physical health on quality of life. Such information was only collected in Patient Survey 1 – this was to keep Patient Survey 2 short to reduce the risk of survey fatigue.

To answer RQ1b (Do depression EMs of primary care patients change over time?), a Wilcoxon signed-rank test (often described as the non-parametric equivalent of the paired t test) was conducted with responses to the modified CONNECT depression instrument in Time 1 and Time 2. One key assumption of the Wilcoxon signed-rank test is the symmetrical distribution of differences around the median, which means skewness (a measure of non-symmetry) should be limited to values between -2 and +2 (Field, 2009). Group comparisons according to the five demographic variables listed earlier were performed for groups with at least 30 participants.

To answer RQ2 (Does high endorsement of biomedical causation for depression correlate with low patient self-stigma?) correlations between endorsement of biomedical causation for depression and self-stigma subscales from the modified ISMI-depression instrument were analysed. I calculated the Spearman's rho correlation coefficient (r_s), a statistic that is often employed in health and medical research because it is suited to the ordinal or ranked data (Pallant, 2013). Pallant also noted that the use of Spearman's rho is increasing in psychology literature as researchers become more cognisant of possible issues arising from assuming that ordinal level ratings, such as Likert scales, are similar to interval level scaling. Correlations were calculated for the entire sample and also for subsets of participants grouped by their demographic and health characteristics. Correlations over .30 that reflect medium effect size according to Cohen (1988) were further analysed using non-parametric partial correlations to explore the relationship between self-stigma and belief in the biomedical causation of depression while controlling for the participant characteristic.

Correlations between the participant scores on the modified CONNECT depression questionnaire and the communication effectiveness instrument were used to answer RQ3 (Do patients with higher levels of agreement to certain aspects of patient EMs perceive greater GP-patient communication effectiveness than patients with lower levels of agreement?).

To answer RQ4 (How aware are GPs of their patient's depression EM, how concordant are GP EMs with patient EMs, and how does awareness and/or concordance correlate to depression outcomes?), GP-patient pairs were first grouped into aware/unaware and concordant/discordant. Pairs were considered aware if GP's perception of patient EM matched the patient's EM and concordant if both GP and patient had similar levels of agreement to the items in their respective EM questionnaires. Depression outcome was reported as a change in CUDOS scores, that is, Patient Survey 1 CUDOS score minus Patient Survey 2 CUDOS score. As noted in Chapter 2, depression symptoms can get better over time (Mueller et al., 1999). Therefore, it was necessary to check for statistically significant ($p < .05$) correlations between severity of Time 2 depression symptoms and the time between Survey 1 and Survey 2, so that adjustments could be made accordingly with hierarchical multiple regression or similar statistical techniques.

To compare effect of awareness and concordance on depression outcomes simultaneously, GP-patient pairs were divided into four subgroups as below:

(1) Aware-concordant, where GP perceives patient's EM correctly and GP's EM matches patient's EM

(2) Aware-discordant, where GP perceives patient's EM correctly but GP's EM is different from patient's EM

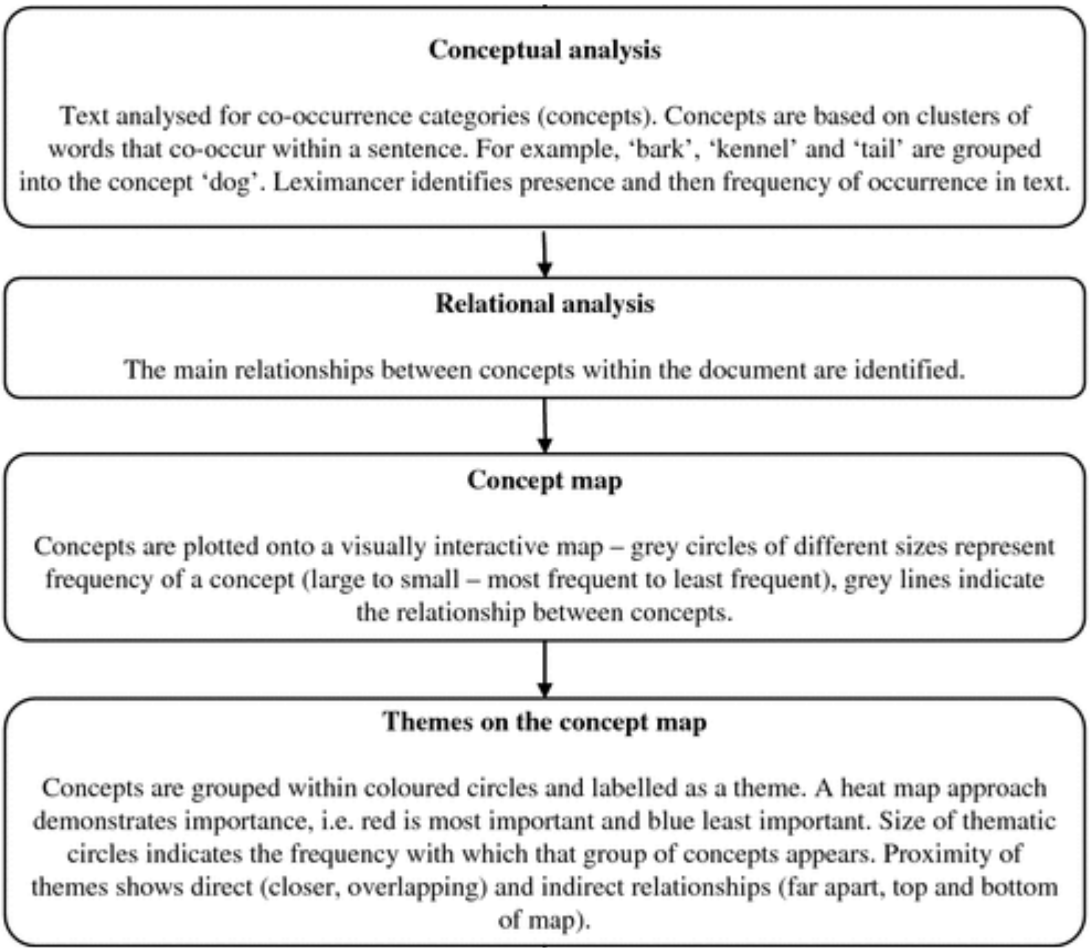
(3) Unaware-concordant, where GP perceives patient's EM incorrectly but GP's EM matches patient's EM

(4) Unaware-discordant, where GP perceives patient's EM incorrectly and GP's EM is different from patient's EM

RQ4 generated three hypotheses: Hypothesis 1 predicted better depression recovery (i.e., greater decrease in symptom severity between Time 1 and Time 2) in GP-patient pairs who were more aware compared to those who are less aware. Hypothesis 2 predicted better depression outcomes for GP-patient pairs who were more concordant compared to those who were less concordant. H1 and H2 were tests of difference in the dependent variable (depression outcome) based on different levels of a single independent variable, that is, awareness or concordance. For tests of difference using both variables of awareness and concordance, GP-patient pairs were first categorised into aware-concordant, aware-discordant, unaware-concordant, and unaware-discordant groups, and then compared with each other for differences in depression outcomes. Hypothesis 3 predicted better outcomes for aware-concordant pairs than the other pairs.

Interview data. Both patient and GP interviews were transcribed verbatim. Patient interview transcripts were analysed manually using a grounded approach (Corbin & Strauss, 2008). The grounded approach is more suited because the topics discussed in the patient interviews differed from one interview to the other, depending on how the participants answered in Patient Survey 1 and 2. This is consistent with the more personal and flexible approach of constructivism (as explained in Chapter 6).

On the other hand, the GP interview transcripts were analysed in a more objectivist and quantitative manner using Leximancer 4.0. Leximancer is a specialised software that analyses textual documents and displays the extracted information visually in a map of concepts and themes (Leximancer, 2011). A concept is defined in Leximancer as a group of words that are generally associated in the text. Concepts that frequently occur within the same portions of text will appear close on the map, and a group of concepts will make up a theme. Leximancer enables the researcher to quantify and show the thematic/conceptual and relational structure of any collection of text, as explained in Figure 2. Moreover, the interactive concept map allows the researcher to explore concepts and how they relate to each other, and to perform directed searches to examine the original textual excerpts linked to the concepts. For example, it is possible to encapsulate implicit references, that is, a concept can be accurately inferred without the need for keywords or phrases if adequate related terms are found within the text portion. Watson, Jones, and Cretchley (2014) noted that this process is better than automatic keyword searching because it is akin to having a human analyst reading and coding the text. The GPs in this interview study were asked similar questions so their responses could be compared with each other. Leximancer content analysis showed which concepts were present and how they related to each other. Further analysis details will be explained together with the interview study results for ease of reference (see Chapter 12).



Conceptual analysis

Text analysed for co-occurrence categories (concepts). Concepts are based on clusters of words that co-occur within a sentence. For example, 'bark', 'kennel' and 'tail' are grouped into the concept 'dog'. Leximancer identifies presence and then frequency of occurrence in text.

Relational analysis

The main relationships between concepts within the document are identified.

Concept map

Concepts are plotted onto a visually interactive map – grey circles of different sizes represent frequency of a concept (large to small – most frequent to least frequent), grey lines indicate the relationship between concepts.

Themes on the concept map

Concepts are grouped within coloured circles and labelled as a theme. A heat map approach demonstrates importance, i.e. red is most important and blue least important. Size of thematic circles indicates the frequency with which that group of concepts appears. Proximity of themes shows direct (closer, overlapping) and indirect relationships (far apart, top and bottom of map).

Figure 2. The process of Leximancer analysis resulting in a concept map.

Adapted from "Written plans: An overlooked mechanism to develop recovery-oriented primary care for depression?" by Palmer et al. (2014) with permission from CSIRO Publishing.

This chapter concludes the Methods section of my thesis and the Results section follows. In the interest of continuity, comprehension and flow, the next four chapters (Chapter 8 to 11) address the four RQs in turn. Each chapter will begin with a short recap of the RQ and procedure before reporting the quantitative results. Discussion of quantitative results in these chapters will include interview quotes that provide additional details to illustrate the questionnaire findings. The integration of qualitative results and discussion within the same section is a common feature of qualitative research papers, so this combined structure will also be in Chapter 12, which contains findings from the GP interview study.

Chapter 8: Depression Explanatory Models of Primary Care Patients

In this chapter I address RQ1 (What are depression EMs of primary care patients and do these change over time?) by presenting quantitative results from the modified depression CONNECT instrument in both Patient Survey 1 and 2. The discussion section that follows contains data and quotes from patient interviews (as described in Chapter 7) to expand on survey results.

The previous chapter showed that the participants for both patient surveys were mostly females, aged between 25 and 39 years, born in Australia. The majority were highly educated and employed and identified as being White Europeans/Caucasians. Approximately a third of participants were from the middle-income group, while another third were from the low-income group. Most of the participants had been formally diagnosed with depression, and the majority had experienced depression for more than five years, with 36% having it for more than 10 years. Less than a fifth had co-morbid psychiatric conditions. Participants were mostly in good or fair physical health, with two-thirds reporting slight or moderate impact of physical health on their quality of life.

Cross-sectional Results (RQ1a)

The first part of RQ1 was answered using descriptive statistics. Table 4 shows the frequency distribution of responses from patients at Time 1. Patients' level of agreement to different items on the modified depression CONNECT instrument varied depending on demographic and health characteristics of participants.

Table 4

Participant Responses to Modified Depression CONNECT Items (Patient Version) at Time 1 (n = 237 or 238)

Statements listed by respective dimensions (order of items corresponds with survey)	Number (%)					
	Strongly disagree	Moderately disagree	Slightly disagree	Slightly agree	Moderately agree	Strongly agree
1. Causation: a. Depression is caused by biomedical factors	22 (9.2)	20 (8.4)	9 (3.8)	45 (18.9)	80 (33.6)	62 (26.1)
1b. Depression is caused by psychosocial factors	33 (13.9)	24 (10.1)	11 (4.6)	46 (19.4)	54 (22.8)	69 (29.1)
2. Internal locus of control: Patient's choices led to depression	49 (20.6)	39 (16.4)	34 (14.3)	59 (24.8)	33 (13.9)	24 (10.1)
3. Sense of agency: Right actions will reduce symptoms	3 (1.3)	9 (3.8)	15 (6.3)	56 (23.6)	94 (39.7)	60 (25.3)
4. Belief in non-medical therapies: a. Complementary medicine	41 (17.2)	53 (22.3)	43 (18.1)	68 (28.6)	24 (10.1)	9 (3.8)
4b. Belief in complementary activities	7 (2.9)	13 (5.5)	16 (6.7)	49 (20.6)	87 (36.6)	66 (27.7)
4c. Belief in non-doctor remedies	11 (4.6)	18 (7.6)	25 (10.5)	90 (37.8)	61 (25.6)	33 (13.9)
4d. Belief in alternative therapy providers	6 (2.5)	9 (3.8)	9 (3.8)	54 (22.7)	85 (35.7)	75 (31.5)

5b. Depression currently plays a major role in life	4 (1.7)	12 (5.1)	13 (5.5)	44 (18.6)	77 (32.5)	87 (36.7)
5c. Depression will change the future	9 (3.8)	11 (4.6)	9 (3.8)	58 (24.4)	81 (34.0)	70 (29.4)
5d. Positive outcomes can arise from depression	13 (5.5)	9 (3.8)	13 (5.5)	59 (24.8)	85 (35.7)	59 (24.8)
6. Antidepressant attitudes: a. Antidepressants are necessary	30 (12.7)	28 (11.8)	20 (8.4)	32 (13.5)	59 (24.9)	68 (28.7)
6b. Antidepressants cause more harm than good	40 (16.8)	55 (23.1)	39 (16.4)	43 (18.1)	37 (15.5)	24 (10.1)
6c. Some doctors are too pro-antidepressants	12 (5.1)	36 (15.2)	33 (13.9)	70 (29.5)	56 (23.6)	30 (12.7)
7. Patient-doctor collaboration key to recovery	6 (2.5)	4 (1.7)	5 (2.1)	32 (13.4)	90 (37.8)	101 (42.4)

Responses across entire sample. For the statement on biomedical causation of depression, those who moderately agreed formed the largest proportion of respondents (33.6%). For the statement on psychosocial causation, those who strongly agreed formed the largest proportion (29.1%). Table 5 shows the crosstabs of participants who strongly or moderately endorsed either the biomedical causation and psychosocial causation for depression, or both. A chi-square test for independence with Yates Continuity Correction indicated no significant association between belief in biomedical causation and belief in psychosocial causation of depression, $\chi2$ (1, $N = 237$) = .002, $p = .96$, *phi* = -.014 (negligible effect size). In addition, there was a statistically significant but weak negative correlation between belief in biomedical causation and belief in psychosocial causation, $r_s = -.23$, $p = .0005$).

Table 5

Cross-tabulation of Participant Responses to Items on Depression Causation (n = 132)

		Biomedical causation of depression		
		Strongly/Moderately Disagree	Strongly/Moderately Agree	Total
Psychosocial causation of depression	Strongly/Moderately Disagree	12	37	49
	Strongly/Moderately Agree	23	60	83
	Total	35	97	132

For the statement on depression arising from personal choices (internal locus of control), those who slightly agreed formed the largest proportion of respondents (24.8%). For the statement that reflected participants having a sense of agency to improve their condition, those who moderately agreed formed the largest proportion of respondents (39.7%). For the statements on efficacy of non-medical or alternative therapies, the largest proportions ranged between 28.6% and 37.8%, and these were participants who either slightly agreed or moderately agreed with the statements. For the statements on significance of depression, the majority of participants believed depression changes their priorities, plays a major role, affects the future, and could have a positive outcome. The proportion of those who strongly or moderately agreed with these statements ranged from 60.5% to 75.7%.

For the statement on antidepressants being necessary, those who strongly agreed formed the largest proportion of respondents (28.7%). For the statement on antidepressants causing more harm than good, those who moderately disagreed formed the largest proportion of respondents (23.1%). For the statement that some doctors prescribe antidepressants too quickly, those who slightly agreed formed the largest proportion of respondents (29.5%). Finally, for the statement on the importance of patient-doctor cooperation, those who strongly agreed formed the largest proportion of respondents (42.4%).

Differences between groups. Participants were grouped according to demographic and health characteristics at Time 1 (as presented in Tables 1 and 2) and tested for significant differences in their responses to the modified depression CONNECT items. This increases understanding of which participant characteristics could have an impact on level of agreement to the items. For characteristics with two levels (e.g., gender), the Mann-Whitney test was used, and for characteristics with more than two levels (e.g., age), the Kruskal-Wallis test with follow-up procedure was used.

Gender. Agreement to the items did not vary significantly between female and males (see Table 6). There was a trend towards significance for the statements on psychosocial causation of depression and belief in complementary activities.

Table 6

Difference in EM Beliefs According to Participant Gender

Statement	Mean rank$_{\text{Female}}$	Mean rank$_{\text{Male}}$	*p*	Effect-size *r*
Psychosocial causation	119	100	.056	-.12
Belief in complementary activities	110	127	.089	-.11

Age. Participant age was a factor in the level of agreement for several items, that is, belief that depression can have positive outcomes, χ^2 (3, N = 228) = 9.80, p = .020; the belief in the necessity of antidepressants, χ^2 (3, N = 227) = 8.52, p = .036; and the belief in the importance of patient-doctor cooperation, χ^2 (3, N = 228) = 8.02, p = .046. Follow-up tests were conducted with the Dunn-Bonferroni method and the *p*-values reported in Table 7 have incorporated the adjustments.

Table 7

Difference in EM Beliefs According to Participant Age (Pairwise Comparisons)

Statement	Mean rank by age				p_{adj}	Effect-size *r*
	18-25	26-39	40-54	55-65		
Depression can have positive outcomes	49.3			29.5	.026	-.29
			31.95	19.07	.036	-.36
Patient-doctor cooperation	53.0		70.1		.030	-.26

Positive outcomes from depression. Participants aged 18-25 were more optimistic than those aged 55-65. Participants aged 40-54 were also more optimistic than those aged 55-65. Effect sizes were medium.

Necessity of antidepressants. Participants' endorsement of antidepressants did not differ significantly by age after incorporating the Bonferroni adjustments.

Patient-doctor cooperation. Participants aged 40-54 believed more strongly in cooperating with their doctor to aid recovery than participants aged 18-24.

Culture/Ethnicity. The number of Asian participants and White Caucasian/European participants was sufficient for a comparison to be made (each group had at least 30 participants) for responses to the modified CONNECT instrument items. White Caucasians/Europeans significantly endorsed the statement on biomedical causation of depression more than Asians. For the statement on antidepressants being harmful, the level of agreement among Asians was significantly higher than for White Caucasians/Europeans. Effect sizes for both results were low-moderate (see Table 8).

Table 8

Difference in EM Beliefs According to Participant Culture/Ethnicity

Statement	Mean rank$_{\text{Asian}}$	Mean rank$_{\text{Caucasian}}$	*p*	Effect-size *r*
Biomedical causation	78.1	106	.007	-.19
Antidepressants harmful	122	95.7	.011	-.18

Education. Participant endorsement of the belief that complementary activities help to alleviate symptoms differed significantly depending on the participant's level of schooling, χ^2 (2, N = 220) = 7.35, p = .025. Pairwise comparisons found that participants with at least a bachelor's degree were significantly more convinced that complementary activities are beneficial (*Mdn* = 5, mean rank = 98.2) than participants with certificates/diplomas/advanced diplomas (*Mdn* = 4, mean rank = 75.5), $U = 2394.00$, $p_{\text{adj}} = .025$, effect-size $r = -.19$.

Household income. A Time 1 x Time 2 cross-tabulation of income responses showed differences in about 25% of the cases so income data was not considered for group comparisons.

Depression diagnosis. Participants who reported a depression diagnosis differed significantly in their agreement with eight modified CONNECT depression statements compared to participants with no depression diagnosis. These are listed in Table 9.

Table 9

Differences in Endorsement of Modified Depression CONNECT Items Between Diagnosed and Undiagnosed Participants

Statement	Mean rank$_{Diagnosed}$	Mean rank$_{Undiagnosed}$	*p*	Effect-size *r*
Biomedical causation	115	94.3	.032	-.14
Belief in complementary activities	106	125	.046	-.13
Belief in non-doctor remedies	104	132	.004	-.19
Depression plays a major role	115	94.6	.033	-.14
Antidepressants are necessary	118	84.1	.001	-.23
Antidepressants cause more harm than good	102	138	< .001	-.24
Some doctors are too pro-antidepressants	105	127	.022	-.16
Patient-doctor cooperation key to recovery	116	93	.017	-.16

Length of depression experience. Participants' length of depression experience was a factor in the level of endorsement for the biomedical causation belief, χ^2 (3, $N = 214$) = 11.1, $p = .011$, and the belief that patient choices led to depression (internal locus of control), χ^2 (3, $N = 214$) = 10.9, $p = .012$. Results of follow-up tests were as follows (see Table 10).

Table 10

Difference in EM Beliefs According to Length of Depression Experience (Pairwise Comparisons)

Statement	Mean rank by depression length			p_{adj}	Effect-size *r*
	≤ 1yr	> 5 and ≤ 10 years	> 10yrs		
Biomedical causation	45.4		65.2	.011	-.29
Patient choices lead to depression	71.9		51.7	.019	-.29
	54.0	38.0		.018	-.31

Biomedical causation of depression. Agreement levels of participants who had depression for over 10 years were significantly higher than participants with up to a year of depression experience. Effect size was moderate.

Patient choices led to depression (internal locus of control). Agreement levels of participants who had depression for over 10 years were significantly lower than participants with up to a year of depression experience. Similarly, agreement levels of participants who had depression for between five and 10 years were significantly lower than participants with up to a year of depression experience. Effect sizes were moderate.

Psychiatric co-morbidity. Extent of EM beliefs differed depending on presence of specified psychiatric condition besides depression (see Table 11). Participants who were co-morbid endorsed the statement on patient choices leading to depression significantly less than participants who were not. Co-morbid participants showed significantly greater endorsement for the item on patient sense of agency than participants without co-morbidity. Co-morbid participants significantly endorsed the statement on belief in alternative providers more than participants without co-morbidity. Effect sizes were all low-moderate.

Table 11

Difference in EM Beliefs According to Presence of Specified Psychiatric Comorbidity

Statement	Mean rank$_{\text{Co-morbid}}$	Mean rank$_{\text{Not co-morbid}}$	p	Effect-size r
Patient choices led to depression (internal locus of control)	96.0	124.7	.011	-.16
Patient sense of agency	137	115	.049	-.13
Belief in alternative providers	139	115	.028	-.14

Current physical health. Participant endorsement of three modified depression CONNECT items differed significantly depending on whether the participant reported being in poor, fair, or good health physically. The statements pertained to the psychosocial causation of depression, χ^2 (2, N = 218) = 15.4, p < .001, belief in complementary activities, χ^2 (2, N = 219) = 12.4, p = .002, and depression playing a major role, χ^2 (2, N = 219) = 10.6, p = .005. Follow-up test results are in Table 12.

Table 12

Difference in EM Beliefs According to Current Physical Health (Pairwise Comparisons)

Statement	Mean rank by physical health			p_{adj}	Effect-size r
	Good	Fair	Poor		
Psychosocial causation	46.5		64.3	.008	-.29
	68.6	97.3		.001	-.28
Belief in complementary activities	62.6		43.2	.002	-.32
		84.8	63.1	.018	-.23
Depression plays a major role	47.5		64.3	.011	-.28
		71.9	94.8	.008	-.24

Psychosocial causation of depression. Agreement levels of participants in good health were significantly lower than participants in poor health. Similarly, agreement levels of participants in good health were significantly lower than levels of fairly healthy participants. Effect sizes were moderate.

Belief in complementary activities. Agreement levels of participants in good health were significantly higher than participants in poor health, with moderate effect size. Agreement levels of fairly healthy participants were also significantly higher than participants in poor health, with low-moderate effect size.

Depression plays a major role. Agreement levels of participants in good health were significantly lower than participants in poor health. Similarly, agreement levels of fairly healthy participant were significantly lower than participants in poor health. Effect sizes were low-moderate

Impact on quality of life. Participant endorsement of three modified depression CONNECT items differed significantly depending on the extent to which physical health impacted on the participant's quality of life. The statements pertained to psychosocial causation of depression, χ^2 (3, N = 217) = 11.6, p = .009; belief that patient choices led to depression (internal locus of control), χ^2 (3, N = 218) = 8.18, p = .042; and depression playing a major role, χ^2 (3, N = 218) = 12.2, p = .007. Follow-up test results are in Table 13.

Table 13

Difference in EM Beliefs According to Health Impact on Quality Of Life (Pairwise Comparisons)

Statement	Mean rank by health impact on quality of life				p_{adj}	Effect-size r
	None	Slight	Moderate	Severe		
Psychosocial causation	46.2		65.9		.005	-.30
Patient choices led to depression (internal locus of control)	48.0	64.7			.043	-.25
Depression plays a major role	34.2			49.1	.010	-.34
		45.8		64.7	.014	-.31

Psychosocial causation of depression. Agreement levels of participants who felt their quality of life was moderately impacted by their physical health were significantly higher than participants who were not impacted at all, with moderate effect size.

Patient choices led to depression (internal locus of control). Agreement levels of participants who felt their quality of life was slightly impacted by their physical health were significantly higher than participants who were not impacted at all, with low-moderate effect size.

Depression plays a major role. Agreement levels of participants whose quality of life was severely impacted by their physical health were significantly higher than participants whose quality of life was not at all impacted by their physical health. Agreement levels of the severely impacted participants were also significantly higher compared to participants whose quality of life was slightly impacted by their physical health. Both effect sizes were moderate.

Longitudinal Results (RQ1b)

RQ1b was addressed using a Wilcoxon signed-rank test of responses from Patient Survey 1 and Patient Survey 2. The assumption check for the Wilcoxon signed-rank test determined that distributions of Time 2 minus Time 1 scores for all 16 items were adequately symmetrical, with skewness values ranging from -.60 to .63. The results of the signed-rank test are in Table 14. The table also shows the Time 1 and Time 2 mean scores of participant responses to the individual statements as this better portrays the magnitude and direction of change compared to listing mean ranks.

Table 14

Comparing Time 1 and Time 2 Scores from Modified Depression CONNECT Instrument – Patient Version (n = 129)

Statement (in order of descending effect size)	Negative ranks[a]	Positive ranks[b]	*z*	*p*	Effect-size *r*
Depression plays a major role	52	23	-4.17	< .001	–.37
Biomedical causation of depression	45	28	-2.14	.033	–.19
Depression will change the future	47	33	-1.90	.058	–.17
Depression has changed priorities	38	26	-1.83	.068	–.16
Antidepressants are necessary	43	25	-1.41	.16	–.12
Belief in complementary activities	34	43	-1.33[c]	.19	–.12
Right action will reduce symptoms (sense of agency)	44	33	-1.28	.20	–.11
Personal choices led to depression (internal locus of control)	52	36	-1.17	.24	–.10
Belief in complementary medicines	39	49	-1.07[c]	.29	–.094
Psychosocial causation of depression	46	41	-.48	.63	–.042
Belief in non-doctor remedies	36	44	-.44	.66	–.039
Cooperation with doctor key to recovery	32	33	-.42[c]	.68	–.037
Belief in alternative healthcare providers	31	28	-.26	.79	–.023
Depression can have positive outcomes	40	42	-.20	.84	–.018
Some doctors are too pro-antidepressants	40	32	-.13	.90	–.011
Antidepressants: more harm than good	37	33	-.009	.99	–.0008

Note. [a]Number of participants whose Time 2 score was lower than Time 1 score. [b]Number of participants whose Time 2 score was higher than Time 1 score. Standardised test statistic *z* calculated from positive ranks unless indicated otherwise. [c]Based on negative ranks.

Changes across entire sample. The output indicated that Time 2 scores for the statement on the major role of depression were statistically significantly lower than Time 1 scores. Participants were less convinced that depression plays a major role at Time 2, $z = -4.18$, $p = .00002$, with a medium effect size (r = -.37). Similarly, Time 2 scores for the statement on biomedical causation of depression were significantly lower than Time 1 scores. Participants were less convinced at Time 2 that depression was caused by biomedical factors, $z = -.214$, $p = .033$, effect-size $r = -.19$. It is worth noting that belief about psychosocial causation of depression did not show significant changes between Time 1 and Time 2 (see Table 14), unlike belief about biomedical causation. The majority of EM beliefs did not change with recovery or longer duration of depression. In particular, the effect sizes were negligible for the statements pertaining to belief in psychosocial causation of depression, efficacy of non-doctor remedies, efficacy of alternative health providers, possible positive outcomes from depression, some doctors being too pro-antidepressants, and antidepressants doing more harm than good.

Changes in and between groups. When the sample was split according to demographic variables established from Time 1 (i.e., gender, age, culture/ethnicity, and education level), other significant changes with generally moderate effect sizes were found in the item responses using the Wilcoxon signed rank test. Longitudinal changes were only reported for groups with $n > 30$ participants. Details are in Table 15.

Table 15

Comparing Time 1 and Time 2 Scores for the Modified Depression CONNECT items – Patient Version by Participant Characteristics

Statement	Participant characteristic	Time 1 versus Time 2 scores	*p*	Effect-size *r*
Biomedical causation	Male	T1 > T2	.027	-.28
Efficacy of complementary activities	Age 25-39	T1< T2	.003	-.29
Depression has changed priorities	Age 40-54	T1 > T2	.043	-.27
Depression plays a major role in my life	Female	T1 > T2	<.001	-.28
	Age 18-24	T1 > T2	.027	-.26
	Age 25-39	T1 > T2	.004	-.28
	Age 40-54	T1 > T2	.037	-.27
	White European/ Caucasian	T1 > T2	.003	-.21
	Has at least Bachelor’s degree	T1 > T2	.00004	-.21
Depression will affect future	Age 40-54	T1 > T2	.026	-.28
Antidepressants are necessary	Age 18-24	T1 > T2	.036	-.25
Antidepressants can be harmful	Male	T1 < T2	.032	-.27

Gender. In female participants, Time 2 scores for the statement that depression plays a major role were statistically significantly lower than Time 1 scores. In male participants, Time 2 scores for the statement on biomedical causation of depression were significantly lower than Time 1 scores, with moderate effect size. Male participants’ Time 2 scores for the statement that antidepressants can be harmful were significantly higher than Time 1 scores.

Between-group comparison of changes in the item responses was possible for gender because $n > 30$ for both male and female groups. A Mann-Whitney test showed a significant change in male endorsement of antidepressants being harmful compared to females, $U = 1167$, $p = .041$, with low-moderate effect-size $r = -.18$. Between Time 1 and Time 2, males became more convinced that antidepressants could cause more harm than good ($Mdn_1 = 2$, $Mdn_2 = 4$), compared to females, whose views were virtually unchanged ($Mdn_1 = 3$, $Mdn_2 = 3$).

Age. Time 2 scores of participants aged 18-24 were statistically significantly lower than their Time 1 scores for two items: depression plays a major role, and antidepressants are necessary.

Time 2 scores of participants aged 25-39 for the statement on depression plays a major role were statistically significantly lower than their Time 1 scores. Time 2 scores for the item on efficacy of complementary activities were significantly higher than Time 1 scores.

For participants aged 40-54, Time 2 scores were statistically significantly lower than Time 1 scores for these items: depression plays a major role; depression changed their priorities; and depression will affect the future.

Between-group comparison of changes in the item responses was possible for age because $n > 30$ for three groups, that is, 18-24, 25-39, and 40-54. A Kruskal-Wallis test compared the changes in endorsement levels between Time 1 and Time 2, using age as the grouping variable. The test was corrected for tied ranks and significant for the belief on the efficacy of complementary activities,
χ^2 (2, $N = 124$) = 6.85, $p = .033$. Follow-up tests found that changes in participant endorsement of the efficacy of complementary activities did not differ significantly by age after incorporating the Bonferroni adjustments.

Culture/Ethnicity. The White European/Caucasian participants who completed both Time 1 and Time 2 surveys comprised the only group with more than 30 participants ($n = 102$). Their Time 2 scores for the statement on depression playing a major role were lower than their Time 1 scores.

Education. The participants with at least a Bachelor's degree who did both surveys comprised the only group large enough ($n = 78$). Their Time 2 scores for the item on the major role of depression were lower than their Time 1 scores.

Discussion

RQ1 asked what are the explanatory models (EMs) adopted by primary care patients and whether these EMs change after approximately six months. This section discusses some possible reasons and implications of my study's findings on the seven dimensions of the modified CONNECT instrument that constitute an individual's EM. As noted earlier, excerpts from patient interviews are included to substantiate the quantitative findings.

Causation. The majority of participants in this study had a strong or moderate belief about whether biomedical and/or psychosocial factors caused their depression. Since previous studies have shown that causation beliefs can influence treatment decisions (Buus et al., 2012; Okello & Neema, 2007), this finding suggests that it is important for doctors to understand what patients believe caused their depression.

Biomedical cause for depression. The finding that White Caucasians/Europeans endorse biomedical causation of depression more than Asians aligns with other studies on differences in attitudes towards mental illness among ethnic groups (e.g., Carpenter-Song et al., 2010; Karasz, 2005). Carpenter-Song et al.'s intensive 18-month ethnographic study of 25 individuals with severe mental illness (including depression) in a US inner city found Euro-Americans having biomedical perspectives about their condition and seeking help from mental health professionals. In contrast, the African-Americans and Latinos in the study focused on non-biomedical interpretations of their problems and were disparaging of mental health services.

The finding that participants with long-term depression believe more strongly in the biomedical causation of their symptoms than participants with up to a year's experience with the condition contradicted the finding that endorsement of biomedical causation decreased between Time 1 and Time 2 across the entire sample. One possible explanation could be that participants with over 10 years of depression would have had repeated exposure to mental health professionals who tend to subscribe more strongly to biomedical EMs, as previously highlighted in Chapter 3 (Andersson et al., 2001; Thomas-Maclean & Stoppard, 2004). As such, their views may have been influenced to be more biomedical. Participant #71 accepted that her depression had biomedical as a default explanation for why her symptoms suddenly stopped after nine years. She has been symptom-free for about 18 months:

> I kind of just stopped being depressed and I can't explain why so that made me feel more like it's, uhm, a chemical or physical brain thing. Yeah it's just, uhm, I can't explain why I was depressed and now I'm not. (participant #71, female, 25-39)

Belief in biomedical causation decreased in males between Time 1 and Time 2 but not in females. Participant #64 explained why he strongly agreed with biomedical causation at Time 1 but changed his response to slightly disagree at Time 2 when he realised that his surroundings played a significant role in triggering his depression.

> If things aren't working out perfectly in your supposed requirements for it, what you imagine it to be, that can set off depression. It doesn't mean it's a chemical reaction, it just means your environment is not working for you, and you've got to change something. Or it's no good taking pills or going to see somebody to fix something which can't be fixed, your environment, you see… but I've got Asperger's, I was only diagnosed in the last so many years… I say that the treatment from others creates a lot of this depression because of their lack of knowledge or understanding or selfishness or don't care, you know people with disabilities get treated especially – I'll give you one example. When I went to Centrelink, I had my wife with me, and she's seen the way I get treated, she reacts, it's disgusting the way

> I get treated by a government department. Funnily enough, when we complained, we get a letter of apology and all that crap but nothing happens. The funny part about it is that she treats me the same way but she is unaware of it. So when you're living in that environment constantly, and it's from all sources, it gets you, it wears you down… me daughter the other day, she went off her nut and abused me, told me to commit suicide, I should have done it years ago, it went on for half an hour. You can't help getting depressed. (participant #64, male, age 55-65)

It is interesting to note that within the same period male participants became more convinced that antidepressants could be harmful compared to females (this finding will be discussed more fully later). These two findings in male participants (decrease in endorsement of biomedical causation and increase in the belief that antidepressants are harmful) are likely related and could have important implications on antidepressant treatment adherence. Future experimental studies could shed more light on the mechanism linking causal beliefs and antidepressant attitudes.

Psychosocial cause for depression. The near significant result for gender differences in extent of endorsement for psychosocial causation is worthy of attention. It could arise from the tendency of women to ruminate more than men, which has been shown in a recent meta-review by Johnson and Whisman (2013). The authors had set out to gather empirical evidence for the response styles theory (RST) (Nolen-Hoeksema, 1987; Nolen-Hoeksema, 1991) which posits that individuals who chronically ruminate are likely to experience more severe depressive symptoms and longer depressive episodes. The RST defines rumination as a repetitive and passive focusing on distress symptoms as well as the possible causes and impact of the symptoms. Consequently, women who ruminate are more likely to find the connection between their circumstances and their depression, which is why they see their condition as being psychosocial. Future research is needed to establish whether rumination does indeed cause women to endorse psychosocial causation for depression more than men.

A further review of literature yielded another possible explanation for the greater endorsement of the psychosocial model among women. It could be because the biomedical model keeps females oppressed, an argument that has been put forth by Neitzke (2016). Neitzke strongly argues that mechanistic biological models sideline the scrutiny of social-structural causes of depression such as gender oppression, thereby allowing such imbalance of power to continue. This argument should be examined in future studies because depression tends to affect women more than men (see Chapter 2).

Another notable finding was how physically healthy participants or those whose physical health had no impact on their quality of life were less convinced about the psychosocial causation of depression than those who were less healthy or whose quality of life was more impacted by their physical health. While it is unclear why this is the case, this finding suggests that GPs may need to explain more clearly the role of psychotherapy on top of pharmacotherapy for depressed individuals who are otherwise physically healthy. This is because studies have shown that combination therapy is more effective than pharmacotherapy and psychotherapy alone (Karyotaki et al., 2016; Schotte et al., 2006). Presumably such healthy, but depressed, patients' acceptance of antidepressants would be similar to patients who were physically unwell on top of their depression since there was no difference in the endorsement of biomedical causation in the two groups. The healthy-depressed patients would not need any convincing to take their medication but may need additional prompting to make an appointment to see a clinical psychologist. This targeted form of psycho-education would benefit such patients and improve likelihood of treatment adherence to combination therapy. Some evidence for this line of reasoning can be found in a meta-analysis by Cuijpers, Van Straten, Van Schaik, and Andersson (2009), which showed that patients referred for psychological treatment by their GPs saw greater improvement than patients recruited through screening. The GPs of those patients would have discussed the referrals and in doing so could have helped the patients understand how psychotherapy benefits them. In addition, as shown earlier in Table 14, patient belief in psychosocial causation tends to stay stable over time. So in healthy-depressed patients, they could remain unconvinced about the psychosocial cause of depression unless their healthcare provider intervenes.

Internal locus of control. Overall, it appears that participants did not believe that their personal choices led to their depression as the percentage who strongly or moderately disagreed with the item (37%) was greater than those who strongly or moderately agreed (24%) at Time 1. When participants were grouped according to length of depression experience, the data showed that individuals who had been depressed for less than a year endorsed the belief of internal locus of control more than those who had been depressed for over five years. The results from the overall longitudinal study corroborate this finding as endorsement in the statement "I believe that choices I made led to my depression" did slightly decrease between Time 1 and Time 2 (although it was not statistically significant). For example, Participant #66, who had depression for 15 years, was convinced that her depression was not within her control:

> I think my depression was out of my hands completely. Cuz [*sic*] my boyfriend and my ex-boyfriend both died two years apart on Christmas Day, so I really don't think, I mean I think I was going to feel depression before one of them died anyway, but I really, really think it was out of my control. I don't think depression was something I chose. I think it came to me…I've friends who got divorced, they were in a loving relationship and then their partner

> left them, and they sort of got depressed. And they had to you know be on medication for their kids and things. But I don't think mine was something I could do or predict. (participant #66, female, age 40-54)

On the other hand, patients who believe that their choices led to their depression could be more susceptible to feeling that they are at fault for their condition. Their healthcare providers and their support network should note this possibility as these parties help such patients learn to manage their depression, especially within the first year.

Interestingly, participants whose quality of life was slightly affected by their physical health agreed more with the statement that their choices led to their depression compared to unaffected participants. It is possible that such participants feel more responsible for how their physical health affects their quality of life because what troubles them could arise from lifestyle choices (e.g., unhealthy eating habits, lack of exercise, smoking, etc.) rather than any acute physical illness. Therefore, the sense of responsibility in such participants, who may think that if they exercised more, they would be fitter and their physical health would impact less on their quality of life, could extend to their depressive symptoms. Further studies would be needed to test these possible explanations so as to understand any links between impaired quality of life and the degree of responsibility one feels for their depression.

Sense of agency. Participants were generally positive that they had the power to decrease their depression symptoms by taking the right actions as 65% showed either moderate or strong agreement with the statement reflecting sense of agency. There was no significant change in responses between Time 1 and Time 2. This is encouraging because previous studies have shown that agency is a key component of hopeful thinking (Snyder, 1995) and that it counters the effects of emotional distress (Hunt, Sweeting, Keoghan, & Platt, 2006). However, one's sense of agency can fluctuate, even if taking action results in tangible change. Participant #66 who lost 30kg after starting to walk for exercise described this:

> I know sometimes I get a bit self-defeating and I go "Aww nothing I do will change", but I know for a fact – it's changed dramatically for me – but I think sometimes if I'm really depressed I just think nothing's gonna change, nothing's gonna change…(participant #66 female, age 40-54)

Another participant shared insight on how her sense of agency needed time to develop:

> I've found that it's something that I had to become aware of. I had to learn that. Maybe many people told me that in the past but I wasn't ready to learn it. And people can only learn something when conditions are right... So I heard it many times I guess and maybe many times people got frustrated with me because I wasn't doing what they advised. But

> then I just think I figured it out. I – the agency is with me. You can't make me better. Music can't make me better, but if I turn it on it can help. So yeah. It took a long time… I think getting rid of a lot of stress was one of the conditions. I think having the right support. I moved houses, to a house that is flooded with natural light so when in the morning I get that natural light. I did that deliberately. I bought the house deliberately for that natural light. Time, I think time, information and finding the right people to give support... (participant #11, female, age 55-65)

Further studies could help identify the factors that help develop a patient's sense of agency would indeed bode well for depression management.

An unusual finding in my study was that participants with psychiatric co-morbidity apparently have a greater sense of agency than participants without such co-morbidity. It contradicts an earlier study by Carson, Katz, and Alegría (2016) on physical co-morbidity in mental health patients, which reported that patients perceived limitations on their sense of agency due to their experience of physical illness. It is possible that psychiatric co-morbidity has a different impact on a patient's sense of agency but further research is needed to confirm this and to understand the reasons behind it.

Belief in non-medical therapies. The percentages of participants who strongly or moderately agreed with the modified depression CONNECT items (as shown in Table 4) were lower for complementary medicines (e.g., vitamins, fish oil, and St John's Wort) compared to complementary activities (e.g., exercise) or non-doctor remedies. Kleinman (2004) argued that it is important for doctors to be aware of and to respond to their patients' self-care or involvement in alternative and complementary therapies because such therapies can affect the biomedical course of treatment.

The following quote from participant #94 reflects a degree of scepticism about complementary medicines as he is convinced about the efficacy of only certain remedies and not others. Although he has not tried any of them, his stance stems from what he thinks would help his condition, which he perceives to be caused by biomedical factors:

> Because I tend to think of my depression as a chemical, largely a chemical problem, I do think that there are medications or vitamins or other things like that, that could improve some of the symptoms of my depression. Some of the alternate treatments I think that were mentioned in that, I don't necessarily believe in, yeah. So the St John's Wort, the fish oil, that sort of thing I tend to be much more sceptical of. (participant #94, male, age 40-54)

In addition, recent meta-analysis studies on the efficacy of 'natural' remedies point to possible reasons for the weak endorsement of these remedies in this study. A meta-review of randomised control trials involving B-vitamins for depression found that short-term consumption for up to three months did not alleviate depressive symptoms, but prolonged consumption for a year reduced the risk of relapse (Ford et al., 2015). In my study, belief in complementary medicines did increase between Time 1 and Time 2. Perhaps some participants were finally experiencing benefit after persisting in taking duration-dependent complementary medicines like B-vitamins. For example, participant #11 (female, age 55-65) started taking niacin (vitamin B3) after hearing about it in a documentary. At the time of the interview, she had been taking it for roughly three months.

> A: ...it was a psychiatrist, psychologist? Somebody in America talking about depression and he said 5000 milligrammes of niacin had improved a number of his patients. He described the improvement and I thought
>
> Q: It couldn't hurt right?
>
> A: Yeah. And suddenly it worked.

Another meta-review showed that intake of fish and dietary n-3 polyunsaturated fatty acids at certain dosages is associated with reduced risk of depression (Grosso et al., 2016). It is possible that participants in this study were unconvinced of the efficacy of non-medical therapies because they were not taking the right dosage. As for St John's Wort, the most recent meta-analysis of clinical studies concluded that it is as efficacious and safe as SSRIs for mild-to-moderate depression (Ng, Venkatanarayan, & Ho, 2017). Considering this, the participants in this study could be unconvinced about the effectiveness of complementary medicines because they are suffering from the more severe forms of depression; as mentioned previously most of the participants have had depression for at least five years. Further evidence for this explanation can be interpreted from an additional finding of this study that participants with a clinical diagnosis of depression were less convinced about the effectiveness of non-doctor remedies (which may include complementary medicines) than undiagnosed participants. The latter possibly had milder forms of depression compared to the former.

Diagnosed participants were also less convinced about the efficacy of complementary activities than undiagnosed participants, possibly for the same reason, that is, their symptoms were more severe, and there was insufficient improvement from engaging in complementary activities like exercise. A 2014 analysis of high-quality studies by Cooney, Dwan, and Mead concluded that exercise results in limited reduction of depression symptoms compared to no treatment, placebo, or active control interventions, such as relaxation or meditation. In a more recent meta-review to evaluate the impact of physical exercise on cognitive symptoms in depressed adults, Brondino et al. (2017) said that no substantial benefit was observed. Endorsement of complementary activities was also lower in participants of this study who reported poor physical health, compared to participants who rated their physical health as fair or good. This could possibly be due to participants with poor physical health being limited in the range of complementary activities they were able to engage in.

Those who tended to endorse complementary activities were males (rather than females) and participants with degrees (rather than those with certificates/diplomas). The gender difference can be explained by results of an earlier study (Mahalik & Rochlen, 2006), which showed that males are likely to cope with depression in typically masculine ways. For example, exercise/workout was indicated as a “somewhat likely to very likely” response by the 153 mostly white and heterosexual undergraduate males in their study. In addition, the authors found that men who subscribed to the masculine norm of winning were more likely to consider exercising/working than men who conformed less to the norm. As for the positive association between education level and degree of endorsement in complementary activities, one possible reason could be that participants with degrees are more exposed to a range of activities because universities have many clubs and societies compared to TAFE (technical and further education) institutions which award certificates/diplomas.

To summarise, various factors are associated with endorsement of alternative therapies and such information could be useful for depression management. Further studies and dissemination of such findings could help depressed individuals and their doctors choose from various possible interventions. Since depression is a multifaceted problem, a multi-pronged approach could yield the best results. As noted in Chapter 2, combining therapies to treat depression is more effective than a one-size-fits-all approach that is heavily reliant on antidepressants.

Significance of depression. Overall, participants believed strongly that depression has changed their priorities, plays major role in their lives, and will change the future, but the endorsement of these beliefs tended to decrease over time. These findings likely reflect that participants have either experienced some improvement in their symptoms, or they have learnt to cope better with the condition. Participant #63 in my study explained that she moderately agreed with the item on depression changing her priorities at Time 1 but changed it to strongly disagree because she felt she had recovered:

> I check in regularly with the doctors and he goes “You are definitely not depressed”…You definitely, you are definitely more selfish when you have depression because you are not thinking, you are not thinking right yourself. So you are not going to be in the right frame of mind to help other people when you can’t even help yourself. (#63, female, age 18-24)

Another participant felt depression had changed his priorities in a good way, of helping him to pace himself. He said:

> I guess one of my main life goals at the minute is just trying to get healthy. At the same time not trying to rush through it. I've had a few incidents this year of, I guess, pushing myself too hard and then flopping into a hole. (#84, male, age 25-39)

The only item related to significance of depression that did not follow the longitudinal trend described above was the belief that depression can have positive outcomes. While the majority either strongly or moderately agreed with this item at Time 1, there was no change in the level of endorsement at Time 2. This is an encouraging finding because individuals who believe that depression can have positive outcomes are more likely to experience those positive outcomes such as increased clarity in priorities and greater empathy. Participant #49 (male, 18-24 years old) observed that he was able to offer much more support to one of his students who had experienced the death of a parent after he had faced depression. He believed that negative emotions have a role to play, and spoke about being able to relate to Riley, the female lead character in the Disney Pixar movie *Inside Out* (Rivera, Docter, & Del Carmen, 2015), who came to realise the benefit of experiencing and expressing sadness. However, my study also found that the sense of optimism can diminish with age. Those aged between 55 and 65 were less optimistic than those aged 18-24 and 40-54.

At Time 1, the belief that depression plays a major role differed depending on participants' health characteristics. Unsurprisingly, participants who were diagnosed endorsed the item more, likely because their symptoms are more severe and have more of an impact on their lives. The burden of depression is probably also more pronounced in participants with poor health or whose physical health severely impacts their quality of life. This would explain why they endorsed the item on depression having a major role more than participants in fair or good health, or whose health had no or only a slight impact on their quality of life. A longitudinal decrease in endorsement of this item was observed in female participants, participants younger than 55 years old, White European/Caucasian participants, and participants with at least a Bachelor's degree. The reasons for these findings are not yet clear and will require further study.

One final result to note is the decrease in belief that depression changes one's priorities and affects the future for participants aged 40-54. Such participants could already have a set view of what is important to them and what their future can hold because they have lived for at least four decades. The clarity of priorities and reasonable certainty of one's future outlook are known to be hallmarks of middle age. Perhaps research can distil such attitudes about depression not completely defining one's life despite being a serious illness, and examine how it can be transmitted to depression patients of other ages. These attitudes, if adopted, could arguably aid people with depression to become more resilient.

Antidepressant attitudes. At Time 1, the overall attitude of participants towards the necessity of antidepressants (more than half strongly or moderately agreed to the relevant item) was tempered by the finding that about a quarter of them strongly or moderately believed antidepressants did more harm than good. Slightly more than third of participants felt strongly or moderately strongly that some doctors could be too pro-antidepressant. These attitudes are quite persistent as they did not significantly vary between Time 1 and Time 2. This finding is important because previous studies show that attitudes towards antidepressants do impact adherence (e.g., Brown et al., 2005; Burnett-Zeigler et al., 2014; Chakraborty, Avasthi, Kumar, & Grover, 2009; Johnston, 2013). Therefore, Johnston argued for better depression management through a patient-centred approach, whereby the process of decision-making is shared between healthcare providers and patients. Support for patient-centredness in my study can be seen from the following quote from participant #63, who said it was very important to have a good relationship with her doctor as it enables clear communication about the effects of antidepressants:

> If you are not saying – okay I was on this antidepressant and it did this and like having all this weight and it made me feel like this and if you don't tell them that then they are not going to be able to try and, you know, change. You know, "Oh this one is better and you shouldn't put on all this weight or you shouldn't have these headaches"…So I think if you don't communicate, then they are not going to know any different because they are not in your body they don't know what you are feeling. And when you have depression your thoughts are distorted anyway so you need to be clear to get the best…the best result for yourself. (#63, female, age 18-24)

Between-group comparisons showed that there was greater endorsement of the item on antidepressants causing more harm than good among Asian participants compared to White Europeans/Caucasians. This finding possibly explains why previous evidence of low antidepressant use among Asians. For example, a study in the US using a nationally representative sample of Asian Americans and non-Latino Whites who met the criteria for depressive and anxiety disorders found that Asian Americans were less likely to report antidepressant use in the past year (González et al., 2010). The authors noted that the disparities were not due to differences in mental health need or socioeconomic barriers to accessing care. My study's finding suggests that doctors may need to take more time to assuage the concerns of Asian patients about the harmful side effects of antidepressants. Pending further confirmatory studies, this practical implication could also hold true for other minority groups that have low rates of antidepressant adherence compared to White Europeans (Rossom et al., 2016).

They might need to do the same for undiagnosed patients if they are to be prescribed antidepressants, as such patients in this study were more convinced about harmful antidepressants compared to those who have a depression diagnosis. Doing so may prove challenging as this study also found that undiagnosed patients tend to believe more strongly that some doctors are too pro-antidepressants compared to diagnosed patients. Diagnosed patients correspondingly endorsed the necessity of antidepressants more than undiagnosed patients. These set of findings indicate that severity of depression symptoms could potentially affect antidepressant attitudes.

Regarding longitudinal changes across groups, males increased their belief that antidepressants are harmful more than females. It is possible that male participants were more affected by side effects of antidepressants than the female participants. An early meta-analysis by Montgomery et al. (1994) showed that effects of antidepressants play a role in non-adherence. Also, there are gender differences in an individual's response to side effects, including sexual dysfunction (Montejo-González et al., 1997). This study's finding that males are more likely to become more convinced of the harm of antidepressants could explain why a previous study found higher adherence to antidepressants in women (Banerjee & Varma, 2013). However, Banerjee and Varma's study was conducted in Kolkata, India, which is arguably not comparable with Australia. Rossom et al.'s (2016) more comparable study in the US found no statistical difference in the antidepressant adherence rates of men and women.

Participants aged 18-24 increased their belief in the necessity of antidepressants between Time 1 and Time 2. It is possible they had started taking antidepressants and found them to be useful. Further study comparing antidepressant attitudes and antidepressant consumption would help to understand the reasons for this finding.

Preference for patient-doctor collaboration. Over 80% of participants either moderately or strongly agreed that collaborating with their doctor is key to recovery at Time 1 and the data at Time 2 reflected no significant change. This indicates a receptivity on the part of patients towards partnering with their doctor, which bodes well for the therapeutic relationship. Participant #68 attested to the importance of having a good relationship with her family's doctors:

> So there are a couple of doctors there which we see and they are very conscious and I know that there was one occasion a year or so ago when [my daughter] was in a really bad way and I was just almost beside myself and I remember broke down to the doctor and he was very good. He actually acted really promptly and you know, got me quite, straight onto the community nurse. They have a community nurse there in that clinic. And you know he was very prompt and on the ball, I thought. (participant #68, female, age 40-54)

Interestingly, those aged 40-54 endorsed the importance of cooperating with their doctors more than those aged 18-24. The older participants could have lived through more situations where they realised the importance of having a good therapeutic relationship with their GPs. Again, as mentioned previously, this could be a valuable perspective that might be beneficial to promote.

Diagnosed participants also believed in importance of GP-patient cooperation more than undiagnosed participants, possibly because the latter group is not as convinced that they have a medical problem and so their appreciation for the importance of patient-doctor collaboration is lower. Further research comparing individuals with major depression and individuals with subclinical depression could further uncover whether such differing perspectives are common and whether they give rise to problems. For example, it is possible that individuals with milder forms of depression could find their symptoms worsening in time if they discount the importance of working with their primary healthcare provider.

Summary

The results in this chapter have shed some light on primary care depression patients EMs. Patients had relatively strong beliefs about what caused their depression but there was no obvious preference for either biomedical or psychosocial models. Belief in internal locus of control was relatively low while patient sense of agency was generally high, which means patients are less likely to blame themselves for their condition and more likely to be proactive in alleviating their symptoms. Patients had faith in the efficacy of certain non-medical therapies but not others. As for antidepressants, patients seem to consider them a necessary evil, with a third of them feeling that some GPs are too pro-antidepressant. There was strong evidence that patients consider depression to be highly significant in their lives, both now and in the future, and they also believed that it is important to cooperate with their GPs. Cross-sectional analyses showed that item endorsement levels varied depending on participant characteristics. Most EM beliefs stayed stable over time, except for belief in biomedical causation and belief that depression plays a major role in their life, both of which decreased between Time 1 and Time 2. Longitudinal analyses showed that extent of EM change over time differed according to participant gender, age, culture/ethnicity, and education. These findings have important implications because patient EMs influence other aspects of depression experience. In the next chapter, the belief in depression having biomedical causation will be examined with respect to patient self-stigma.

Chapter 9: Belief in Biomedical Causation of Depression and Self-Stigma in Primary Care Patients

In this chapter, I present and discuss the quantitative results of the longitudinal correlational study to address RQ2 (Does higher endorsement of biomedical causation for depression correlate with lower self-stigma?). Lower self-stigma could potentially improve levels of access and adherence to treatment. As explained in Chapter 4, it is predicted that patients who more strongly endorse biomedical causation for depression would experience lower self-stigma at Time 1 than patients who less strongly endorse biomedical causation (Hypothesis 1). Two additional hypotheses were possible with the longitudinal data. Hypothesis 2a predicted that patients who endorsed biomedical causation of depression at both Patient Survey 1 and 2 would have lower self-stigma at Time 2 compared to Time 1. Hypothesis 2b predicted that patients who endorse biomedical causation at both surveys would have lower self-stigma levels at Time 2 than patients who disagreed with biomedical causation at both surveys. The discussion of results that follows incorporates exemplar quotes from patient interviews to supplement the quantitative findings.

The data for this study are from the 238 participants who completed Patient Survey 1 (see Chapter 7). The demographic and health characteristics of the participants can be found in Chapter 7. Using a 6-point Likert scale, participants rated their agreement to the statement on depression causation in the modified depression CONNECT instrument – patient version (1 = *strongly disagree*; 6 = *strongly agree*). The statement was "I have depression because something in my body is not functioning efficiently (e.g., brain chemistry imbalance)." Using the same 6-point Likert scale, participants rated their agreement to 23 statements mostly modified from the Self-Stigma of Depression Scale and Internalised Stigma for Mental Illness instrument (see Appendix D for full list). The 23 statements were categorised into six subscales. Four of these subscales were used as they had acceptable Cronbach alphas (see Chapter 7): alienation, discrimination experience, help-seeking inhibition, and social withdrawal.

Quantitative Results

Assumption checks. Scatterplots consisting of the self-stigma subscales on the y-axis and participant responses to the biomedical causation statement on the x-axis showed linearity in all four cases (see Appendix N). There were no outliers, but the distributions violated the normality and homoscedasticity assumption for parametric tests. Therefore, the relationship between the self-stigma subscales and endorsement of biomedical causation for depression was investigated using Spearman's rho correlation coefficient.

Cross-sectional analysis. Correlations between belief in biomedical causation for depression and patient self-stigma levels at Time 1 for the entire sample and for subsets of participants grouped according to their demographic and health characteristics. Positive correlation represents an adverse outcome whereby self-stigma increases with increased belief in biomedical causation for depression. Negative correlation on the other hand reflects a beneficial outcome whereby self-stigma decreases with increased belief in biomedical causation.

Overall sample correlations. There was a positive correlation between the alienation self-stigma subscale scores of participants and their level of agreement with the biomedical causation statement, $r_s = .14$, $n = 230$, $p = .032$. Cohen's (1988) guidelines for the strength of relationship classify correlation coefficients ranging between .10 and .29 as weak (.30 to .49 as moderate, and .50 to 1.0 as strong). Greater endorsement of depression being a biomedical condition was associated with higher levels of alienation self-stigma. There was no significant relationship between the discrimination experience subscale and agreement with biomedical causation of depression, nor was there a significant correlation between the social withdrawal subscale and endorsement of the biomedical causation statement. However, there was a statistically significant but weak negative correlation between the help-seeking inhibition self-stigma subscale and endorsement of biomedical causation for depression, $r_s = -.15$, $n = 229$, $p = .024$. The more an individual believes that depression has biomedical roots, the less inhibited they are about seeking help.

Correlations of participants based on their characteristics. Subsets based on patient demographic and health characteristics with $n > 30$ were analysed for correlations between belief in biomedical causation and self-stigma subscales. Significant ($p < .05$) or near significant ($p < .10$) Spearman correlation coefficients (r_s) are in Table 16 with their exact p-values. Participant characteristics which showed statistically significant correlations between biomedical belief and any self-stigma subscales were further analysed with partial correlations, to determine if the characteristics had any influence upon the variables of interest.

Table 16

Correlations Between Self-Stigma and Belief in Biomedical Cause of Depression According to Participant Characteristics

Characteristics	Alienation	Discrimination experience	Help-seeking inhibition	Social withdrawal
Gender				
Female (*n* = 169)	.051 (ns)	-.017 (ns)	-.16*	-.032 (ns)
Male (*n* = 56 or 57)	.31*	.15 (ns)	-.19 (ns)	.14 (ns)
Age (years)				
18-24 (*n* = 74 or 75)	.087 (ns)	.061 (ns)	-.345**	-.087 (ns)
25-39 (*n* = 95)	.11 (ns)	.006 (ns)	-.071 (ns)	.028 (ns)
40-54 (*n* = 40 or 41)	.058 (ns)	-.072 (ns)	-.17 (ns)	.095 (ns)
Culture/Ethnicity				
Asian (*n* = 37)	.46**	.33*	-.31 (.065)	.16 (ns)
European (*n* = 162 or 163)	.024 (ns)	-.019 (ns)	-.17*	.011 (ns)
Education				
≥ Bachelor (*n* = 137 or 138)	.075 (ns)	-.001 (ns)	-.25**	.011 (ns)
Cert/Dip/Adv Dip (*n* = 46)	.19 (ns)	-.072 (ns)	.022 (ns)	-.053 (ns)
Up to Year 12 (*n* = 36)	.24 (ns)	.25 (.067)	.005 (ns)	.16 (ns)
Diagnosed with depression				
Yes (*n* = 168 or 169)	.11 (ns)	.049 (ns)	-.12 (ns)	.029 (ns)
No (*n* = 51)	.18 (ns)	-.038 (ns)	-.21 (ns)	.016 (ns)
Earliest depression onset				
≤ 1 year ago (*n* = 38 or 39)	.21 (ns)	-.001 (ns)	-.25 (ns)	-.11 (ns)
> 1 and ≤ 5 yrs ago (*n* = 48)	.27 (ns)	.14 (ns)	-.26 (.079)	.16 (ns)
> 5 and ≤ 10 yrs ago (*n* = 50)	.19 (ns)	.16 (ns)	.047 (ns)	.12 (ns)
>10 yrs (*n* = 76 or 77)	.015 (ns)	-.11 (ns)	-.11 (ns)	-.082 (ns)
Psychiatric co-morbidity				
Yes (*n* = 187 or 186)	.19**	.077 (ns)	-.15*	.063 (ns)

No (n = 43)	-.10 (ns)	-.21 (ns)	-.13 (ns)	-.090 (ns)
Current physical health				
Poor (n = 45)	.098 (ns)	.051 (ns)	.035 (ns)	.26 (.085)
Fair (n = 111)	.088 (ns)	.022 (ns)	-.17 (.075)	.020 (ns)
Good (n = 62 or 63)	.27*	.053 (ns)	-.30*	-.091 (ns)
Impact on quality of life				
Severely (n = 33 or 34)	.18 (ns)	-.045 (ns)	.005 (ns)	.079 (ns)
Moderately (n = 69)	.23 (.059)	.092 (ns)	-.080 (ns)	.17 (.079)
Slightly (n = 69)	.026 (ns)	-.084 (ns)	-.26*	-.15 (ns)
Not at all (n = 45 or 46)	.056 (ns)	.10 (ns)	-.26 (.086)	-.081 (ns)

Note. p-values are in parentheses. *$p < .05$, **$p < .01$.

Partial correlations. Ten non-parametric partial correlations were conducted, that is, four for biomedical belief and alienation while controlling for gender, culture/ethnicity, psychiatric co-morbidity, and current physical health; one for biomedical belief and discrimination experience, controlling for culture/ethnicity; and seven for biomedical belief and help-seeking inhibition, controlling for all participant characteristics except depression diagnosis and length of depression. No partial correlations involving biomedical belief and social withdrawal were required as no statistically significant (i.e., $p < .05$) correlations were found in the subset of participants who had been grouped according to the nine characteristics (see Table 16).

The partial correlations involving biomedical belief and alienation ranged between .139 and .154 (all p-values < .05), reflecting negligible differences from the zero-order correlation of .141. This result suggests that gender, culture/ethnicity, psychiatric co-morbidity, and current physical health had little effect on the strength of the relationship between biomedical belief and alienation. As for the partial correlation of biomedical belief and discrimination experience controlling for culture/ethnicity, it was .050 (p = .45), reflecting a very small difference from the zero-order correlation of .033 (p = .62). This finding indicates that culture/ethnicity had almost no effect on the strength of the relationship between biomedical belief and discrimination experience. Finally, the partial correlations involving biomedical belief and help-seeking inhibition ranged between -.143 and .156 (all p-values < .05), reflecting negligible differences from the zero-order correlation of .149. This finding suggests that gender, age, culture/ethnicity, education, psychiatric co-morbidity, current physical health, and impact of physical health had little effect on the strength of the relationship between biomedical belief and help-seeking inhibition. Overall, the partial correlations indicated that participant characteristics did not influence the correlations between belief in biomedical causation for depression and self-stigma.

Longitudinal analysis. The availability of Time 2 data made it possible to further explore the original research question on the correlation between belief in biomedical causation for depression and patient self-stigma. Two follow-up hypotheses were proposed:

Hypothesis 2a: Patients who moderately or strongly agreed with biomedical causation of depression at Time 1 and Time 2 would have lower self-stigma levels at Time 2 compared to Time 1 (as reflected in the scores on the four self-stigma subscales);

Hypothesis 2b: Patients who moderately or strongly agreed with biomedical causation of depression at Time 1 and Time 2 would have lower self-stigma levels at Time 2 than patients who moderately or strongly disagreed with biomedical causation in both surveys.

Before testing H2a and H2b, I checked for the effect of time on depression symptoms. Previous studies have reported that depression episodes last for between three and six months (Eaton et al., 2008; Lopez & Murray, 1996) and half of depressive episodes go into remission without intervention (Mueller et al., 1999). Reduced severity of symptoms could be associated with reduced patient self-stigma. The period of time between the two surveys ranged between 181 and 370 days (an extreme outlier of 608 days was excluded from analysis), $M = 229$, $SD = 55$. No statistically significant correlation was found between severity of depression symptoms at Time 2 and the inter-survey period ($r_s = .051$, $p = .570$). As such, no further analysis to control for time was needed.

Hypothesis testing with a Wilcoxon signed-rank test that showed alienation subscale scores at Time 2 ($Mdn_2 = 3.67$) were statistically significantly lower than at Time 1 ($Mdn_1 = 3.75$) for participants who either moderately or strongly agreed on biomedical causation in both patient surveys ($n = 64$), $z = -1.97$, $p = .049$, small-to-medium effect size ($r = .25$). These participants' scores for the other subscales were no different at Time 2 compared to Time 1. Testing H2b with a Mann-Whitney U test showed no statistically significant differences between self-stigma subscale scores of patients who moderately/strongly disagreed with biomedical causation for depression and self-stigma subscale scores of patients who moderately/strongly agreed with biomedical causation.

Discussion

Hypothesis 1 predicted that primary care patients who believe more strongly in biomedical causation for depression would experience lower self-stigma, as measured by the four subscales of alienation, discrimination experience, help-seeking inhibition, and social withdrawal at Time 1. The hypothesis was partially supported. For help-seeking inhibition subscale, there was a statistically significant weak negative correlation. Higher biomedical belief was associated with less reservation about seeking help. For alienation subscale, the correlation was statistically significant and positive, although weak. Higher biomedical belief was associated with greater alienation. This finding was unexpected. The overall correlations between biomedical belief and the other two self-stigma subscales, discrimination experience and social withdrawal, were not statistically significant.

Partial correlations indicated that participant characteristics did not meaningfully affect the correlations between biomedical belief and the relevant self-stigma subscale. In addition, correlations of participant sub-samples grouped according to demographic and health characteristics reflected differences by participant gender, age, culture, and current physical health. Positive correlation between biomedical belief and alienation self-stigma was stronger in males than females, in Asians than White Europeans/Caucasians, and in participants who rated themselves as physically healthy compared to those who did not. A positive correlation for discrimination experience was also present in Asians, but the same correlation was negative in White Europeans/Caucasians. Finally for help-seeking inhibition, the negative correlations were stronger in those aged 18-24 compared to other age groups, and those who rated themselves as physically healthy compared to those who did not.

Hypothesis 2a predicted that patients who moderately or strongly agreed with biomedical causation of depression at Time 1 and Time 2 would have lower self-stigma levels at Time 2 compared to Time 1 (as reflected in the scores on the four self-stigma subscales). It was supported for the alienation subscale only. Hypothesis 2b predicted that self-stigma levels (measured at Time 2) of patients who consistently endorsed biomedical causation (i.e., moderately or strongly agreed in both Time 1 and Time 2 surveys) would be lower than for patients who consistently did not endorse biomedical causation (i.e., moderately or strongly disagreed in both surveys). It was rejected for all self-stigma subscales.

This final section discusses the possible reasons and implications of this study’s results. Overall correlations across the sample, and correlations by participant characteristics that were at least .30 (representing moderate strength of relationship), are discussed. As noted earlier, I provide quotes from the qualitative interview data where relevant to enrich the findings.

Alienation. The positive association between belief in biomedical causation of depression and alienation for the entire sample suggests that primary care doctors should use standalone biomedical explanations with caution. Although there was a statistically significant decrease in alienation for patients who moderately or strongly believed in biomedical causation at both Time 1 and Time 2 (support for Hypothesis 2a), it was only a marginal decrease. Median at Time 2 was

0.08 lower than at Time 1. Moreover, rejection of Hypothesis 2b showed that patient self-stigma levels for all subscales were not lower in participants who consistently endorsed biomedical causation compared to those who did not. This means there is no benefit of self-stigma reduction in maintaining biomedical belief over time.

On the contrary, there is a risk of greater alienation self-stigma. Patients who score high on the alienation subscale are likely to feel isolated, ashamed, inferior, and disappointed in themselves for having depression. They also believe it is impossible for others without depression to understand them, and that others cannot get close to them because of their illness. Such alienation could exacerbate suicidal tendencies, given the well-documented link between depression and suicide. A meta-analysis by Cavanagh, Carson, Sharpe, and Lawrie (2003) reported that more than half of all individuals who committed suicide met the criteria for current depressive disorder. GPs have a key role in effective suicide prevention; for example, Pearson et al. (2009) found that 91% (n = 224) of individuals with mental illness who committed suicide had at least one GP consultation in the year prior to death. Half had their final appointment a month before death, and one-sixth had one in the week leading to death. Perhaps if GPs presented more holistic explanations for depression, instead of only describing biomedical causation, they could help reduce the associated alienation self-stigma of their patients, which in turn could help reduce suicides. Further research could determine the presence of such causal mechanisms.

When the patient sample was analysed as subsets, this study found the correlation between biomedical belief and alienation was more pronounced in males than females. This finding is salient to suicide prevention as studies show that males with depression are at a greater risk of suicide than females (Hawton, Casañas i Comabella, Haw, & Saunders, 2013). In light of this, doctors should be particularly careful with using standalone biomedical explanations for depression with male patients. The correlation between biomedical belief and alienation was also stronger in Asian participants of this study compared to White Europeans/Caucasians. A recent report on suicides (Ho, Au, & Amerasinghe, 2015) reviewed literature from Asian countries and Western countries with significant Asian immigrant populations and reported that depression is a risk factor for suicide among Asians. Other risk factors associated with being immigrants were also identified such as family conflict, social isolation, and loss of social support networks, in addition to the stigma of mental illness. As these factors could potentially add to the negative effects of alienation self-stigma, doctors may want to exercise caution over using strictly biomedical explanations for depressions with patients of Asian ethnicity. In my interview study, I spoke to a half-Asian participant who related how he felt awkward talking about depression to his Asian mother. He said:

“I don’t think she believes depression exists. She just said there are plenty of people who work harder than you and they don’t get depressed” (participant #49, male, aged 18-24).

A finding of interest is the small-to-moderate correlation between biomedical belief and alienation in participants in good current physical health. Although it did not meet the minimum criteria of .30, it is worth discussing because such physically healthy patients could face difficulties trying to convince others about their inner struggles. One participant who rated his physical health as good, said he no longer speaks openly about having depression:

> I’ve changed my mind again, because I’ve put it out there and it’s come back and bitten me every time so...every time I do say something and expect some sort of understanding, it doesn’t happen, even when you go these social workers or psychologists, they have little clue about the feeling. It comes straight out of a book; they don’t know…unless you’ve got it, you don’t know shit. (participant #64, male, aged 55-65)

This participant also moderately or strongly agreed with five out of the six items on the alienation subscale (reverse-coded items adjusted accordingly). For example, he strongly felt isolated and inferior because of his depression.

In sum, the biomedical explanation for depression could have unintended negative consequences with respect to alienation self-stigma. The alternative is to incorporate a more holistic perspective of depression in clinical encounters. Since most experts agree that depression has biopsychosocial elements in cause and recovery, future research could examine the relationship between a more nuanced portrayal of depression (i.e., a biopsychosocial instead of a biomedical model) and patient self-stigma. Incorporating psychosocial aspects of depression could increase a patient’s sense of agency; for example, a greater willingness to learn new stress management skills or change current thinking patterns rather than expecting a pill to fix everything. This position is in line with earlier studies on biomedical EMs for depression and stigma; for example, Schreiber and Hartrick (2002) who expressed concerns over their interview study participants who adopted a biomedical EM to the exclusion of a more in-depth and contextual understanding of depression. Mausner-Dorsch and Eaton (2000) also underscored the importance of psychosocial context in depression when they showed that high psychological strain and low decision authority at work were associated with incidence of major depressive episode, depressive syndrome, and dysphoria in employees.

Discrimination experience. Correlation between belief in biomedical causation of depression and discrimination experience was positive and moderate for Asian participants in this study. Individuals who score highly in the discrimination experience self-stigma subscale feel that others discriminate against them, ignore them and are not keen to be close to them. One possible reason could be that the patients in this study attributed discrimination that they face for being an ethnic minority as discrimination due to their mental illness. White European/Caucasian participants who are in the majority in Australia would not experience discrimination due to one's minority status. This could possibly explain why the same correlation was markedly different for these participants – the coefficient was negative, almost negligible and not statistically significant.

The existence of public discrimination that can influence one's view of self has been shown in previous studies (Dunn, Forrest, Burnley, & McDonald, 2004; McAllister & Moore, 1989). In a telephone survey of over 5,000 New South Wales and Queensland residents, Dunn et al. reported persistence of intolerance against Asian Australians and other out-groups such as Jewish Australians and Muslim Australians. For example, 27.4% of those surveyed by Dunn et al. indicated some level of concern if their close relative married someone of Asian background. An earlier nationwide polling study by McAllister and Moore had also revealed strong anti-Asian sentiments. One application of my study's finding could be more targeted anti-stigma initiatives in GP clinics attended by high proportions of Asian patients (e.g., posters in the waiting area or restrooms). In addition, future research on discrimination experience among other ethnic minorities could yield valuable insights on how to effectively fight such self-stigma.

Help-seeking inhibition. The finding that belief in biomedical causation is negatively correlated with help-seeking inhibition can be explained by a couple of factors. Firstly, it could be due to selection bias as participants in this study had already sought help from a general practitioner for their depression. As such they might not score highly on feeling embarrassed about seeking professional help, or feeling embarrassed if others knew they were seeking help (two of the three items in the help-seeking inhibition subscale). The participants endorsing a biomedical model may have a higher rate of treatment with antidepressant medication than other participants (this information was not requested in the questionnaires) so their reservations about visiting a GP (for a prescription) could be lower for that reason. The median for this subscale across the entire sample is 3.0, indicating a medium-to-low level of help-seeking inhibition, which can be interpreted as further evidence of the selection bias described above.

Another possible reason for the negative correlation between biomedical belief and help-seeking inhibition could be that participants with a biomedical EM for their depression relate well to their doctors, who also tend to adopt biomedical perspectives, and therefore such patients are open to seeking intervention. As mentioned in Chapter 3, doctor EMs tend to prefer biomedical EMs (Andersson et al., 2001; McPherson & Armstrong, 2012; Thomas-Maclean & Stoppard, 2004). The importance of the GP-patient relationship was highlighted in Kravitz et al.'s study (2011) that showed how individuals with depression might decide against seeking help or disclosing about their problems due to barriers in their relating with their doctor. These barriers include perceptions of the GP's mental-health related competence and preferences. It could also be true that patients who are not inhibited about help-seeking tend to seek medical help more actively, and as such, their perspectives could have been shaped by repeated interactions with their doctors. Further research is needed to understand the causal mechanisms for this correlation result.

A recent qualitative synthesis of patients' experiences with regards to help-seeking behaviour for depression reported that help-seeking is often delayed because it is seen as a threat to identity (Doblyte & Jiménez-Mejías, 2017). The study also identified mental illness stigma as one of the main barriers to help-seeking. Understanding how biomedical EM of depression can help reduce help-seeking inhibition would be beneficial since the biomedical perspective for mental illness is prevalent (Kvaale et al., 2013). Interestingly, the negative correlation between belief in biomedical causation of depression and help-seeking inhibition in this study was strongest among participants aged 18-24, compared to the other two age groups with at least 30 participants. This correlation was also found among participants with good current physical health, at .30 (moderate strength of relationship). No statistically significant correlations were found in patients with poor or fair physical health. Further study is needed to determine reasons for this finding and to understand causal mechanisms. If biomedical belief does result in more help-seeking behaviour, one possible practical application of this result could be to target people in the relevant age group and those who are physically in good health with biomedical portrayals of depression. For example, the young can be reached using platforms like Instagram and Twitter, and employing phrases that would resonate with them. The physically healthy could be reached through posters at locations they frequent such as exercise centres and health food stores.

A note of caution though: the negative correlation between biomedical belief and help-seeking inhibition in this study was of limited magnitude. As such, further study is needed to determine whether biomedical belief is able to promote actual help-seeking and healthy discussions about depression interventions. The experience of one participant who was born in North America, a region with high rates of antidepressant use (Mamdani & Wilby, 2013), suggests that widespread acceptance of biomedical treatment may not lead to less stigma or more open conversations about depression treatment. After living in Australia for several years, he felt there was more public discourse about mental health issues here than his birth country:

> There is a lot more public awareness, public service announcements and things like that related to depression here in Australia. I find that, generally I find that reaffirming in some strange way just in the sense of I think at least recognising that mental health is important. In the US I don't think that they value it quite as highly and you just don't – there aren't as

> many prompts in the media or in general life to talk about that in the same way that you would have here in Australia… certainly in the US I knew a lot of people who were taking medications for depression but we never really talked about it…there was still an element of where raising that as a topic of conversation was not something – there was still a, stigma is a bit of a strong word, but a bit of a stigma about raising that conversation. (participant #94, male, age 40-54)

Summary

This chapter has shed some light on whether greater endorsement of biomedical causation for depression does correlate with lower patient self-stigma. There were no statistically significant overall correlations between biomedical belief and the subscales of discrimination experience and social withdrawal, but biomedical belief was associated with greater alienation and lower help-seeking inhibition. Essentially a stronger belief in biomedical causation of depression could have no effect on self-stigma as the opposing directions of the alienation and help-seeking inhibition subscale could cancel each other out, particularly as both had small effect sizes. Some possible explanations for these apparently contradictory findings were offered: that is, selection bias of participants and exposure to biomedical belief from their GPs. Perhaps the patients in the sample who believed strongly in biomedical causation do experience alienation towards significant others in their life, but the alienation does not prevent them from consulting their GPs.

Alienation could exacerbate suicidality, whereas less inhibition about help-seeking could translate into more active help-seeking by patients. With this knowledge, GPs could potentially wield biomedical explanations more effectively as anti-stigma tools. For example, they may decide against standalone biomedical models for severely depressed individuals. On the other hand, for less depressed individuals, especially if they are in their late teens to early 20s, using biomedical explanations could help promote help-seeking. Studies show the GP-patient relationship could itself be a barrier to help-seeking (Doblyte & Jiménez-Mejías, 2017). Individuals who think that their GPs' knowledge, skills, and expertise for depression treatment are limited would not likely seek help through primary health care (Kravitz et al., 2011). In addition, participants in Kravitz et al.'s study found it hard to engage verbally with their GPs and/or explain their troubles. The issue of whether GP-patient communication effectiveness is associated with certain aspects of patient explanatory models will be examined in the next chapter.

Chapter 10: Depression Explanatory Model and Primary Care Patients' Perception of Communicative Effectiveness with Their General Practitioners

In this chapter I present and discuss the quantitative results of an exploratory correlational study between depression explanatory models (EMs) of primary care patients and their perceptions of communication effectiveness with their GPs. As explained in Chapter 5 effective communication between patients and doctors is key in managing depression in primary care. Using communication accommodation theory (CAT: Gallois et al., 2005; Giles, 1973), I examined how patient endorsement of EM items correlates with patient perception of GP-patient communication effectiveness (RQ3). I also tested the hypothesis that patient ratings of GP-patient communication effectiveness are higher in patients who feel their GPs share their views about their depression compared to patients who did not. These were ancillary analyses to supplement the primary analyses for RQ3. Finally, patient interview excerpts and free-text responses are presented to support the quantitative data and illustrate effective CAT strategies.

Data for this study are from the 238 participants who completed Patient Survey 1, described in Chapter 7. Participant demographic and health characteristics are also in Chapter 7. Participants rated their agreement to 16 EM statements in the modified CONNECT depression instrument (see full list in Appendix C1) using a Likert-type scale (1 = *strongly disagree*; 6 = *strongly agree*). Similarly, they rated their agreement to 16 statements on the use of CAT strategies (see full list in Appendix E). The CAT survey statements formed four subscales with acceptable Cronbach alphas that is, emotional expression, discourse management, interpretability, and interpersonal control (details in Chapter 7).

To determine whether GP-patient communication effectiveness ratings are higher in patients who perceived GP-concordance compared to patients who perceived non-GP concordance, patients were asked whether their doctors shared their views on five EM dimensions (see list in Appendix O). Responses were limited to yes, no, and do not know/unsure. Patients who answered yes were categorised as the concordant perceptions group, while negative responses constituted the discordant perceptions group. The Mann-Whitney U procedure tested the hypothesis that GP-patient communication effectiveness is higher (i.e., higher scores in the CAT subscales) in the concordant perceptions group compared to the discordant perceptions group.

Quantitative Results

Assumption checks. Scatterplots consisting of CAT subscale scores on the y-axis and participant responses to the EM statements on the x-axis consistently showed linearity. There were no outliers, but the distributions violated the normality and homoscedasticity assumption for parametric tests. Therefore, the relationship between the four CAT subscales and endorsement of EM statements was investigated using Spearman's rho correlation coefficient. As this was an exploratory study, with no directional hypothesis, I used the two-tailed test for significance.

Correlations from primary analyses. Table 17 shows correlations between the 16 modified CONNECT depression items and the four CAT subscales. Significant Spearman correlation coefficients (r_s) ($p < .05$) and near-significant correlations ($p < .10$) are listed with exact *p*-values. Only correlations above .30, which is the recommended threshold for a medium effect size (Cohen, 1988), were used to guide further analysis. The correlations between preference for GP-patient cooperation and the four CAT subscales, with coefficients ranging between .43 and .49, met this cut-off. The same correlations were then calculated within subsets of participants grouped according to their demographic and health characteristics. As detailed in Chapter 7, these were gender, age, ethnicity, education level, the presence of depression diagnosis, the length of depression, psychiatric co-morbidity, current physical health, and impact of physical health on quality of life. Almost all resulting correlations were statistically significant (p < .05 or p < .01) and the statistically significant coefficients ranged from .26 to .64 (table of coefficients is in Appendix P). Therefore the correlations between preference of GP-patient cooperation and the CAT subscales can be interpreted as a main effect, as it is clearly reflected across the entire sample, regardless of any participant characteristic.

Table 17

Correlations Between Modified CONNECT Depression Items and Communication Accommodation Strategy Subscales

Measure	Emotional expression	Discourse management	Interpretability	Inter-personal control
Biomedical causation of depression	.17 (.010)	.18 (.005)	.15 (.026)	.22 (.001)
Psychosocial causation	.061 (*ns*)	.120 (.069)	.095 (*ns*)	.084 (*ns*)
Patient choices led to depression (internal locus of control)	-.032 (*ns*)	-.096 (*ns*)	-.055 (*ns*)	-.053 (*ns*)
Right action will reduce symptoms (sense of agency)	.13 (.042)	.18 (.007)	.12 (.063)	.17 (.011)
Belief in complementary medicines	-.002 (*ns*)	.016 (*ns*)	.048 (*ns*)	.021 (*ns*)
Belief in complementary activities	-.055 (*ns*)	-.005 (*ns*)	-.037 (*ns*)	-.002 (*ns*)
Belief in non-doctor remedies	.002 (*ns*)	.027 (*ns*)	.013 (*ns*)	.005 (*ns*)
Belief in alternative providers	.15 (.026)	.19 (.004)	.18 (.008)	.22 (.001)
Depression has changed priorities	.068 (*ns*)	.15 (.027)	.11 (.091)	.16 (.014)
Depression plays a major role	.093 (*ns*)	.12 (.082)	.10 (*ns*)	.17 (.010)
Depression changes one's future	.090 (*ns*)	.13 (.053)	.084 (*ns*)	.14 (.032)
Depression can have positive outcome	.11 (.084)	.17 (.011)	.14 (.034)	.15 (.023)
Antidepressants are necessary	.22 (.001)	.22 (.001)	.23 (.0004)	.23 (.0005)

Antidepressants: more harm than good	-.20 (.002)	-.18 (.006)	-.18 (.007)	-.20 (.003)
Some GPs are too pro-antidepressants	-.19 (.004)	-.17 (.010)	-.17 (.011)	-.18 (.007)
Cooperation with GP key to recovery	.43*	.49*	.45*	.45*

Note. p-values are in parentheses. *ns* = not significant. **p* < .0001.

Results of ancillary analyses.

Concordance/non-concordance. The majority of participants believed their GPs shared concordant views of their depression for all five dimensions (52.8% to 74.9%) (see Figure 2). The dimension of internal locus of control had the highest percentage of unsure responses (21.4%), whereas the dimension of patient sense of agency had the lowest percentage (10.5%). The implications of these findings will be further discussed in the final section of this chapter and illustrated with exemplars of free-text responses from participants who elaborated on their answers in Patient Survey 1.

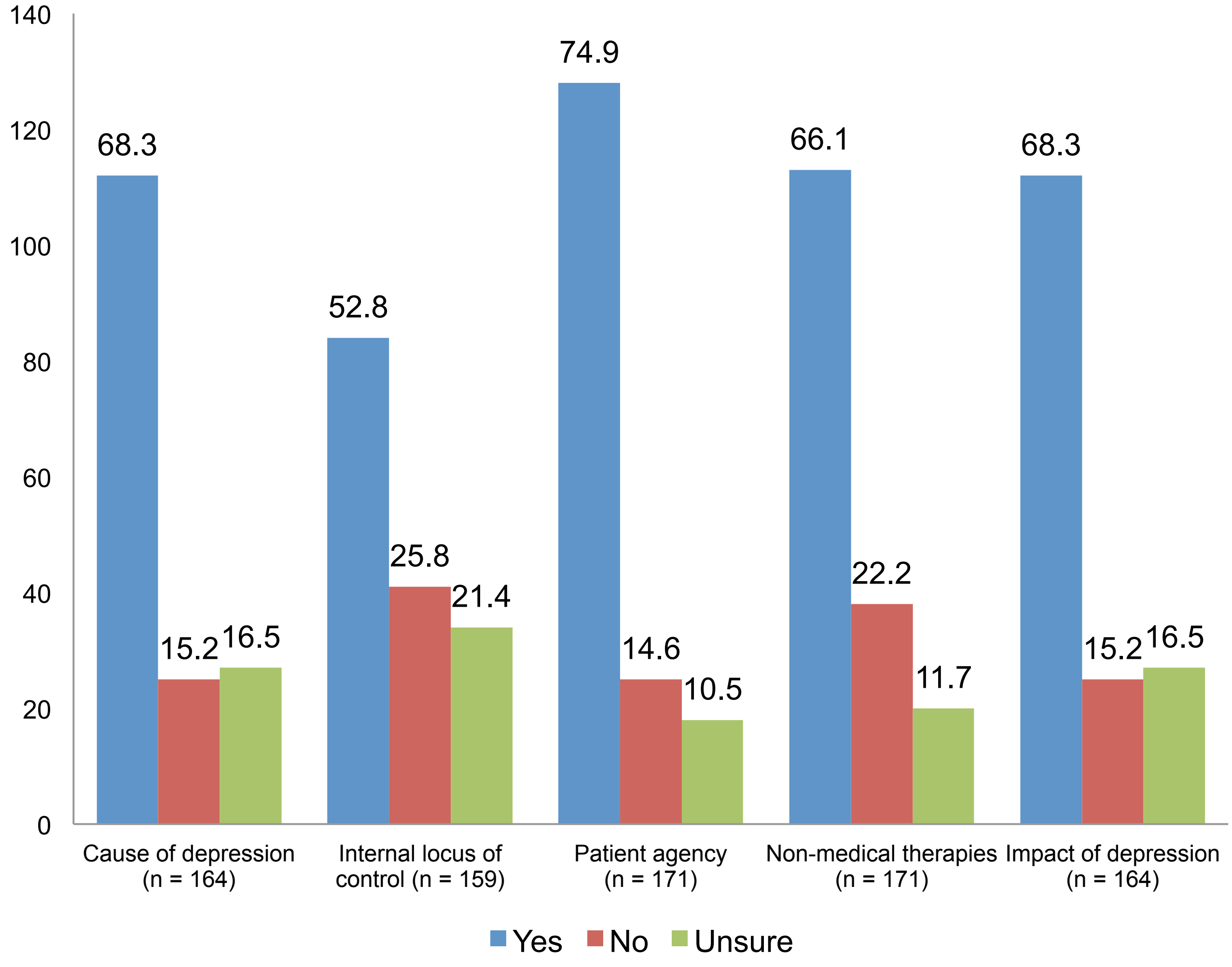


Figure 3. Patient response when asked if GP shares their view about their depression (x-axis represents absolute number of participants, percentages as column data labels).

Unfortunately, many participants did not answer the questions about their GP's views, as the proportion of missing values ranged between 28% and 33%. This was potentially due to survey fatigue as the questions were at the end of the questionnaire. Consequently further analysis (i.e., testing whether patient ratings of GP-patient communication effectiveness are higher in patients who feel their GPs share their views about their depression compared to patients who did not) was limited to two EM dimensions. These were internal locus of control and belief in non-medical therapies, where both the concordant and discordant groups were sufficiently large (*n* > 30).

Hypothesis testing. According to results of the Mann-Whitney U analysis, patient ratings of GP-patient communication effectiveness are higher in the concordant group than the discordant group for the internal locus of control dimension that is, the belief that patient choices led to depression (internal locus of control) – see Table 18. Effect sizes were moderate-to-strong. The same pattern of results was also obtained for the non-medical therapies dimension for all four CAT subscales (see Table 19). However, effect sizes were not as strong, as they were in the low-moderate range.

Table 18

GP-Patient Communication Effectiveness in Concordant versus Discordant Patients for Internal Locus of Control Dimension

	Concordant perceptions		Discordant perceptions		*p*	Effect-size *r*
	Mdn	Mean rank	*Mdn*	Mean rank		
Emotional expression (*n* = 124)	5.6	72.8	4.6	41.6	.000004	-.41
Discourse management (*n* = 125)	5.9	73.5	4.8	41.6	.000002	-.43
Interpretability (*n* = 125)	5.7	71.4	4.7	45.8	.0001	-.34
Interpersonal control (*n* = 125)	6.0	72.8	5.0	42.9	.000006	-.41

Table 19

GP-Patient Communication Effectiveness in Concordant versus Discordant Patients for Efficacy of Non-Medical Therapies Dimension

	Concordant perceptions		Discordant perceptions		*p*	Effect-size *r*
	Mdn	Mean rank	*Mdn*	Mean rank		
Emotional expression (*n* = 150)	5.3	81.5	4.6	57.9	.004	-.24
Discourse management (*n* = 151)	5.6	81.3	4.9	60.4	.009	-.21
Interpretability (*n* = 151)	5.3	81.8	4.7	58.9	.004	-.23
Interpersonal control (*n* = 151)	5.5	81.8	5.0	58.7	.003	-.24

Discussion

RQ3 asked whether certain patient EM beliefs correlate with perception of greater GP-patient communication effectiveness. Primary analyses showed that most of the overall correlations in Table 17 were weak and/or not statistically significant. However there were three noteworthy trends and one set of moderately strong correlations that were consistent across the entire sample. In addition, ancillary analyses showed that the majority of patients believed that their GPs shared their views on the five EM dimensions, that is, causation, internal locus of control, sense of agency, efficacy of non-medical therapies, and significance of depression. At the same time, there were considerable proportions of participants who felt their GPs did not share their views or they were unsure. Hypothesis testing showed support for the prediction that patient ratings of GP-patient communication effectiveness (i.e., scores on the four CAT subscales) is higher in patients who feel their GPs share their views compared to patients who did not in the EM dimensions internal locus of control and belief in non-medical therapies.

The possible reasons and implications of these results are discussed in the following section and supplemented where appropriate with exemplar quotes from participants' free-text responses and patient interviews described in Chapter 7. The quotes also show how the CAT measures of emotional expression, discourse management, interpretability, and interpersonal control operate in patient encounters with their GPs. For example, a patient who feels their doctor engages them and is a good listener, has benefitted from the strategy of good discourse management.

Primary findings (correlations).

Trends. The first trend in the results pertained to the correlations between biomedical causation belief and the four CAT subscales, and the correlations between belief in necessity of antidepressants and four CAT subscales. The coefficients of these correlations were positive and statistically significant ($p < .05$), ranging between .15 and .23 (weak to low-moderate effect size). Taken together, they indicate that the more a patient adopts a biomedical orientation about their depression, the more they tend to view their GP-patient communication as effective. The second trend of results was opposite to the first. It involved correlations between the four CAT subscales and the belief that antidepressants are more harmful than beneficial, and correlations between the CAT subscales and the belief that some doctors are too pro-antidepressant. These coefficients of these correlations were also statistically significant ($p < .05$) and showing weak to low-moderate effect size, but they were negative (ranging from -.17 to -.20). This suggests that the more 'anti-biomedical' a patient is (at least concerning antidepressants), the less they tend to view their GP-patient communication as effective. Further study is needed to determine whether anti-biomedical attitudes are confined to antidepressants, or whether they indicate a more psychosocial outlook on depression. Perhaps such patients find their perspectives clashing with that of their doctors who have a more biomedical orientation about depression, and this leads to decreased communication effectiveness. Studies reviewed in Chapter 3 have shown that doctors tend to endorse biological explanations of depression and believe in the efficacy of antidepressants (e.g., Ogden et al., 1999; Street & Haidet, 2011) more than patients. Alternatively, it is possible that ineffective GP-patient communication could erode a patient's belief in the biomedical perspectives for depression shared by their doctors, and it could also increase scepticism about doctors being pro-antidepressant. More research (e.g., using vignettes varying by condition) will help uncover the causal mechanisms behind these correlations.

Nevertheless, the pattern of positive and negative correlations described above could shed light on an earlier vignette study by Swenson, Zettler, and Lo (2006) on adult patients' preferences of doctor communication styles. Swenson et al. reported that the majority (69%) of patients who preferred the doctor with a patient-centred communication style, liked how the doctor worked with the patient, respected their wishes, and explored what they wanted. The rest who preferred the biomedical, or doctor-centred, communication style liked that the doctor pre-empted harm, exhibited medical authority, and delivered information with clarity. Swenson et al. said the implication of their study was on how understanding diversity of patient communication preferences could result in more effective and individualised care. I propose an alternative interpretation in that the patients in Swenson et al.'s study who preferred a biomedical communication style could have had a biomedical orientation about their condition. The others who wanted their doctors to respond to their ideas and emotions about their illness, a hallmark of patient-centred communication (Epstein, 2006), could have had a more psychosocial orientation. It is possible that the more doctors can accurately perceive their patient's EM (i.e., the greater the awareness of patient EM), the more they can discuss depression in a way that resonates with the patient and help aid recovery. This proposition is particularly salient for depression because the condition has diverse explanations (as shown in Chapter 2), and will be explored in the next chapter.

The third trend in the results pertains to the correlations for the beliefs that right action will reduce symptoms (patient sense of agency); that alternative providers can provide helpful remedies; and that depression can have positive outcomes such as increased empathy. All of these items had positive correlations with the four CAT subscales. Spearman's correlation coefficients ranged from .11 to .19 (weak to low-moderate effect size), and all were statistically significant ($p < .05$), except for $r_{\text{agency x interpretability}} = .12$ ($p = .063$) and $r_{\text{positive outcome x emotional expression}} = .11$ ($p = .084$). Collectively these results suggest that a patient with a hopeful outlook, that is, has a sense of agency, is open to exploring other help-seeking avenues, and able to maintain optimism, tends to rate their GP-patient communication as effective. This could be because these patients' natural inclination towards optimistic thinking, which has remained intact despite their depression, positively influences how they perceive the GP-patient communication. A study by Jensen, King, Guntzviller, and Davis (2010) found that optimistic thinking as a coping mechanism was among other factors that predicted communication satisfaction among low-income patients. Future research into such patient perspectives could distil ways to encourage optimism and resilience, such as asking why patients choose to believe that depression can bring about positive outcomes. A possible instrument that can be used is the Silver Lining Questionnaire (Sodergren & Hyland, 2000), which was developed for patients with a variety of conditions including depression.

It is also possible that the third trend in results as detailed above is due to GPs finding it easier to engage such optimistic patients. As such their communication is perceived by the patient as more effective, and the GP is also likely to share their perception. Further research with GP participants could help uncover the role of having optimistic EM dimensions in communication and treatment outcomes for depression.

Strong correlations. The strongest correlations in Table 17 involved endorsement of GP-patient cooperation as key to recovery. The positive correlation coefficients with all four CAT subscales ranged from .43 to .49 ($p < .0001$), which is close to Cohen's (1988) .50 guideline for large effect size. Similar correlations in terms of size and direction were also found when patients were grouped according to their characteristics (see Appendix P). This highlights the importance of patient belief in GP-patient collaboration for optimising GP-patient communication effectiveness. The two factors are mutually reinforcing. The more a patient believes in working with their doctor, the more they tend to rate their GP-patient communication as effective. At the same time, the more effective the communication is perceived to be, the more the patient thinks it is important to work with their doctor. This exemplar quote from participant #94, who strongly endorsed GP-patient cooperation in both patient surveys, highlights the discourse management strategies that help him engage in the GP consultations, which in turn encourages him to continue collaborating:

> I would say, she's fairly direct about asking questions. Also is fairly good about prompting for any additional things that we might have to talk about and has generally been [pause] also fairly open about getting additional assistance. So with a couple of the health things that I've talked with her about she's very supportive of seeing a specialist or something like that for various purposes or to get tests done when needed. So – and personally I find that very reassuring that they are listening to me, that she's listening to me and that I feel she's valuing my input into the discussion. (male, age 40-54)

This result aligns with previous studies on how effective communication during clinical encounters results in better treatment outcomes (Street et al., 2009) particularly for mental health conditions (Horvath et al., 2011; Sharf et al., 2010).

The main correlational result also means that participants who express dissatisfaction with their GPs' communication effectiveness will tend to discount partnering with their doctor in their recovery. For example, participant #64, male, age 55-65, decreased his endorsement of the statement on GP-patient collaborating from moderately agree in Patient Survey 1 to slightly agree in Patient Survey 2. His ratings of his GP's communication effectiveness also went down in the same period because:

> [H]e's always busy doing something. Never been concentrated on me, comes out with short one-liners, then goes and does something else. And then comes in and offers you more pills, or goes and sees somebody else with some of this psych stuff or whatever. One comment he makes all the time and he throws at me, is "Oh you should have been dead years ago, you're lucky you're here, just keep going." I mean that's the last thing I want to hear. (male, age 55-65)

Surprisingly, he has continued to see his GP for 22 years, because “he’s the best of the worst ones I’ve come across”. This patient’s experience reflects the effects of poor interpersonal control, as defined by CAT, where he is essentially just another member of the ‘patient’ group. The power differentials between doctor and patient are reinforced in such high intergroup relationships, as explained in the communication literature review (Chapter 5). Perhaps this patient does not feel he has a strong therapeutic alliance with his doctor even after two decades because their interactions have been exchanges at the intergroup level, instead of a balance between intergroup relations and interpersonal engagement.

For patients who rate their GP-patient communication as not effective, their GPs’ accurate perception of their EMs (awareness) and/or their GPs’ sharing their EMs (concordance) could aid in depression recovery. The next chapter examines whether patients whose GPs are aware of and/or concordant with their EMs have better depression outcomes.

Ancillary findings (GP-concordance and perception of communication effectiveness).

Even as the primary analyses showed how patient EM beliefs are associated with perception of GP-patient communication effectiveness (RQ3), it was valuable to see how patient perception of GP-concordance with their EMs impacts communication perception. The free-text replies were particularly insightful on how the use of CAT strategies (or the lack thereof) influences the therapeutic alliance.

Perception of GP-concordance. The results at Time 1 were encouraging in that the majority of patients believed that their GPs shared their views about the key dimensions of their EM. Many of these patients said in their free-text responses that there had been discussion and/or they perceived empathy from their GPs regarding causation, internal locus of control, sense of agency, efficacy of non-medical therapies, and impact of depression. The responses suggest that concordance is valuable to participants from a wide range of backgrounds, not only with respect to demographic characteristics like gender and age, but also factors such as experience of trauma/loss, antidepressant attitudes, and physical co-morbidity. Perception of concordance means such patients are more likely to feel that their GPs are their allies in managing depression. This sentiment is reflected in the exemplars below:

> He does seem to understand that recent illness and loss has added to loss experienced at a very early age. He is supportive of anything I want to try because I don’t want to take anti-depressants. (causation, participant #76, female, age 55-65)
>
> She listens non-judgementally and seems understanding when sometimes my self-care decreases (internal locus of control, participant #61, female, age 18-24)

> My understanding of the physical/chemicl [sic] aspects it has come largely from my GPs, and they know and understand the emotional/historical causes" (participant #81, male, age 40-54).

The quotes above indicate presence of good emotional expression (participants #76 and #61), that is, the CAT strategy of meeting the emotional or relational needs of interactants. Patients benefit from sharing concerns with their doctor and receiving responses of empathy and understanding. Participant #81's understanding of biomedical causation from his GPs shows good interpretability, the CAT strategy which entails correctly judging a speech partner's communication competence (Watson et al., 2012) and matching it to improve comprehension.

Furthermore, responses showed that patients appreciated input from other therapists such as psychiatrists and counsellors, as well as the interaction between their GP with these professionals. However, some patients said that their GP's knowledge of their depression causation was superficial compared to the specialists, such as participant #177 (female, age 18-24) who said: "I don't feel like my GP knows all my history and symptoms because she just referred me to a psychologist and psychiatrist. But I think my psychologist is on the same page as me in terms of the origin/cause." The lack of depth in discussing causation could be due to patient preference, such as participant #150 (female, age 25-39) would discuss depression causes more with specialists, and another felt it was more beneficial to focus on symptom treatment (participant #45, female, age 18-24). Similarly, for impact of depression, a handful of patients said their doctors do not share their views because they chose not to disclose how they have been affected.

Some patients recognised that their doctors may hold different views but they appreciated having sufficient discussion or having the respect of their doctors. Participant #59 (female, age 25-39) said: "My GP prefers the biological brain chemistry explanation but also aware of extensive trauma background." Participant #104 (female, age 55-65) felt her doctor of seven years did not share her views on the effectiveness of non-medical therapies and was unsure of his views on three other EM dimensions. However, she related how her GP treats her as an individual (good interpersonal control) in an incident that happened after she obtained her doctoral degree:

> [The GP] gave me a script for something and it said Ms and then he tore it up and he went into his system and typed in Doctor. And he's the only one who's ever, it was a nice touch... He doesn't, he's never talked down to me. And I think that one little thing was the recognition that you know I had climbed, despite everything I had climbed the mountain.

However, such patients are in the minority as the majority who felt their GPs did not share their views expressed clear dissatisfaction. These results support previous research on how divergent views can lead to clashing therapies, patient misgivings about the health care system, or patient doubts over prescribed treatments (Diette & Rand, 2007). Platt and Keating (2007) said the perception gap could negatively influence treatment choices and impact outcomes. Exemplars from this current study indicate that the impact of discordance is exacerbated if doctors come across as uncaring or closed-minded. Just as appropriate emotional expression was reflected in earlier examples from patient who perceived concordance, the converse is true in that patients who perceived discordance encountered poor emotional expression, such as participant #21, #128, and #57 below:

> He just thinks I'm too fat. He's completely fixated on weight and is pretty much ignoring everything else. (causation, participant #21, female, age 25-39)
>
> I feel that they can't be bothered trying to figure it out as they probably consider me as too complex and automatically box me into a 'crazy/too complicated/damaged goods category' and without realizing, start to become patronizing/frustrated and/or judgmental. …I attempt to figure out my own treatment with my current Doctor despite it not being an ideal dynamic… I often feel I am left on the sidelines. (causation, participant #128, female, age 25-39)
>
> I believe my GP is of the opinion that I am ultimately unaffected as an individual by my condition, when often it feels like an inability to face a social setting is being blamed as laziness. (internal locus of control, participant #57, female, age 18-24)

Patients also seem to encounter poor discourse management when they think their GPs do not share their views. For example, GPs are perceived to dominate topic selection during consultations as was the experience of participant #167 (female, age 25-39), said: "I mentioned that I was seeking help from a naturopath once and she was not very encouraging on that front."

Similarly, ineffective communication is reported by patients who are unsure about GP concordance. The proportion of such patients ranged between 10 and 20 per cent depending on the EM dimension in question. It is possible patients have discussed the EM dimension with their GPs, but they do not want to presume to know what their GPs actually think. E.g. participant #132 (female, age 18-24) who said about causation: "I'm sure the doctor has their own ideas about depression, which are probably adequate for their role." However, many free-text responses showed that patients and GPs have not discussed the EM dimensions in-depth. Patients often said their GPs did not enough time or were not interested. In some instances, there was evidence of poor interpretability, such as participant #99 (female, age 55-65) said she was unsure about GP's views on sense of agency because "[h]e doesn't know me doesn't ask the right questions or review".

Blease (2014) argued strongly that it is the duty of doctors and medical researchers to carefully explain to patients about their diagnosis, particularly in depression. I would add that explanations should include discussion of the key EM dimensions listed above. To explore what GPs think of the role of EMs in depression management, results from a small GP interview study is presented in Chapter 12.

Support for hypothesis. Patients who believed their GPs shared their views (perceived EM concordance) about internal locus of control and non-medical therapies had higher ratings for GP-patient communication effectiveness in all four CAT subscales compared to patients with perceived EM discordance. This suggests that concordance is a good strategy to improve communication effectiveness. In particular, concordance in the locus of control dimension showed medium-to-moderate effect sizes. However, concordance appears to be lacking for this dimension as the proportion of patients who perceived concordance (i.e., they believed their GPs shared their views of how much their actions contributed to their depression) was lowest among the other dimensions (52.8%). At same time, the percentage of patients who perceived discordance was highest (25.8%) and the percentage of unsure responses was also the highest (21.4%). There were 31 free-text responses where participants explicitly said that they have not discussed this dimension with their doctors, and these responses were from the no/unsure group.

The lack of discussion over locus of control is of concern because meta-analysis research shows that external locus of control, that is, the perception that situations in life are outside one’s control, is positively correlated with depression (Benassi, Sweeney, & Dufour, 1988). Benassi et al. analysed 97 studies and found the average correlation coefficient to be .31 (moderate effect size). Their finding is evidence for Seligman’s theory of learned helplessness (1975) that individuals become depressed when facing events perceived as uncontrollable, which was elaborated on in Chapter 2. However, depressed individuals also tend to blame themselves for failure, as per Beck’s (1967) negative self-schemas (also reviewed in Chapter 2). The depressive paradox (Abramson & Sackeim, 1977), where self-blame and belief in uncontrollability co-exist in depressed individuals, makes it even more critical that doctors and patients actively discuss locus of control. The ideal would be patients who believe their depression is caused by situations beyond their control so that they are not burdened by self-blame, but from the point that they recognise they have depression, they partner with their GP to take back control of their lives and get better. Participant #213 (female, age 25-39) reflected this optimal balance in their free-text response about internal locus of control: “I have developed a good understanding of both the condition and of my own behaviours and exercise a high degree of accountability towards both.” In addition, if such patients have a positive outlook it would foster a stronger sense of agency as mentioned earlier in this chapter (p. 172). This in turn would be helpful to bring about change and better management of their depression.

Summary

This chapter highlighted EM statements and dimensions that had correlations with perceptions of effective GP-patient communication. In particular, the more patients endorse the importance of GP-patient collaboration for recovery, the higher their rating for communication effectiveness as reflected in the CAT subscales of emotional expression, discourse management, interpretability, and interpersonal control. Additionally, there was evidence for widespread perception of GP concordance (i.e., patients believe their GPs share their view about their depression) on the EM dimensions of causation, internal locus of control, sense of agency, efficacy of non-medical therapies, and impact of depression. Patients who perceived GP concordance for internal locus of control and efficacy of non-medical therapies reported higher ratings of GP-patient communication effectiveness than patients who did not. Studies have shown that more effective communication between doctors and patients leads to better outcomes (Stewart et al., 2000; Street et al., 2009). Therefore, to test whether actual EM concordance between GPs and patients influences depression recovery, I compared the EMs of patients with that of their doctors through a matched-pair study. The results will be presented in the next chapter.

Chapter 11: Doctors' Awareness of and Concordance with Patients' Depression Explanatory Models, and Potential Impact on Recovery

In this chapter, I present the results and discussion of a matched-pair study (*n* = 29) to address RQ4 (How aware are GPs of their patient's depression EM, how concordant are GP EMs with patient EMs, and how does awareness and/or concordance correlate to depression outcomes?). The previous study in Chapter 10 showed that patients who think their GP shares their views on certain EM dimensions rate their GP-patient communication as more effective (i.e., across a number of the CAT strategies) than patients who perceive discordance. Keeping in mind the link between doctor-patient communication and therapeutic outcomes, this matched-pair study tested for differences in depression symptom change depending on the awareness and concordance shared by the GP-patient pair. Ogden et al.'s 1999 study showed that GPs and patients often differed in their explanatory models of depression for individual patients, but there has been no study to date of the potential impact of such differences. More recently, Street and Haidet (2011), using the original CONNECT instrument, concluded that doctors are poor judges of patient health beliefs in a general outpatient clinic sample, but this work has not been replicated with GPs and depression patients to date.

Although the number of GP-patient pairs in this current study did not meet the minimum sample size derived from power calculations (refer to Chapter 7), the study provides novel insights into the extent of GP awareness and concordance. Pairs were considered aware if GP's perception of patient EM matched the patient's EM and concordant if both GP and patient had similar levels of agreement to the items in their respective EM questionnaires. In addition, the study examines the possible consequences of awareness and concordance simultaneously on recovery. This will promote a better understanding of the importance of EMs in primary care depression management.

The participant sample for this study on RQ4 differs from the sample described in Chapter 7 for RQ1-3. Therefore participant characteristics are reported first, followed by a recap of the study procedure in greater detail than the overview in Chapter 7. Results and discussion of hypothesis testing will conclude the chapter. RQ4 generated three hypotheses: Hypothesis 1 predicted better depression recovery (i.e., greater decrease in symptom severity between Time 1 and Time 2) in GP-patient pairs who were more aware compared to those who are less aware. Hypothesis 2 predicted better depression outcomes for GP-patient pairs who were more concordant compared to those who were less concordant. H1 and H2 were tests of difference in the dependent variable (depression outcome) based on different levels of a single independent variable, that is, awareness or concordance. For tests of difference using both variables of awareness and concordance, GP-patient pairs were first categorised into aware-concordant, aware-discordant, unaware-concordant, and unaware-discordant groups, and then compared with each other for differences in depression outcomes. Hypothesis 3 predicted better outcomes for aware-concordant pairs than the other pairs.

Participants

The data for this study were from 29 participants who completed Patient Survey 1 and consented to have their GPs contacted by the researcher, and whose GPs completed the GP survey online. Of the 238 participants who completed Patient Survey 1, less than a third (32.0%) shared their GP contact details. Of the 68 GPs contacted, 30 (44%) completed the online doctor survey. One GP-patient pair was excluded from analysis as the patient subsequently disclosed about having bipolar symptoms in Patient Survey 2, leaving 29 pairs.

Demographic and health characteristics of RQ4 participant sample are in Table 20. They were generally comparable to the total participant sample for RQ1, RQ2, and RQ3 in terms of relative proportions for most characteristics. For example, there were more females than males in the total sample and similarly, there were more females than males in this RQ4 sample. The only exceptions were for income and current physical health. For income, this study sample's largest group were from the low-income bracket (44.8%) whereas the total sample's largest group were middle-income participants (34.8%). For physical health, participants in poor and good health were of equal proportions in this study and they constituted the biggest group (34.5%), whereas the total sample's biggest group were participants in fair health (50.7%).

Table 20

Characteristics of Patients in Matched-Pair Study, in Actual Numbers and Percentages

Characteristics (*n* = 29 unless otherwise specified)	Number (%)
Gender	
Male	12 (41.4)
Female	17 (58.6)
Age in years	
18-24	7 (24.1)
25-39	11 (37.9)
40-54	8 (27.6)
55-65	3 (10.3)
Born in Australia	24 (82.8)
Culture/Ethnicity	
White Caucasian/European	25 (86.2)
Other	4 (13.8)
Education	
Bachelor degree or higher	19 (65.5)
Certificate/Diploma/Adv Diploma	7 (24.1)
Up to Year 12 or equivalent	3 (10.3)
Employed	15 (51.7)
Average household income	
High income	6 (20.7)
Middle income	8 (27.6)
Low income	13 (44.8)
Don’t know/prefer not to disclose	2 (6.9)
Diagnosed with depression	25 (86.2)
Earliest depression onset (*n* = 28)[a]	
≤ 1 year ago	3 (10.7)
> 1 and ≤ 5 years ago	4 (14.3)
> 5 and ≤ 10 years ago	7 (25.0)
> 10 years	14 (50.0)
Co-morbidity with mental illness	10 (34.5)
Current physical health	

Poor	10 (34.5)
Fair	9 (31.0)
Good	10 (34.5)
Impact on quality of life	
Severely	8 (27.6)
Moderately	5 (17.2)
Slightly	8 (27.6)
Not at all	8 (27.6)

Note. ^{a}Variation in *n* due to missing/unspecific responses.

GP characteristics are in Table 21. There were almost equal numbers of male and female doctors, and the sample reflected diversity in terms of practice size and type of billing. The majority of the GPs were of White Caucasian/European descent and had over 10 years of experience. Over two-thirds had completed Mental Health Training Level 1 training and almost four-fifths had experience of depressive symptoms either affecting them first-hand or someone close to them.

Table 21

Characteristics of Doctors in Matched-Pair Study, in Actual Numbers and Percentages

Characteristics (*n* = 29 unless otherwise specified)	Number (%)
Gender	
Male	15 (51.7)
Female	14 (48.3)
Born in Australia	21 (72.4)
Culture/Ethnicity	
Asian	3 (10.3)
White Caucasian/European	26 (89.7)
Length of GP experience	
≤ 10 years	3 (10.3)
>10 years	26 (89.7)
Mental Health Training (*n* = 27)[a]	
Level 1	19 (70.4)
Level 2	6 (22.2)
None/informal (e.g., up-skilling stint in psychiatric ward)	2 (7.4)
Size of practice	
Small (≤ 4 doctors)	6 (20.7)
Medium (5 to 10 doctors)	12 (41.4)
Large (>10 doctors)	11 (37.9)
Type of billing	
Bulk	9 (31.0)
Private	7 (24.1)
Mixed	13 (44.8)
Personal/up-close experience of depressive symptoms	
Yes	23 (79.3)
No	3 (10.3)
Don't think it is relevant	3 (10.3)
Personal/up-close experience of other mental health issues	
Yes	15 (51.7)
No	11 (37.9)
Don't think it is relevant	3 (10.3)

Note. [a]Variation in *n* due to missing/unspecific responses.

Procedure

As described in Chapter 7, patient EM, GP EM, and GP-perceived patient EMs were measured with respective versions of the modified depression CONNECT instrument. For example, the item on biomedical causation of depression in the patient EM version was “I have depression because something in my body is not functioning efficiently.” The corresponding item on the GP EM version was “This patient has depression because something in his/her body is not functioning efficiently” and the item on the GP-perceived patient EM version was “I think this patient thinks that he/she has depression because something in his/her body is not functioning efficiently.” Patients and doctors rated their agreement to statements using a Likert-type scale (1 = *strongly disagree*; 6 = *strongly agree*). Pairs were grouped as EM-aware if the GP-perceived patient EM score did not differ by more than two Likert scale units from the patient EM score. Pairs were grouped as EM-concordant if the GP EM score did not differ by more than two units from the patient EM score. The same cut-off was used to categorise the pairs into the four awareness-concordance groups (i.e., aware-concordant, unaware-concordant, aware-discordant, unaware-discordant).

For each of the 16 items on the modified depression CONNECT instrument, awareness was calculated by deducting the patient EM score from the GP-perceived patient EM score. Positive values mean that GPs overestimated patient endorsement of that item, while negative values reflect GPs’ underestimation of patient endorsement. The values range from zero (complete awareness, that is, no difference between patient EM and GP-perceived patient EM scores) to +/− 5 (least awareness); for example, patient strongly disagrees on an item, but their GP thinks they strongly agree for that item, or vice versa. Similarly, concordance was calculated for each of the 16 items by deducting the patient EM score from the GP EM score. Positive values mean greater GP endorsement of that item than their patients, while negative values show that lesser GP endorsement than their patients. The values range from zero (complete concordance, i.e., no difference between patient EM and GP EM scores) and +/− 5 (least concordance); for example, patient strongly disagrees with biomedical causation of their depression, but GP strongly believes that it is, or vice versa.

The measure used for depression severity was the Clinically Useful Depression Outcome Scale (CUDOS: Zimmerman et al., 2008). The possible range of CUDOS scores is 0 to 64. Patient symptom surveys were conducted six months apart. Change in depression severity (i.e., ΔCUDOS) was obtained by subtracting Time 2 scores from Time 1 scores. A positive ΔCUDOS indicates improvement, while a negative ΔCUDOS reflects deterioration in symptoms.

Analysis. Negative values for awareness and concordance were recoded into positive values so that hypothesis testing could focus on the potential impact of absolute awareness and/or concordance on depression outcomes, that is, regardless whether GPs overestimate/underestimate their patient's EM, or have greater/lesser endorsement of the EM items.

For the sake of meaningful comparisons due to the small study sample, statistical tests were performed on groups only if the levels of awareness and/or concordance contained at least five GP-patient pairs. The Kruskal-Wallis procedure tested the difference between depression outcomes for GP-patient pairs according to their levels of awareness or concordance. Statistically significant ($p < .05$) or near statistically significant results ($p < .10$) were followed up with the Mann-Whitney U test to determine the direction of difference (i.e., which group recovered more). The Dunn-Bonferroni correction was applied where appropriate. As for testing the levels of awareness-concordance, the initial plan was to compare the four awareness-concordance groups of GP-patient pairs with each other on their ΔCUDOS using the Kruskal-Wallis test for each of the 16 items. However, some groups did not have the minimum number of GP-patient pairs ($n \geq 5$) so these were excluded from analysis. Where only two-way comparisons were possible, the Mann-Whitney U test was used instead.

Results

Absolute EM awareness or concordance. Table 22 shows the number of GP-patient pairs in the aware/unaware and concordant/discordant groups for the 16 modified depression CONNECT instrument items. Overall, the majority of pairs in the study sample were EM aware (86%) and EM concordant (91%). The items with the largest proportions of EM aware and EM concordant pairs were "Depression plays a major role", and "Cooperation with GP is key to recovery." The item with the biggest proportion of EM unaware and EM discordant pairs was the belief that patient choices led to depression (internal locus of control).

Table 22

GP-Patient Pairs in Concordant/Non-Concordant and Aware/Unaware Groups, in Actual Numbers and Percentages

EM items (according to survey order)	Aware	Unaware	Concordant	Discordant
Biomedical causation of depression	25 (86.2)	4 (13.8)	27 (93.1)	2 (6.9)
Psychosocial causation	22 (75.9)	7 (24.1)	22 (75.9)	7 (24.1)
Patient choices led to depression (internal locus of control)	17 (58.6)	12 (41.4)	18 (62.1)	11 (37.9)
Right action will reduce symptoms (sense of agency)	25 (86.2)	4 (13.8)	28 (96.6)	1 (3.4)
Belief in complementary medicines	22 (78.6)	6 (21.4)	24 (82.8)	5 (17.2)
Belief in complementary activities	26 (89.7)	3 (10.3)	27 (93.1)	2 (6.9)
Belief in non-doctor remedies	24 (82.8)	5 (17.2)	26 (89.7)	3 (10.3)
Belief in alternative providers	27 (93.1)	2 (6.9)	26 (89.7)	3 (10.3)
Depression has changed priorities	26 (92.9)	2 (7.1)	26 (89.7)	3 (10.3)
Depression plays a major role	28 (96.6)	1 (3.4)	29 (100)	0
Depression changes one's future	26 (92.9)	2 (7.1)	25 (89.3)	3 (10.7)
Depression can have positive outcome	21 (75.0)	7 (25.0)	26 (89.7)	3 (10.3)
Antidepressants are necessary	27 (93.1)	2 (6.9)	27 (93.1)	2 (6.9)
Antidepressants: more harm than good	24 (85.7)	4 (14.3)	24 (82.8)	5 (17.2)
Some GPs are too pro-antidepressants[a]	25 (92.6)	2 (7.4)	20 (74.1)	7 (25.9)
Cooperation with GP key to recovery	28 (96.6)	1 (3.4)	29 (100)	0

Note. n = 28 or 29 unless specified otherwise. [a]*n* = 27.

Direction of discordance. Items with over 20% absolute discordance (i.e., psychosocial causation, internal locus of control, and some GPs being too pro-antidepressants) were noteworthy as it translates to at least one in five GPs holding different beliefs than their patients. The direction of discordance was further examined by looking at the original concordance scores (i.e., any score ranging between −5 and +5) to determine whether it was GPs who had greater/lesser endorsement than their patients, or the reverse. The frequencies of negative scores (lesser GP endorsement than patient) and positive scores (greater GP endorsement than patient) for these three items are presented in Table 23.

Table 23

GP-Patient Pairs with Less, Equal, or Greater GP Endorsement Compared to Patients, in Actual Numbers And Percentages

EM items	GP EM score < patient EM score	GP EM score = patient EM score	GP EM score > patient EM score
Psychosocial causation	6 (20.7)	11 (37.9)	12 (41.4)
Patient choices led to depression (internal locus of control)	16 (55.2)	7 (24.1)	6 (20.6)
Some GPs are too pro-antidepressants[a]	4 (14.8)	6 (22.2)	17 (63.0)

Note. n = 29 unless specified otherwise. [a]*n* = 27.

In slightly more than two-fifths (41.4%) of the GP-patient pairs, the GPs believed more firmly than their patient's that this depression episode had psychosocial causes. Half of that figure believed the reverse. In more than half (55.2%) of the pairs, the patients believed more firmly than the GPs that their choices were responsible for their depression. In contrast, only 20.6% of pairs reported the GPs blaming the patients more than the patients blamed themselves. Almost two-thirds of GP-patient pairs (63%) had GPs endorsing the belief that some GPs are too pro-antidepressants more than their patients. Less than 15% of the pairs had patients endorsing this item more strongly than their GPs.

Testing H1 and H2. As mentioned earlier, owing to the small sample size, statistically significant results were not expected. However, the Kruskal-Wallis test for the differences in the distribution of ΔCUDOS across EM awareness levels for the biomedical causation item approached significance, χ^2 (4, N = 29) = 8.29, *p* = .081. Follow-up test was conducted between levels with at least five pairs each. There were eight pairs in the Level 0 group (no difference between GP-perceived patient EM score and patient EM score) and 13 pairs in the Level 1 group (GP-perceived patient EM score differed from patient EM score by one unit of the Likert scale). The other levels did not have the minimum number of pairs ($n \geq 5$). The Level 0 group (*Mdn* = 4.5, mean rank = 14.1) reported slight improvement in depression symptoms whereas the Level 1 group reported slight deterioration (*Mdn* = -2.0, mean rank = 9.1), Mann-Whitney *U* = 27.5. The result approached statistical significance, *p* = .075, and had a moderate-to-strong effect-size (*r* = -.39).

Kruskal-Wallis tests showed that the distribution of ΔCUDOS was statistically significantly different across levels of EM concordance for the sense of agency item (i.e., the belief that the patient can get better by taking the right actions), χ^2 (3, N = 29) = 8.95, *p* = .030. Follow-up tests were performed for the nine pairs in the Level 0 group (no difference between GP EM score and patient EM score) and the 15 pairs in the Level 1 group (GP EM score and patient EM score differ by one unit of the Likert scale). The other levels had less than five pairs each. The Level 0 group (*Mdn* = -5.0, mean rank = 8.4) reported slight deterioration in depression symptoms, whereas the Level 1 group reported slight improvement (*Mdn* = 5.0, mean rank = 15.0), *U* = 30.5, *p* = .027, effect-size *r* = .45. On closer examination, patients in 11 of the 15 pairs in Level 1 group believed more strongly than their GP that they have the power to alleviate their symptoms (sense of agency).

Testing H3. The number of GP-patient pairs in the four categories of aware-concordant, aware-discordant, unaware-concordant, and unaware-discordant, for each of the 16 items are in Table 24.

Table 24

Number of GP-Patient Pairs in the Awareness-Concordance Categories

EM items	Aware-Concordant	Aware-Discordant	Unaware-Concordant	Unaware-Discordant
Biomedical causation of depression	24	1	3	1
Psychosocial causation	21	1	1	6
Patient choices led to depression (internal locus of control)	15	2	3	9
Right action will reduce symptoms (sense of agency)	24	1	4	0
Belief in complementary medicines[a]	20	2	3	3
Belief in complementary activities	26	0	1	2
Belief in non-doctor remedies	24	0	2	3
Belief in alternative providers	26	1	0	2
Depression has changed priorities[a]	24	2	1	1
Depression plays a major role	28	0	1	0
Depression changes one's future[b]	24	1	1	1
Depression can have positive outcome[a]	20	1	5	2
Antidepressants are necessary	27	0	0	2
Antidepressants: more harm than good[a]	21	3	2	2
Some GPs are too pro-antidepressants[c]	19	5	0	2
Cooperation with GP key to recovery	28	0	1	0

Note. $n = 29$ unless specified otherwise. [a]$n = 28$, [b]$n = 27$, [c]$n = 26$.

The majority of items reported 20 or more pairs in the aware-concordant group. As such only four items had other groups containing five or more GP-patients pairs. The items pertained to belief in psychosocial causation for depression, belief in internal locus of control (i.e., patient choices led to depression), belief that depression can have positive outcomes, and belief that some GPs are too pro-antidepressants. For each of these items, the Mann-Whitney U test compared the aware-concordant pairs and pairs that were either aware-discordant, unaware-concordant, or unaware-discordant. The results are presented in Table 25.

Table 25

Depression Outcomes in Aware-Concordant (AC) Pairs versus Unaware-Concordant/Aware-Discordant/Unaware-Discordant Pairs

EM items	Median change in ΔCUDOS[a]		*p*	Effect-size *r*
	Aware-concordant (AC)	Not aware-concordant		
Psychosocial causation (AC vs. unaware-discordant)	4.0	6.5	.50	-.13
Patient choices led to depression/internal locus of control (AC vs. unaware-discordant)	1.0	8.0	.22	-.25
Depression can have positive outcome (AC vs. unaware-concordant)	3.5	4.0	.66	-.09
Some GPs are too pro-antidepressants (AC vs. aware-discordant)	4.0	-3.0	.39	-.17

Note. [a] ΔCUDOS equals Patient Survey 1 CUDOS score minus Patient Survey 2 CUDOS score. Positive ΔCUDOS indicate improvement, negative ΔCUDOS reflect deterioration.

Again as expected, there were no statistically significant ($p < .05$) results. Effect sizes were generally small. However, the results suggest that variation in awareness-concordance levels of GP-patient pairs could have different impacts on recovery depending on the EM item. For the belief that some GPs are too pro-medication, aware-concordant pairs saw improvement while unaware-discordant pairs reported deterioration. In contrast, for the belief that patient choices led to depression (internal locus of control), unaware-discordant pairs recovered much more than aware-concordant pairs. Although not significant, the direction of this result was unexpected. For the items on psychosocial causation and the belief that depression can have positive outcomes, the difference in the extent of recovery was minimal, that is, both groups still saw improvement, but the aware-concordant group saw less decrease in symptoms (i.e., smaller positive ΔCUDOS).

Discussion

Research Question 4 had two aims: first, to obtain some indication, using a subset of the current sample, of GPs awareness of their patient's EM for depression and how concordant are GP EMs with patient EMs. Descriptive results indicated high levels of awareness and concordance for the 16 modified depression CONNECT item in the 29 GP-patient pairs recruited, with only a fair degree of discordance for three of the items.

The second aim (using a subset of the current sample) was to obtain some indication about whether depression recovery differs depending on levels of EM awareness and/or EM concordance within GP-patient pairs. Hypothesis 1 predicted that pairs with greater levels of EM awareness would report a greater decrease in symptom severity compared to pairs with lower levels of EM awareness. There was limited support for this hypothesis as there were no statistically significant results ($p < .05$). The only result approaching statistical significance was for the EM item on biomedical causation ($p = 0.81$). Further testing showed that pairs with complete EM awareness (i.e., no difference between GP-perceived patient EM score and patient EM score) saw a slight improvement in depression symptoms, while pairs with a slight difference (one Likert scale unit out of possible five) reported slight deterioration. This result also approached significance ($p = .075$). Hypothesis 2's prediction that pairs with greater levels of concordance would see a greater decrease in symptom severity was not supported. However, there was a statistically significant finding ($p < .05$) in the opposite direction. GP-patient pairs who were completely concordant (i.e., no difference between GP EM score and patient EM score) for the sense of agency item reported slight symptom deterioration while pairs that differed by one Likert scale unit showed slight improvement. This finding was unexpected. Testing for Hypothesis 3 yielded no statistically significant results but results seem to suggest that the interaction between GP-patient EM awareness-concordance and depression outcomes is more complex than previously thought. The possible reasons and implications of these results are discussed in the following section.

GP awareness of patient EM. The high levels of awareness could have been due to length of time that the patients have been treated by their GP. This variable was not measured in the study this potential explanation could not be tested. However, there was anecdotal evidence from several GPs I interviewed for a separate qualitative study (presented in the next chapter). These

GPs said time is needed to build rapport and to understand their patients' perspectives. One GP said rapport is particularly important in cross-cultural general practice because patients often do not divulge everything upfront.

> [I]t takes some time to actually build that rapport to be able to get to understand and similar to being cross cultural trying to understand my cultural interpretation on their cultural interpretation of depression…An example a guy I saw yesterday, a Korean fellow who it's now the sixth time that I've seen him that I've shifted my view that his depression is possibly more related to a social anxiety disorder than it is a true depression. And so therefore my model is actually changing with him because of actually a greater understanding of things that he is divulging. (GP #2, male, 28 years' experience)

It is also true that high levels of EM awareness can help build rapport. Further research can illuminate how these two factors mutually reinforce each other to improve depression management in primary care.

Discordance between GP EM and patient EM. The pattern of discordance for the items of psychosocial causation, internal locus of control, and belief that some GPs are pro-antidepressant were interesting to note for further research. The preponderance of GPs believing in psychosocial causation and the greater level of endorsement about the pro-antidepressant tendencies of their fellow GPs were surprising considering previous studies that attest to the biomedical bias of doctors (Andersson et al., 2001; Thomas-Maclean & Stoppard, 2004). The reasons could have been related to the work and personal experience of the RQ4 sample GPs. Almost 90% had over 10 years of GP experience and close to 80% had experienced depression either first-hand or in someone close to them. As such these GPs were attuned to the myriad of psychosocial factors implicated in depression and they could have had direct encounters with doctors who were too quick to prescribe antidepressants.

The discordance for the internal locus of control item was because nearly three times as many patients assigned greater blame on themselves for their depression compared to their GPs. This could have been due to the confounding effects of self-stigma, which includes self-blame over the inability to cope (see Chapter 4). Future research on the relationship between self-blame and belief in internal locus of control could illuminate how they mutually reinforce each other so that such unhelpful beliefs can be better addressed.

Awareness and depression outcomes. The lack of statistically significant results ($p < .05$) was probably due to the small sample size. However, the near-significant result for biomedical causation belief does suggest that even relatively small inaccuracy in GP perception of patient EM can influence depression outcomes, particularly for aetiology. This is because beliefs about depression causation are arguably foundational to developing individually tailored management strategies. The experience of Participant #49 (male, age 18-24) gives an idea of how beliefs about causation prompted him to make certain changes such as taking time off from school, work, and other commitments. He said the original trigger for his depression was not having a single day off for four months. His GP's awareness of his beliefs helped as he was able to obtain a medical certificate for two weeks, even though he did not think that his doctor shared the same beliefs. He said: "I think he thought about it in terms of medicine, in terms of chemical imbalances and stuff, and I saw it more from the external triggers…he gave me the certificate, but he still felt it was more chemical."

Studies have shown that patients' beliefs about the cause of their depression can influence help-seeking, treatment preferences and outcomes, coping strategies, and treatment compliance (Bann et al., 2004; Brown et al., 2007; Fernandez Y Garcia, Franks, Jerant, Bell, & Kravitz, 2011). In particular, disagreement about biomedical causation of depression was associated with rejection of a depression diagnosis (Van Voorhees et al., 2005), while agreement over biological causes of depression correlated with belief in the efficacy of medication (Budd, James, & Hughes, 2008). Therefore, future studies with greater numbers of GP-patient pairs are needed to increase understanding of how GP awareness of patient causation beliefs aids in depression recovery.

Concordance and depression outcomes. The unexpected finding from testing H2 where GP-patient pairs with slight discordance in beliefs about patient agency did better than perfectly concordant pairs could have been due to several reasons. The first could be that the pairs were concordant because the patient has been going to the same GP for many years. This could breed familiarity and limit how much the patient recovers. The patient surveys did not include this information, so its potential as a confound could not be verified. However, the following quote from participant #11 (female, age 55-65) who had perfect concordance with her GP on the item for sense of agency, shows that over-familiarity can be a problem:

> I'd have to say my GP for six years – and confidentially, I think he stopped listening to me. I think he sees my face, oh here she comes, the depressed person is here. I think he has just stopped, to the extent that he hasn't diagnosed pre-diabetes that I have. I had to go to another GP to get that…He was very supportive initially because when I came to [name of university] I had a lot of issues and now that I'm better I just think he's not listening. I mean that's, I still think he's a good doctor and he's very kind and he has been very supportive but I think he just sees me, oh another middle-aged depressed woman.

Another reason why the Level 1 group patients (*n* = 15) fared better overall could have been because 11 patients in the group believed more strongly than their GPs that they had the power to alleviate their symptoms. This indicates that that patient beliefs about their own agency could be a key factor in recovery and GPs would do well to nurture this sense of agency, or at least not inadvertently dampen it when they personally are less convinced than their patients that their patients can get better by taking the right actions.

Awareness-concordance and depression outcomes. The majority of pairs were aware-concordant resulting in a ceiling effect of sorts. This could be due to self-selection bias where patients and doctors who consented to participate were more likely to be on the same page with each other, especially if they have had a long association. This can be avoided in future studies with a more targeted recruitment of patients who are newly diagnosed with depression, and their GPs. In Williams and Healy's (2001) study at a community mental health service in North Wales, new referrals with no known history of mental health issues were interviewed before and after their first appointment. The study reported that patients had a range of perceived causes for their depression and that these beliefs were fluid. Therefore, such patients would be more likely to have different EMs from their doctors. Recruiting them for future studies would enable further insight into the impact of variation in EM awareness and EM concordance between GPs and patients.

Nonetheless this current study's results suggest that awareness and concordance could have differing impacts on recovery depending on the EM belief. For certain key topics, awareness and concordance are needed to maximise recovery from depression. The symptom deterioration of patients in unaware-discordant pairs for the belief that some doctors are too pro-medication indicates that candid and thorough discussions about antidepressants are needed. Perhaps the use of effective communication strategies outlined in earlier chapters could help patients and their doctors be on the same page. The results for the items on psychosocial causation and belief that depression can have positive outcomes, where median ΔCUDOS scores did not differ markedly between the groups being compared, imply that awareness-concordance could be less important factors for those EM beliefs. For the belief that patient choices led to depression/internal locus of control, the results were contrary to expectations in that unaware-discordant pairs did better than aware-concordant pairs. This is possibly because unawareness on the part of GPs for these beliefs somehow has some benefit, just as discordance has benefits as explained earlier. Further research is needed to understand what these benefits could be as it goes beyond the scope of this small study.

Summary

This chapter has provided some insight into the potential impact of EM awareness and concordance in GP-patient pairs on depression recovery. It appears that there could be different optimal levels of awareness and concordance that varies depending on the EM belief. The results were not generalisable due to the small sample size and limited numbers of unaware and/or discordant pairs but they indicate possible directions for future research. One possible area of research is to explore GPs' views on the importance of EM awareness and concordance. This was undertaken in a small interview study and the results will be presented in the next chapter.

Chapter 12: General Practitioners' Views on Role of Explanatory Models in Depression Management

In this chapter, I present the qualitative results of an interview study on the importance of primary care doctors understanding their patients' explanatory models (EMs) of their depression. The findings for Research Question 3 (reported in Chapter 10 indicated that patients who perceived concordance with their GPs over certain EM beliefs rated their GP-patient communication as more effective. In other words, these patients feel their doctors do understand their perspective. The other side of the coin is to ask whether GPs think patient EMs play a role in depression management and how GPs practically communicate about EMs. Additionally, the pattern of results from Research Question 4 (reported in Chapter 11) point to the possibility that EM awareness and concordance between GPs and patients can affect depression recovery. It would be important to understand whether GPs believe that knowing patient EMs would help improve outcomes. As such, the three questions for this interview study were labelled (a) to (c) to distinguish them from the overarching RQs for the entire thesis:

(a) Do GPs think it is important to know patients' EMs for their depression?
(b) What do GPs discuss with patients that pertain to the patient's depression EM?
(c) Do GPs believe that knowing their patients' EM helps improve treatment outcomes?

The data-mining program Leximancer (more details in the next section) is used to analyse the interviews for findings related to these questions.

Methods

Participants. I recruited 10 GPs by convenience sampling using flyers (see Appendix K) and word-of-mouth. There were six female and four male participants. Most of the doctors had between 20 to 30 years' of experience, except for the youngest participant, who had been a GP for about three years.

Data collection. I used a semi-structured interview guide consisting of open-ended questions about topics relevant to the RQs listed above. The full question set is in Appendix Q. Sample questions relating to RQ (a) were "When you diagnose and treat someone with depression, how important is it for you to know whether the patient has an explanatory model?" and "How much effort do you put into discussing with your patient about the cause and prognosis of their depression?." For RQ (b), doctors were asked "How much variation is there in how you explain a patient's depression?." They were also asked to comment on whether they would use four of the eight suggested questions proposed by Kleinman et al. (1978) to elicit patient EMs. These had been modified to my study on depression, and were as follows:

i. What do you think caused your depression?

ii. How has depression affected your life?

iii. Is there anything you can do to alleviate it?

iv. What kinds of treatment do you think would be beneficial for you?

v. What are the preferred outcomes you hope to achieve in the next three months?

For RQ (c), sample questions were "To what extent does knowing your patient's EM influence how you talk to that patient?" and "In your view, how does understanding your patient's EM of depression affect your patient's recovery?." At the end of the interview, GPs were also asked if they wanted to discuss any topic that had not been raised. Eight of the doctors were interviewed one-on-one and in person as they were based in Brisbane. The other two were phone interviews with GPs based in other Australian cities. The average length of interviews was 29 minutes (range 21-44 minutes). The interviews were digitally recorded and transcribed verbatim.

Analysis. As mentioned earlier, interview transcripts were analysed using Leximancer 4.0 (see Chapter 7). Compared to manual coding, Leximancer is much faster and can handle high volumes of textual information. The 10 interviews in this current study did not generate a large amount of text but Leximancer was employed for the added benefit of limiting bias that can arise from researcher-driven analyses (Watson, Smith & Watter, 2005). Leximancer has been validated (Penn-Edwards, 2010; Smith & Humphreys, 2006), and used in mental health research. For example, one study showed that carers of people with schizophrenia employ varied strategies to adjust to the latter's behaviour (Cretchley, Gallois, Chenery, & Smith, 2010). Another study by Watson et al. (2014) used Leximancer examined how doctors, nurses, and allied health professionals viewed the topic of time in clinical handovers. Similarly, this current study used Leximancer to analyse how GPs view the subject of patient EMs in depression management. However, Watson et al. noted some limitations to using Leximancer; for example, most of the concepts that emerge would be nouns. As such, the output may not be as relevant when the research question is about a process, such as RQ (b). Another limitation that Watson et al. pointed out is that Leximancer takes a literal approach that is only able to identify what occurs in the data, but it cannot help explain the absence of a concept.

Processing stages. Before uploading the transcripts to Leximancer Version 4, dialogue tags were added to identify the speakers. There was minimal intervention with the data set (i.e., default/automatic settings were employed as much as possible) to allow existing concepts to emerge from the data naturally. However, the following adjustments were necessary:

1. Text processing options (under Generate Concept Seeds): turn off auto-paragraphing, merge word variants (e.g., talk and talking) and apply dialogue tags

2. Concept seeds (under Generate Thesaurus):
   a. Unmerge depressants from depressed, depressing, depression, and depressive, then exclude depressants as an automatic concept seed because review of excerpts to understand meaning of concepts shows that the term always occurs as antidepressants
   b. Unmerge medical from medication and medications, and merge the latter two back together because the text excerpts showed that medical was used differently than the other two terms.
3. Concept coding settings (under Run Project): Words of researcher were excluded using the Kill Concepts function
4. Theme size was adjusted to 40% instead of the default 33% to reduce the list of themes to less than 10 (this enables a more in-depth discussion of the themes)

Results and Discussion

The findings from Leximancer and specific quotes from doctors that pertain to the three RQs are reported below. The reasons for and implications of these results will also be discussed in the same section.

RQ (a): GP views on the importance of patient EMs. Figure 4 shows the map of themes identified by Leximancer from the interview transcripts. Concept names are excluded for the sake of legibility. As explained earlier, the more important themes are shown in warmer colours, while the less important themes are in cooler colours. The themes by order of importance are depression (red), patient (brown), people (light-green), work (green), different (dark-green), person (blue), question (indigo), and family (purple). The red colour of the depression theme was expected considering the interviews were about depression.

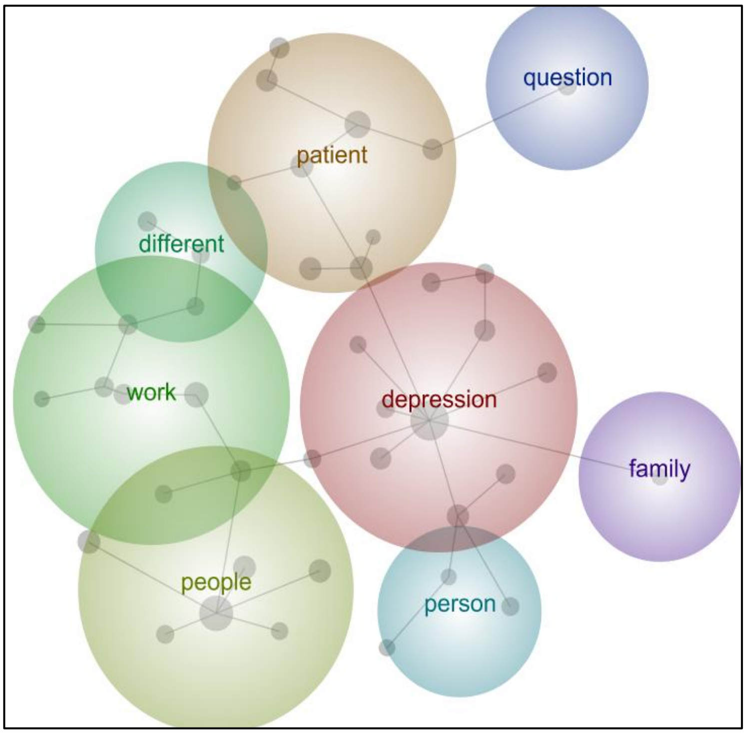


Figure 4. Leximancer map of themes in GP interviews on the role of patient explanatory models

Figure 5 shows the same concept map but with concept names included. It shows the concepts of explanatory, model, patient, and important linked by a single line, which indicates their relationship to each other. Their inclusion in the second-most important theme of patient shows their relative importance compared to other concepts. Most of the analysis for RQ (a) focused on the patient theme because the concepts within the theme (listed alphabetically here: explanatory, important, medical, model, patient, perspective, probably, understand, and use) are highly relevant to how GPs answered the questions about patient EMs.

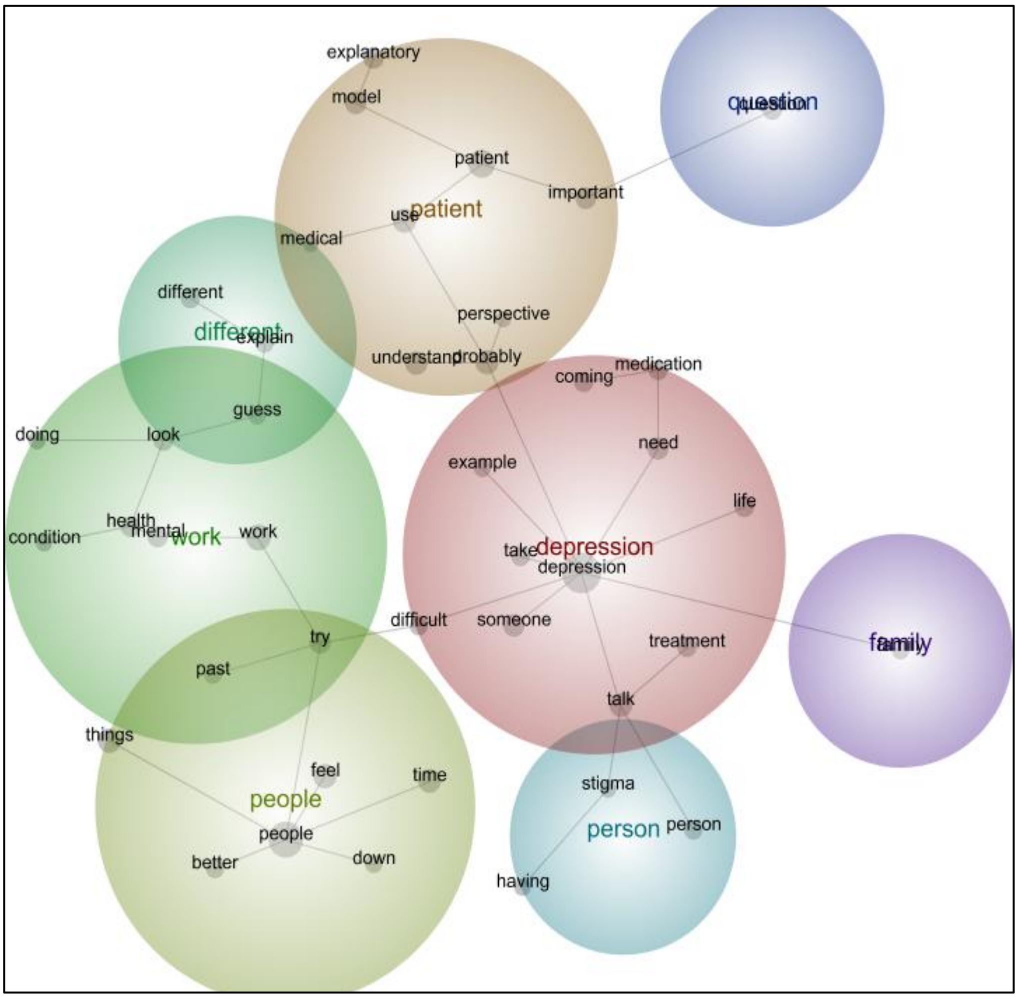


Figure 5. Leximancer map of concepts in GP interviews on the role of patient explanatory models

A manual review of the transcripts showed that seven of the 10 GPs accorded importance to establishing whether patients have EMs or pre-existing notions about their depression. The manual method was necessary because a review of all Leximancer textual excerpts associated with concepts of explanatory and model did not reflect quotes from all the GPs. This indicated that the software did not capture all GP responses about patient perspectives as its approach is too literal, a limitation of the method as mentioned earlier. GP #7 (two year's experience) explained understanding patient EM serves as a crucial starting point:

> [W]ith patients or people in general, they can have a very different view as lots of things impact on their view of depression. And just even the knowledge of whether they think they're depressed or not is important because if you can clearly see that they are depressed, but they don't see it, yeah, then you sort of go "Well okay, what do you think depression is?" So yeah, definitely important.

The importance placed on how patients perceive their condition is shown by GP #10's practice of writing down exactly what patients said and later transfer it to case notes on the computer, so she could refer to it in future.

Besides being a starting point, several GPs also said that knowing patient EM was key to treatment. E.g. Both GP #1 and GP#8 said it would change their approach and treatment, while GP #5 (25 years' experience) said:

> So I listen to their story and what's going on. And that involves their perceptions of what's going on. And I often ask them why they are there and what they want because sometimes that's the only way you work it out.

The quote by GP #5 implicitly refers to patient EM without using the actual term explanatory model. This could explain why there are no other lines connecting explanatory to any other concept besides model, and model is only connected to patient. The Leximancer output showed that the concept explanatory was mentioned 49 times across the 10 transcripts, far fewer times than the most frequent concept depression, which occurred 237 times. Similarly, depression occurred four times more frequently than the concept model, which was identified 58 times. The term EM was not frequently used by GPs to discuss aspects of primary care depression, possibly due to lack of familiarity with the term.

In addition, several GPs' responses seem to suggest possible reasons for why patient EMs do not feature prominently when GPs discuss depression management. As mentioned earlier, Leximancer cannot highlight absent concepts, and as such the plausible reasons for why some GPs may not use patient EMs were manually inferred from reading the transcripts. The first reason is that a doctor's more biomedical EM gets prioritised in an attempt to defuse patient stigma. GP #4 (25 years' experience) described such encounters:

Another reason is the lack of reflexivity on the part of GPs, which leads to them operating under assumptions. GP #1, who has been in general practice since 1990, said:

> We are very poor judges of what we do…I probably don't really have a sense of the patient's explanatory model of depression often. I know all sorts of things but I suppose I don't always find out exactly why they think they have depression. You know it's interesting – it's easy to make assumptions actually.

Another doctor, GP #9 (25 years' experience), candidly said that doctors are largely to blame for ignoring patient EMs:

> [A] large part of it is our fault of course as doctors. We sort of carry around these sort of dual sets of depression as an illness and we've got these behavioural explanations and biochemical explanations to a sense and then we sort of pull them out depending on which

> one we think is most appropriate to the patient and it's much more likely that we apply that to the patient without asking the patient straight up what they think their explanatory was, what their explanatory model is.

The final reason GPs may not delve deeply into patient perspectives is because they are focused on whether such discussions fulfil a therapeutic function in their opinion. For example, GP #9 only brings up EMs so patients can better appreciate that what he is suggesting, whether be it pharmacotherapy or psychotherapy, would help alleviate their depression. It serves to motivate them towards treatment adherence. He said, "I rarely ask patients directly their explanatory framework…Part of being successful in helping the patient is motivating the patient to undertake what I think is going to be needed to be done." This could explain why, in the previous study for RQ4 (Chapter 11), there was more recovery in unaware-discordant or unaware-concordant GP-patient pairs than aware-concordant pairs for certain EM items. The GPs in those pairs may have discerned correctly what interventions their patients needed, without having to know their existing beliefs for those EM dimensions.

RQ (b): Process of eliciting EMs. The results for this RQ were obtained from manual readings of the transcripts. The GPs generally felt that the questions suggested by Kleinman et al. (1978) would be helpful. Some topics would be used more consistently than others, for example, the GPs said they would almost always ask about past coping strategies. GP #10 (at least 20 years' experience) elaborated that she asks what has worked because she wants the patient to have one or two things in mind that they can do in the meantime before their next appointment. Several GPs noted that it was important to have open phrasing of questions and to be careful with the choice of words. GP #2 said he tends to avoid using the word depression:

> I would be saying "What do you think has made you feel the way that you are feeling at the moment." So leave it more as personal interpretation to them. Because for some people the word depression is a label. It can become a barrier.

Given that the doctors interviewed were mostly experienced GPs, many of them would have likely described themselves as GP #2 (28 years' experience) did; he considered himself "unconsciously competent" because he does not think about the process of communicating about depression. When asked if training would benefit GPs on knowing how to elicit patient EMs, GP #8 (at least 20 years' experience) said:

> I think we all just do a lot of our own reading, keeping up with different models of care, looking at different medications, but I think for something like depression and anxiety, the most important thing is how you take a history and how you connect with the patient, you know, and how, what relationship you have, and then how you treat them. I think that's the

> most important thing and I don't know whether going off and doing training is going to make any difference. Maybe 20 years ago, when I first started, but once …when you've been doing it for awhile, I mean you develop a certain style and technique and you know what works, and you know if your patient is coming back, if your patients aren't coming back.

She added that for any training to be useful, it would have to be in relation to building the therapeutic alliance, such as developing the skill of empathy.

GP #6 (32 years' experience) had an instructive method for eliciting patient perspectives in which he maps anyone with mental health problems on a hand-sketched 3-D graph (represented in Figure 6) during his consultations. One axis represents circumstances such as job loss, relationship breakdown; the second represents personality strengths and weaknesses that one is born with, and the last axis is for medical conditions that can mimic depression or exacerbate depression. For example, if the doctor is aware of the possibility of low iron levels or an underactive thyroid in the patient, there would be tests run to check

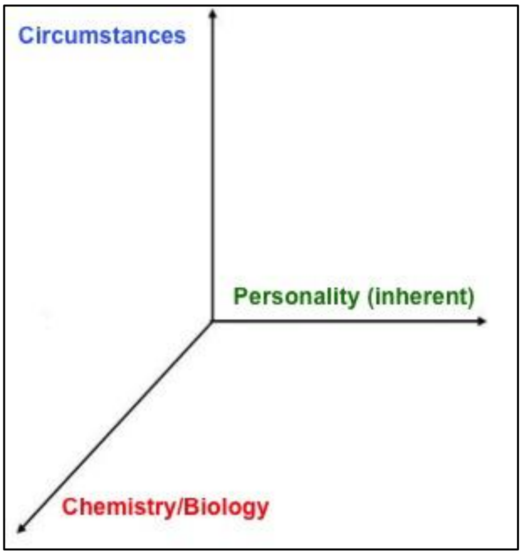


Figure 6. Graph drawn by doctor (GP #6) when discussing mental health issue with patients

GP #6 said the graph is useful because three axes give a good handle on managing depression. For example, looking at how contributing circumstances can be modified such as a Centrelink referral for someone who is jobless, or what can be done about to resolve a patient's marital issues. As for personality, he said:

> [I]t's kind of partly a learned attitude and partly a personality trait that makes you look at the world and see the glass as half empty rather than the glass is half full... It doesn't necessarily

> make it right or wrong but it does mean if you have an attitude to life like that well then it's probably going to be more likely that depression is something that you're going to have to live with. And just recognising that that is the case and saying well okay that's you…is an important thing.

For those whose responses to circumstances and personality are disproportionate, such as feeling despondent and mentally reliving a failed exam repeatedly six months after it occurs, the intensity of their response indicates a problem that could be biochemical in nature. GP #6 thinks such individuals should be considered for antidepressant treatment.

Arguably, the three dimensions in GP #6's map are useful to apply Engel's biopsychosocial model (briefly mentioned in Chapter 2) to depression. Epstein (2014, p. 275) praised Engel's biopsychosocial model, calling it a passionate, "simultaneously scientific and humanistic", protest against the reductionist approaches that were increasingly distancing clinical care and research from the patient's subjective experience. However, even though empirical evidence continues to mount for the connection between patient lived experience and health outcomes, Epstein said it is still a challenge for clinicians to be practically biopsychosocial. Indeed, the biopsychosocial model has been criticised for being too general (Vögele, 2015) and Holmes (2000) asserts that biopsychosocial psychiatry is only an ideal that Western-trained doctors pay lip service to. Vögele added that although behavioural medicine tries to include biopsychosocial aspects in research and treatment, it can be hard to derive specific practical strategies at the individual/clinical level. GP #6's method could be one such specific strategy of discussing depression with patients. Should future research yield evidence of its effectiveness, it could be more widely applied in primary care and even specialist settings.

RQ (c): Impact of knowing patient EM: Manual reading of the transcripts showed that all but one GP said that having an accurate perception of their patients' EMs can help improve recovery. For example, GP #1 said it is important for GPs to understand patient perspectives but argued that having the same views is not a necessity, and could even be harmful:

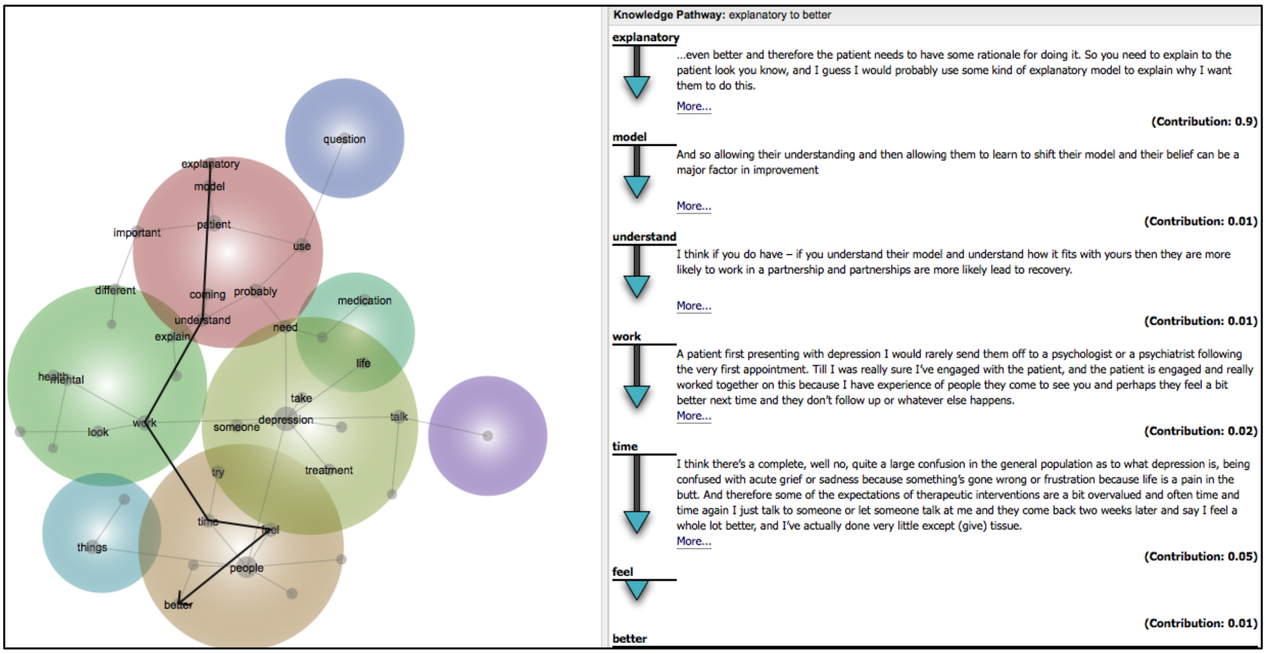
> I certainly think a feeling of being understood is enormously powerful. You know no matter how it comes... I think the feeling of connecting and being listened to incredibly powerful. Do I have to have the same model as them? I'm not quite sure about that. I don't know, I think perhaps I would very often have a slightly different model and I suppose I tend to think that can open up other possible avenues of being and recovering if the model is extended...sometimes a blinkered kind of model, a uni-dimensional model is perhaps the least helpful. I think if both the therapist and the patient had a, say, very medication-oriented model I suppose that would worry me slightly if anything.

She elaborated that only using medication would be like “putting all your eggs in one basket”, which is generally not helpful for conditions as complex as mental illness. However, she recognised that having completely contradictory models would be problematic too:

> The best balance somewhere in between with a therapist that feels okay about gently challenging some of the patient’s models particularly when it is a model that is not working. If someone is coming to you for help generally speaking, generally speaking they need some reframing, some other possibilities.

The sole doctor who contradicted the trend was GP #3, who felt it is more important for patients to understand for themselves what is happening to them, more so than for the doctors to have an accurate perception.

Leximancer’s toggle pathway mode function was used to further explore how GPs link patient EMs with recovery. Under this mode, when two concepts are selected, the map will show the most probable pathway between them (Leximancer, 2011). The concept used for patient EM was ‘explanatory’ and the concept used for recovery was ‘better’ in the analysis shown in Figure 7. The black line shows the possible concepts linking ‘explanatory’ and ‘better’, and the probability of each path segment is on the right panel (in parentheses). The panel also shows textual excerpts for the concepts along the pathway. The excerpts for the explanatory-better pathway are expanded below to give a clearer picture of how GPs connect EMs with patient recovery.



Knowledge Pathway: explanatory to better

explanatory

…even better and therefore the patient needs to have some rationale for doing it. So you need to explain to the patient look you know, and I guess I would probably use some kind of explanatory model to explain why I want them to do this.

More…

(Contribution: 0.9)

model

And so allowing their understanding and then allowing them to learn to shift their model and their belief can be a major factor in improvement

More…

(Contribution: 0.01)

understand

I think if you do have – if you understand their model and understand how it fits with yours then they are more likely to work in a partnership and partnerships are more likely lead to recovery.

More…

(Contribution: 0.01)

work

A patient first presenting with depression I would rarely send them off to a psychologist or a psychiatrist following the very first appointment. Till I was really sure I've engaged with the patient, and the patient is engaged and really worked together on this because I have experience of people they come to see you and perhaps they feel a bit better next time and they don't follow up or whatever else happens.

More…

(Contribution: 0.02)

time

I think there's a complete, well no, quite a large confusion in the general population as to what depression is, being confused with acute grief or sadness because something's gone wrong or frustration because life is a pain in the butt. And therefore some of the expectations of therapeutic interventions are a bit overvalued and often time and time again I just talk to someone or let someone talk at me and they come back two weeks later and say I feel a whole lot better, and I've actually done very little except (give) tissue.

More…

(Contribution: 0.05)

feel

(Contribution: 0.01)

better

Figure 7. Pathway from the concept explanatory to the concept better

The first excerpt had a high probability contribution (.90), which meant it was highly relevant. It pertained to the concept of 'explanatory' and was from GP #9:

> …the patient needs to have some rationale for doing it...I guess I would probably use some kind of explanatory model to explain why I want them to do this. So if I want them to take an antidepressant I might say look you know one of the ways of thinking about depression is that it's possibly a relative lack of serotonin as a neurotransmitter and whilst that's probably an over-simplification, one way we can think about how these medications work is that they increase the amount of serotonin for your brain to use for example. So yes I have provided a framework, I'm hoping that that is a motivator for them to take the medication.

The second excerpt (associated with the concept model) is from GP #2 who believes it is important to know patient EMs because they may contain harmful notions that need to be changed:

> I think sometimes their explanatory model needs correction, clarification. Sometimes – someone I saw yesterday that has severe postnatal depression after rape, working through their explanatory model is a huge deal of guilt and working with them to reframe that it's not actually them who has done the wrong thing. And so allowing their understanding and then allowing them to learn to shift their model and their belief can be a major factor in improvement.

Admittedly, the rape example is extreme, but more generally GP #2 believed in the value of understanding his patients' EMs. His following quote is associated with the concept understand along the pathway in Figure 7: "[I]f you understand their model and understand how it fits with yours then they are more likely to work in a partnership, and partnerships are more likely lead to recovery." This relates back to the RQ3 study findings (Chapter 10) that showed how preference for partnership is associated with higher ratings of GP-patient communication effectiveness, which in turn results in better outcomes.

Good communication can often be a prelude to recovery, as was the experience of GP #3 (35 years' experience), whose quote below is associated with the concept of time in Figure 7. He said in many cases, letting a patient talk about their condition was all it took to help them feel better:

> Some of the expectations of therapeutic interventions are a bit overvalued and often time and time again I just talk to someone or let someone talk at me and they come back two weeks later and say I feel a whole lot better, and I've actually done very little except [give] tissue.

In other words, the act of GPs eliciting EMs from patients could benefit the patients simply for the cathartic effect and the feeling of being listened to. This is also a possible reason why the non-aware-concordant GP-patient pairs in the previous study saw more recovery than aware-concordant pairs, that is, the patients in the former group could have benefited more from their GPs being good listeners than their GPs knowing or sharing their views. Future research is needed to verify this. If true, it is further evidence of the importance of communication skills such as attentive listening (which is part of the CAT strategy of discourse management as discussed in Chapter 5).

Discussing EMs can also help certain patients with self-stigma, said GP #6, which in turn helps with recovery. He said:

> There are some people who are chronically depressed every day of their life and may need treatment every day of their life, which sounds awful. It sounds like…I'm inadequate I can't cope. It's kind of the unwritten message. What I say there is I say okay let's just say you have diabetes and your body is not making something. It's not making insulin so you replace it. Does that mean you are an inadequate person because you need to replace something your body is not making? Let's talk about this organ instead of, the head as opposed to the pancreas where insulin is made. And I find that that kind of explanation although it's simplistic it helps them to, it takes the stigma and the pain out of it. And I find that really helps them.

This is in line with the RQ2 study (Chapter 9) that reported help-seeking inhibition self-stigma is lower with higher endorsement of biomedical causation for depression.

Summary

This chapter has presented results from a small interview study with 10 GPs. Leximancer analysis helped to answer RQ (a) on whether GPs view patient EMs as important and RQ (c) on how knowing patient EM can improve recovery. The sample of GPs interviewed mostly expressed their belief that patient EMs are important but it is unclear whether they consistently discuss it. The findings for RQ (b) showed that GPs adopted different methods of eliciting patient EMs. Future studies could possibly tease out the practical aspects of how to prioritise patient EMs since the act of doing so can be beneficial. Improved outcomes can arise from catharsis (possibly contingent on the GP's listening skills), better treatment adherence from understanding how medications/therapy works, and reduced patient self-stigma in a help-seeking context. Chapter 13 brings together the broader implications of the results from the RQ1-RQ4 studies are discussed, along with strengths, limitations, and direction of future research.

Chapter 13: Conclusion

In 1999 Alistair Wright, editor of the British Journal of General Practice, wrote that despite decades of depression research "we still 'see through a glass darkly'" (p. 91) when it comes to understanding the condition. In response to its complex nature he called for a variety of approaches to tackle depression – a situation that is as true today as it was almost two decades ago. This thesis did not present an approach, instead it explored the utility of a meta-approach. That is, not merely attempting to see or illuminate what depression is or is not, but in the first place recognising that we use different lenses to see. These lenses are analogous to Kleinman et al.'s (1978) explanatory model (EM), which formed the theoretical framework for this thesis. The primary aim of this thesis was to gain a better understanding about the role of Ems in the primary care management of depression. In particular I was interested in the potential interactions between EMs and two important factors that impact depression outcomes, that is, patient-stigma and GP-patient communication effectiveness. This chapter summarises the main findings from the present program of research, as well as their implications for understanding and treating depression. The limitations are highlighted and future research is proposed.

Key Findings

This thesis had four research questions, as outlined in Chapter 1. They were:

RQ1. What are depression EMs of primary care patients and do these change over time?

RQ2. Does high endorsement of biomedical causation for depression correlate with low patient self-stigma?

RQ3. Does agreement to certain aspects of patient EMs correlate with patient perception of greater GP-patient communication effectiveness?

RQ4. How aware are GPs of their patient's depression EM, how concordant are GP EMs with patient EMs, and how does awareness and/or concordance correlate to depression outcomes?

I now discuss the main findings of each RQ and what the results mean for primary care depression management. This is followed by a discussion of the GP interview study findings, which arose from further investigation of RQ3 and RQ4.

RQ1: Acknowledge multifaceted aspects of patient EMs. In contrast to studies on depression EMs that examine the general population's perspectives (see Chapter 3), the study for RQ1 focused on the EMs adopted by people with lived experience of depression or depressive symptoms. This increases its construct validity as previous studies have shown that beliefs about depression differs markedly between people who have recovered from depression and those who have never experienced depression (Furnham & Kuyken, 1991; Kirk, Haaga, Solomon, & Brody, 2000). For example, Furnham and Kuyken concluded that participants who self-reported as having depression considered interpersonal problems as more of a cause for depression than participants who had no experience of depression. Researchers also found that people taking antidepressants believe differently about the causes of their own depression compared to depression as a general condition (Read, Cartwright, Gibson, Shiels, & Magliano, 2015). Read et al.'s large-scale study (*n* = 1,829) in New Zealand noted clear differences in the substance, form, and endorsement levels of causal beliefs regarding one's personal experience of depression compared to the same sample's beliefs about depression in general. This underscores the value of the RQ1 study findings that asked depression patients for their beliefs about their depression.

Use of the 16-item modified depression CONNECT instrument provided insights into varied facets of patient EMs, including beliefs about causation, treatment, and significance of depression. Depending on the item in question the extent of agreement differed according to gender; age; culture; educational level; length of depression; presence of depression diagnosis; presence of psychiatric co-morbidity; and current physical health. In short, almost all the demographic and health characteristics of participants seemed to have a bearing on various aspects of patient EM in one way or another. These findings are in line with Read et al.'s (2015) study that also reported significant variation between depression causal beliefs of demographic groupings. The implication of the RQ1 study is not that GPs need to be aware of how patient EMs could differ depending on these many characteristics, but to keep in mind that depression EMs can and do vary. The authors (Haidet et al., 2008) of the original CONNECT instrument have generally recommended using the instrument to improve clinical practice. My study has shown that a derivative of the CONNECT instrument can indeed capture salient perspectives about the depression experience. Further studies to refine the instrument could enhance its suitability to be used in GP clinics in future.

In addition, the RQ1 study showed that most EM beliefs were relatively stable within a period of six months, with the notable exception of two items: biomedical causation and the belief that depression played a major role in their life. Endorsement of these two items decreased between Patient Survey 1 and Patient Survey 2. These RQ1 study results support the findings of previous interview studies that reported the fluidity of depression patients' perspectives (e.g., Petersen & Madsen, 2017; Williams & Healy, 2001).

RQ2: Consider impact of belief in biomedical causation on self-stigma. The biomedical explanation for depression is thought to have reduced some public stigma (Goldstein & Rosselli, 2003), although some researchers have challenged this (e.g., Jorm & Griffiths, 2008; Jorm & Oh, 2009; Rusch, Kanter, & Brondino, 2009). Much less known is the association between belief in biomedical causation for depression and self-stigma of depression patients. The RQ2 study highlighted how different aspects of self-stigma (i.e., alienation, discrimination experience, help-seeking inhibition, and social withdrawal) correlate with belief in biomedical causation for depression. High belief in biomedical causation was correlated with high alienation and low help-seeking inhibition simultaneously. This contributes to a more nuanced understanding of the mechanisms behind patient self-stigma. The RQ2 study also found that participants who consistently endorsed the biomedical causation belief did not see a reduction in any of the four self-stigma subscales.

The small effect size and opposing direction of the correlations at Time 1 and the finding that Time 2 self-stigma did not decrease all point to how the biomedical explanation of depression causation does not seem to have the purported benefits of reducing stigma. The finding that it could carry some risk of alienation means that GPs should consider moving away from a reductionist biomedical EM when explaining depressive illness to patients.

RQ3: Encourage patient beliefs associated with effective GP-patient communication. When Haidet et al. (2008) introduced the CONNECT instrument, they speculated that such measurements could help to improve communication within medical encounters. Using the modified depression CONNECT instrument, the RQ3 study examined the relationship between patient EMs and GP-patient communication effectiveness. Combining EM theory and communication accommodation theory (CAT: Gallois et al., 2005; Giles, 1973) was a novel approach. As it was necessarily exploratory in nature, the generalisability of RQ3 results is yet to be determined in future studies. Nonetheless, patterns emerged indicating that patients' beliefs about biomedical causation, the necessity of antidepressants, and the harmful nature of antidepressants could impact on patients' rating of their GP-patient communication effectiveness. Evidence for effective communication was evaluated in terms of CAT strategies, namely emotional expression, discourse management, interpretability, and interpersonal control. Moreover, correlational trends suggest that patients having a strong sense of agency and believing that depression can lead to desirable outcomes such as greater empathy could positively influence GP-patient communication. Besides these trends the RQ3 study found a strong positive correlation between belief in GP-patient collaboration as key to recovery and patient ratings of GP-patient communication effectiveness. Future research to determine causation would underscore the importance of perspective taking in clinical practice. Larger samples of GP-patient pairs can also examine whether CAT strategies can help patients and GPs to maintain a strong therapeutic alliance despite having different EMs.

The patient surveys also asked whether patients believed that their GPs shared their views about their condition. Patients who perceived GP concordance for beliefs on internal locus of control and efficacy of non-medical therapies rated their GP-patient communication as more effective than patients who did not. As far as I am aware, this is the first time that the patient perceptions of their doctors' subjective beliefs about the patient's depression have been examined, albeit in a very basic manner. Recent examples of patient perception studies have focused on perception of doctor communication skills in general (Kenny et al., 2010) or of the doctor-patient interaction in cancer (Meggiolaro et al., 2016). The results for perceived GP concordance in this current work suggest this line of inquiry has much potential for meaningful research. It is likely that the findings and implications of such research would be particularly far-reaching for depression and other forms of mental illness due to their highly subjective nature. Patients' perspectives of their GPs' depression beliefs could be captured by creating a fourth version of the modified depression CONNECT instrument, that is, in addition to patient EM, GP EM, and GP-perceived patient EM surveys that were used in this thesis. A sample statement on the patient-perceived GP EM survey could read "I think my doctor thinks that I have developed depression because something in my body is not functioning efficiently." Future studies could also examine CAT surveys of GP's perception of GP-patient communication effectiveness. It was not within the scope of this thesis to examine doctors' ratings of CAT strategies, but it should be a topic of further research. Just as communication has often been described as a two-way street, perception about communication is also a two-way, dynamic process.

RQ4: GPs should know patient EMs but they do not need to share the same views. It is insufficient to study depression EMs and to understand their importance solely from the perspective of depression patients. As Kleinman et al. (1978) noted, patient and doctor EMs should both be examined so that major differences in perspectives can be resolved for the sake of effective clinical practice. Studies of either patient or doctor EMs are relatively common but dyadic studies such as the RQ4 study are more rare.

In contrast to Street and Haidet's findings (2011), which showed that GPs were inaccurate with perceiving patient beliefs, the RQ4 study showed high levels of EM awareness by the GP. This study's high levels of alignment could have been the result of how long these patients have been with their respective GPs.

The RQ4 study's results were mostly not statistically significant due to the small sample size. However, one result nearing significance indicated that GP awareness of the extent that patients endorse biomedical causation could help improve depression outcomes. Surprisingly, there were no similar results linking GP EM concordance and better depression recovery. Instead, one statistically significant result showed that GP-patient pairs who were slightly EM discordant for sense of agency (patient and GP EM Likert rating differed by one unit in either direction) reported greater improvement in symptoms after six months. This could have been because the majority of patients in this group believed more strongly than their GPs that they can get better by taking the right actions. The sense of agency could have propelled such patients to try more ways to get better, resulting in the improved outcomes. Future research with larger sample sizes would be required to test this proposition.

On the surface, the overall lack of evidence for recovery of concordant GP-patient pairs seems to contradict the point made earlier that patient-perceived GP concordance (i.e., patients thinking that their GPs share their views) could help improve GP-patient communication effectiveness, which in turn leads to better outcomes. I propose the following explanation for the apparent contradiction: GPs do not have to actually share the views of their patients in order to improve communication and outcomes. However, it is important for patients to perceive that their GP shares their views because this helps the patients feel that they have a valuable ally in their battle against depression. For GPs who hold different views from their patients about their depression, the perception divide does not have to undermine the therapeutic alliance. I would argue that the alliance can be strong and beneficial if doctors intentionally and effectively communicate empathy using the CAT strategies outlined in earlier chapters. Empathy is not an innate attribute that one either has or does not have – it can be a learned skill and it is key that doctors learn it. At least two GPs interviewed for this thesis expressed this opinion, and it was particularly heartening that one of them is in-charge of statewide training of other GPs. Of course GPs can only be intentional about bridging perception gaps and communicating to their patients that they are ‘on the same page’ if they are cognisant of the patient’s EM, which underscores once again the importance of EM awareness.

It is not only the GPs who need to be proactive and intentional – patients should also do the same, as best they can considering the energy-draining symptoms of depression. Street and Haidet (2011) found that the GPs judged more accurately the beliefs of patients whom they considered proactive. Future studies could examine how patients with depression can be more proactive and utilise CAT strategies to have better communication with their doctors.

Interview RQs: GPs say patient EMs are important but admit potential inconsistency in eliciting them. The GP interview study provided richer insights into how GPs perceived the role of EMs in depression management. Most of the 10 GPs who participated said it was important to elicit patient EMs, which was an encouraging qualitative finding considering the results from the quantitative surveys. However, several GPs expressed that they may not elicit their patients’ EMs as often and as consistently as they should. The interviews thus provided a good opportunity for the doctors in the sample, 90% of whom were very experienced in general practice, to be reflexive about how they approach patients with depression.

The practical methods that GPs use to elicit patient EMs for depression would benefit from future studies. One possible study design would be to videotape actual consultations between pairs of GPs and patients, and to analyse them for ways to elicit patient EM beliefs. GPs and patients could then be interviewed to distil which questions, analogies, or deliberate promptings were particularly useful in helping facilitate a thorough discussion of the patient EM. The consultations could also be analysed to determine whether CAT strategies (including approximation, which could not be studied in this thesis) were being employed consciously or unconsciously.

So far I have highlighted key findings and implications of my studies. In the next section I will evaluate my studies' methodological strengths and limitations.

Strengths and Limitations

The studies for RQ1 to RQ4 benefited from some key strengths in terms of study design. However, there were certain design limitations in each of the study, partly due to my inexperience as a researcher and also the lack of financial resources to have more extensive piloting and recruitment.

Strengths. These strengths were derived from having clear goals and research questions from the outset of the thesis project. In addition, the availability of online survey tools enabled data collection from a sample population that can be difficult to reach due to mental illness stigma.

Balance of theoretical and empirical approach. Various theories that have significant empirical standing formed the basis of this thesis. The central theoretical framework of Kleinman's EM approach was linked to the relatively well-documented phenomenon of self-stigma for RQ2, a new approach that yielded meaningful results. EMs were also examined in conjunction with CAT strategies and this fresh approach produced clear indications of how to focus future efforts for improving GP-patient communication effectiveness.

Involvement of actual depression patients and their doctors. The patient sample was recruited based on self-reports of experiencing depression, which improves the generalisability of the findings to other groups of depressed individuals. The analysis for RQ4 was conducted on patients with recent contact with their GPs, so GPs felt able to answer survey questions about their patients' depression. Having participants who have experienced depression recently improves construct validity, as explained in Chapter 6.

Use of both quantitative and qualitative methods. While this thesis mainly focused on questionnaire surveys of patients and doctors, efforts were made to collect qualitative data from participants. The free-text questions in the surveys and semi-structured interviews of both patients and doctors helped provide a rich output of results and painted a clear picture of the role of EMs in primary care depression management. The involvement of actual patients and doctors, and the combination of quantitative and qualitative methods should be retained for future studies on depression EMs.

Consideration of impact on patients. The self-stigma instrument contained nine reverse-coded items to reduce the risk of patients feeling overwhelmed by negativity when completing the survey. The number of such items was higher than the original Internal Self-Stigma of Mental Illness instrument's stigma resistance subscale, which had five items (Ritsher et al., 2003).

Researcher's first-hand experience of depressive episodes. As mentioned in the prologue, I have experienced depressive episodes as part of my bipolar disorder diagnosis. This gave me an advantage in relating to the patients that I interviewed for this thesis. Interviewer-respondent rapport plays a key role in ensuring that the respondent completes the interview and feels comfortable enough to answer potentially personal questions (Carley-Baxter, 2008).

Limitations. Most of the limitations involved methodological oversight for the instruments used in the studies or in the collection of demographic information. These limitations impacted upon data analysis and reduced the range of statistical analyses that were possible. Most analysis in this thesis involved non-parametric methods that are less powerful in detecting true differences that exist (Pallant, 2013). These issues can be prevented through a more thorough and systematic piloting process in the future.

Explanatory model instrument

Use of individual Likert scale items instead of subscales. The exclusion of items that were considered repetitive, which was an outcome of the piloting process, resulted in the modified depression CONNECT instrument being analysed as a collection of individual statements instead of subscales. This was either because there were not enough remaining items to make up the subscales, or the subscales had unacceptably low Cronbach alphas. Subscales are preferable because they can be tested for reliability and the data produced is in continuous form, allowing for more sophisticated statistical analysis.

Belief in biomedical and psychosocial causation was not framed exclusively. As much of depression discourse centres around biomedical versus psychosocial causation, the respective items in the EM surveys could have been combined into a single item. Participants would have to choose one or the other, thereby giving a clearer picture of the extent of biomedical or psychosocial causation belief among primary care depression patients to better determine the impact of causal beliefs on self-stigma and communication effectiveness. Admittedly, this would have forced a dichotomy but it could have been mediated by allowing participants to provide free-text responses. Having said that, the existing response items did allow participants to show varying levels of agreement with both models. Given the complexity of depression, it is reasonable to assume that some people could simultaneously hold strong biomedical and psychosocial understandings of depression.

Self-stigma survey

Missing important subscales. Some aspects of self-stigma that have been previously linked to causal belief, such as feelings of being at fault for one's condition (Schreiber & Hartrick, 2002), could not be studied due to inadequate internal reliability. The problem could have been avoided by using either the Depression Self-Stigma Scale (DSSS: Kanter et al., 2008) or the Self-Stigma of Depression Scale (SSDS: Barney et al., 2010) instead of combining items from the SSDS with the ISMI (Ritsher et al., 2003). The rationale was to retain the content validity of the SSDS because it had been developed in Australia from people with direct or indirect experience of depression while incorporating the psychometrically superior ISMI (Stevelink et al., 2012). However the resulting instrument was too long and in eliminating items to make it of optimal length, several important subscales (i.e., self-blame and stereotype endorsement) had to be dropped from analysis due to low Cronbach alphas. The self-stigma subscale of self-blame in particular could have yielded stronger correlations with the biomedical causation belief if it had been included.

Communication effectiveness survey

Ceiling effect. A large proportion of patients reflected maximum scores on the CAT survey for GP-patient communication effectiveness. It was especially pronounced for the interpersonal control subscale, where more than a third of the participants (35.3%) scored six. Vogt (2005) explained that a ceiling effect presents a problem in data analysis because the degree of true variation in the data is not captured by the instrument scores.

Health demographics

Missing question for length of treatment by GP. Patients were not asked how long they had been with the same GP. Some respondents answered in the free-text responses, but this was not sufficient to determine whether treatment length was a confounding variable for RQ3 and RQ4. Participants who have been with their GPs longer would be inclined to rate their GP-patient communication effectiveness as high, which could have led to a response bias.

Sample size and potential bias

The findings from this program of research have limited generalisability due to size of the convenience samples relative to the number of variables in the questionnaires. There could also have been response bias; for example, participants who answered the online questionnatire after reading a social media post about the study are not representative of the depressed population in Australia, some of whom do not have easy Internet access. However, there was a range of recruitment methods to counter this and the exploratory findings reported in this thesis can be used to develop future studies. More time and resources will also need to be allocated towards recruiting doctors to participate.

Future research

The next study to follow up on the findings from this thesis should determine how the various factors of depression EM beliefs, self-stigma level, and GP-patient communication effectiveness interact. It would be interesting to determine how these factors, which were studied in paired combinations (e.g., biomedical causation belief and self-stigma, or patient EMs and GP-patient communication effectiveness) would simultaneously affect depression recovery outcomes. Answering this question is contingent upon whether strong correlations are found between the factors, as this would allow for a plausible model to be tested. As explained in the limitations section, there are definite ways to improve future studies and with a larger sample size, the possibility of getting stronger correlations is high.

In addition, there should be greater focus on the use of qualitative methods in future studies, given that the interviews conducted for this thesis has yielded numerous useful insights. Thematic analysis and other more exhaustive analysis methods will be particularly helpful in gaining a broader understanding of depression EMs in primary care.

Conclusion

The quest to understand depression has been recorded in antiquity and continues unabated today due to depression’s prevalence and complexity. A cursory online search of latest peer-reviewed journal articles published in the same month as the writing of this chapter with the keyword depression runs the gamut from a systematic review on depression and ceramides (a kind of waxy lipid molecules in cell membranes) (Dinoff, Herrmann, & Lanctôt, 2017), to a study on collectivism as a protective factor for depression (Knyazev, Kuznetsova, Savostyanov, & Dorosheva, 2017). In the midst of such diverse and new lines of research, the consensus remains that stigma reduction can improve detection of depression and promote help-seeking, leading to improved outcomes. We also know that good therapeutic alliance built on effective doctor-patient communication aids with depression management. The present study contributes to the body of knowledge about the link between EMs and patient self-stigma and GP-patient communication effectiveness. Standalone biomedical causation for depression should be avoided due to its potential to increase alienation self-stigma. Acknowledgement of patient EMs by GPs is key to satisfying GP-patient communication.

The focus on Kleinman's EM framework is aligned with calls to go beyond symptoms in the primary care management of mental illness in recent years (Lynch, Askew, Mitchell, & Hegarty, 2012). Lynch and colleagues asserted that the core values of GPs impact what is considered important when assessing their patients, which influences the process and content of assessment. However, they argued that these values are currently ill-defined and consequently, the current default primary care scenario is over-reliant on the psychiatric model of treatment. As a result, it constrains diagnoses and management strategies to be solely based on symptom criteria. Although they were writing about psychological distress in general, Lynch et al.'s observations are highly applicable to depression, as it is the most common mental illness handled by GPs (Harman et al., 2006; Katon & Schulberg, 1992). Instead of only focusing on symptoms, depression management needs to take into account how patients perceive their condition and whether their GP are aware of their beliefs. Adopting Kleinman's EM approach consistently, such as through combining the use of an EM-based questionnaire like the CONNECT instrument and ensuring that there is adequate discussion of patient EMs during the consultation, could be an important first step in what Lynch et al. argue for in their article, that is, the redefining of primary care mental health assessment priorities, process, and content. These conversations should be promoted globally and involve GPs and medical researchers, and I would add that they must involve patients themselves. Depression will ever remain a lonely path to walk, but if GPs can come alongside at strategic points, the rest stops being analogous for the clinical consultations, this could stand the patient in good stead to continue their individual journeys to recovery.

References

Åberg-Wistedt, A. (1989). The antidepressant effects of 5-HT uptake inhibitors. *The British Journal of Psychiatry, 155* (Suppl. 8), 32-40. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/2692638

Abolghasemi, A., Gholami, H., Narimani, M., & Gamji, M. (2015). The effect of Beck's cognitive therapy and mindfulness-based cognitive therapy on sociotropic and autonomous personality styles in patients with depression. *Iranian Journal of Psychiatry and Behavioral Sciences*, *9*(4), doi:10.17795/ijpbs-3665

Abramson, L. Y., & Sackeim, H. A. (1977). A paradox in depression: Uncontrollability and self-blame. *Psychological Bulletin, 84*(5), 838- 851. doi:10.1037/0033-2909.84.5.838

Abramson, L. Y., Seligman, M. E., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology*, *87*(1), 49-74. doi:10.1037/0021-843X.87.1.49

Acharya, D. R., Thomas, M., & Cann, R. (2016). Validation of a questionnaire to measure sexual health knowledge and understanding (Sexual Health Questionnaire) in Nepalese secondary school: A psychometric process. *Journal of Education and Health Promotion*, *5*, 18. doi:10.4103/2277-9531.184560

Ackerman, S. J., & Hilsenroth, M. J. (2003). A review of therapist characteristics and techniques positively impacting the therapeutic alliance. *Clinical Psychology Review, 23*, 1-33. doi:10.1016/S0272-7358(02)00146-0

Adams, A., Realpe, A., Vail, L., Buckingham, C.D., Erby, L.H., & Roter, D. (2015). How doctors' communication style and race concordance influence African-Caribbean patients when disclosing depression. *Patient Education and Counseling, 98*(10), 1266-1273. doi:10.1016/j.pec.2015.08.019

Ahn, W., Proctor, C. C., & Flanagan, E. H. (2009). Mental health clinicians' beliefs about the biological, psychological, and environmental bases of mental disorders. *Cognitive Science, 33*, 147-182. doi:10.1111/j.1551-6709.2009.01008.x

Albaum, G. (1997). The Likert scale revisited: An alternate version. *Journal of the Market Research Society*, *39*(2), 331-332. Retrieved from https://numerons.files.wordpress.com/2012/04/36-the-likert-scale-revisited.pdf

Alemi, Q., Weller, S. C., Montgomery, S., & James, S. (2016). Afghan refugee explanatory models of depression: Exploring core cultural beliefs and gender variations. *Medical Anthropology Quarterly*, 1-21. doi:10.1111/maq.12296

Allen, E. I., & Seaman, C. A. (2007). Likert scales and data analyses. *Quality Progress, 40*(7), 64. Retrieved from http://rube.asq.org/quality-progress/2007/07/statistics/likert-scales-and-data-analyses.html

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Andersson, S. J., Troein, M., & Lindberg, G. (2001). Conceptions of depressive disorder and its treatment among 17 Swedish GPs: A qualitative interview study. *Family Practice, 18*(1), 64-70. doi:10.1093/fampra/18.1.64

Andrews, L. (2010). *Encyclopedia of depression.* Santa Barbara, CA: Greenwood Press.

Andrews, G., Issakidis, C., & Carter. (2001). Shortfall in mental health service utilisation. *The British Journal of Psychiatry, 179*, 417-425. doi:10.1192/bjp.179.5.417

Andrews, G., Slade, T., & Peters, L. (1999). Classification in psychiatry: ICD-10 versus DSM-IV. *The British Journal of Psychiatry, 174*, 3-5. doi:10.1192/bjp.174.1.3

Angermeyer, M. C., Holzinger, A., Carta, M. G., & Schomerus, G. (2011). Biogenetic explanations and public acceptance of mental illness: Systematic review of population studies. *The British Journal of Psychiatry, 199*, 367-372. doi:10.1192/bjp.bp.110.085563

Angermeyer, M. C., Holzinger, A., & Matschinger, H. (2009). Mental health literacy and attitude towards people with mental illness: A trend analysis based on population surveys in the eastern part of Germany. *European Psychiatry, 24*(4), 225-232. doi:10.1016/j.eurpsy.2008.06.010

Ardito, R., & Rabellino, D. (2011). Therapeutic alliance and outcome of psychotherapy: Historical excursus, measurements, and prospects for research. *Frontiers in Psychology, 2,* Article 270. doi:10.3389/fpsyg.2011.00270

Arora, N. K. (2003). Interacting with cancer patients: The significance of physicians' communication behavior. *Social Science & Medicine, 57*(5), 791-806. doi:10.1016/S0277-9536(02)00449-5

Australian Bureau of Statistics. (2007). *National Survey of Mental Health and Wellbeing: Summary of Results*. Canberra: Author.

Australian Bureau of Statistics. (2012). *Australian Health Survey 2011-2012*. Retrieved from http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/4364.0.55.001Chapter1202011-12

Australian Bureau of Statistics. (2013a). *Household income and income distribution, Australia, 2011-12.* Retrieved from http://www.abs.gov.au/AUSSTATS/abs@.nsf/Previousproducts/6523.0Main%20Features22011-12?opendocument&tabname=Summary&prodno=6523.0&issue=2011-12&num=&view=

Australian Bureau of Statistics (2013b). *Measures of Australia's progress*, cat. no. 1370.0. Retrieved from http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/1370.0main+features292013

Australian Bureau of Statistics. (2017a). *Migration Australia, 2015-2016*. Retrieved from www.abs.gov.au/ausstats/abs@.nsf/mf/3412.0

Australian Bureau of Statistics. (2017b). *Census of population and housing: Australia revealed, 2016,* cat. no. 2024.0. Retrieved from http://www.abs.gov.au/ausstats/abs@.nsf/Latestproducts/2024.0Main%20Features22016?opendocument&tabname=Summary&prodno=2024.0&issue=2016&num=&view=

Australian Department of Health. (2012). *Better access to mental health care: Fact sheet for patients*. Retrieved from http://www.health.gov.au/internet/main/publishing.nsf/Content/0F792912834609B4CA257BF0001B74FA/$File/patients2.pdf

Australian Department of Social Services. (2011). *The people of Australia – Australia's multicultural policy*. Retrieved from https://www.dss.gov.au/sites/default/files/documents/12_2013/people-of-australia-multicultural-policy-booklet.pdf

Babbie, E. R. (2015). The practice of social research (14th ed.). Boston, MA: Cengage Learning.

Baker, S., Gallois, C., Drieger, S. M., & Santesso, N. (2011). Communication accommodation and managing musculoskeletal disorders: Doctors' and patients' perspectives. *Health Communication, 26(4)*, 379-388. doi:10.1080/10410236.2010.551583.

Baker, B. O., Hardyck, C. D., & Petrinovich, L. F. (1966). Weak measurements vs. strong statistics: An empirical critique of S. S. Stevens' proscriptions on statistics. *Educational and Psychological Measurement, 26*(2), 291-309. doi:10.1177/001316446602600204

Baker, S. C., & Watson, B. M. (2015). How patients perceive their doctors’ communication: Implications for patient willingness to communicate. *Journal of Language and Social Psychology, 34*, 621-639. doi:10.1177/0261927X15587015

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, *84*, 191-215. doi:10.1037/0033-295X.84.2.191

Banerjee, S., & Varma, R. P. (2013). Factors affecting non-adherence among patients diagnosed with unipolar depression in a psychiatric department of a tertiary hospital in Kolkata, India. *Depression Research and Treatment*, *2013*, 1-12. doi:10.1155/2013/809542

Bann, C., Parker, C., Bradwejn, J., Davidson, J., Vitiello, B., & Gadde, K. (2004). Assessing patient beliefs in a clinical trial of Hypericum perforatum in major depression. *Depression and Anxiety, 20*(3), 114-122. doi:10.1002/da.20036

Barbui, C., Cipriani, A., Patel, V., Ayuso-Mateos, J., & Van Ommeren, M. (2011). Efficacy of antidepressants and benzodiazepines in minor depression: Systematic review and meta-analysis. *The British Journal of Psychiatry, 198*(1), 11-16. doi:10.1192/bjp.bp.109.076448

Barney, L. J., Griffiths, K. M., Christensen, H., & Jorm, A. F. (2010). The Self-Stigma of Depression Scale (SSDS): Development and psychometric evaluation of a new instrument. *International Journal of Methods in Psychiatric Research, 19*(4), 243-254. doi:10.1002/mpr.325

Baudry, A., Mouillet-Richard, S., Launay, J.-M., & Kellermann, O. (2011). New views on antidepressant action. *Current Opinion in Neurobiology*, *21*(6), 858-865. doi:10.1016/j.conb.2011.03.005

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects*. New York: Harper and Row.

Beck, A. T. (1972). *Depression: Causes and Treatment.* Philadelphia: University of Pennsylvania Press.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

Beck, C. S., Benitez, J. L., Edwards, A., Olson, A., Pai, A., & Torres, M. B. (2004). Enacting health communication: The field of health communication as constructed through publication in scholarly journals. *Health Communication, 16*(4), 475-492. doi:10.1207/s15327027hc1604_5

Beidas, R. S., Stewart, R. E., Walsh, L., Lucas, S., Downey, M. M., Jackson, K., … Mandell, D. S. (2015). Free, brief, and validated: Standardized instruments for low-resource mental health settings. *Cognitive and Behavioral Practice*, *22*(1), 5-19. doi:10.1016/j.cbpra.2014.02.002

Beisecker, A. E. (1990). Patient power in doctor-patient communication: What do we know? *Health Communication, 2*(2), 105-122, doi:10.1207/s15327027hc0202_4

Beisecker, A. E., & Beisecker, T. D. (1990). Patient information-seeking behaviors when communicating with doctors. *Medical Care, 28*(1), 19-28. doi:10.1097/00005650-199001000-00004

Benassi, V., Sweeney, P., & Dufour, C. (1988). Is there a relation between locus of control orientation and depression? *Journal of Abnormal Psychology, 97*(3), 357-367. doi:10.1037/0021-843X.97.3.357

Ben-Porath, D. (2002). Stigmatization of individuals who receive psychotherapy: An interaction between help-seeking behavior and the presence of depression. *Journal of Social and Clinical Psychology, 21*, 400-413. doi:10.1521/jscp.21.4.400.22594

Berant, E., Mikulincer, M., & Shaver, P. (2008). Mothers' attachment style, their mental health, and their children's emotional vulnerabilities: A 7-year study of children with congenital heart disease. *Journal of Personality, 76*(1), 31-66. doi:10.1111/j.1467-6494.2007.00479.x

Bettmann, J. (2006). Using attachment theory to understand the treatment of adult depression. *Clinical Social Work Journal, 34*(4), 531-542. doi:10.1007/s10615-005-0033-1

Bhugra, D., & Mastrogianni, A. (2003). Globalisation and mental disorders. *The British Journal of Psychiatry*, *184*, 10-20. doi:10.1192/bjp.184.1.10

Bhui, K., & Bhugra, D. (2002). Explanatory models for mental distress: Implications for clinical practice and research. *The British Journal of Psychiatry*, *181*, 6-7. doi:10.1192/bjp.181.1.6

Bhui, K. & Bhugra, D. (2004). Communication with patients from other cultures: The place of explanatory models. *Advances in Psychiatric Treatment, 10*(6), 474-478. doi:10.1192/apt.10.6.474

Black Dog Institute. (n. d.). *Treatments for depression* [Fact sheet]. Retrieved from https://www.blackdoginstitute.org.au/docs/default-source/factsheets/treatmentsfordepression.pdf

Black, I. (2006). The presentation of interpretivist research. *Qualitative Market Research, 9*(4), 319-324. doi:10.1108/13522750610689069

Blau, P. M. (1964). *Exchange and power in social life*. New York: John Wiley.

Blazer, D. G., Kessler, R. C., Mcgonagle, K. A., & Swartz, M. S. (1994). The prevalence and distribution of major depression in a national community sample: The National Comorbidity Survey. *The American Journal of Psychiatry*, *151*(7), 979. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/8010383

Blease, C. (2014). The duty to be well-informed: The case of depression. *Journal of Medical Ethics, 40*(4), 225. doi:10.1136/medethics-2012-101122

Blumhagen, D. (1980). The meaning of hypertension. In J. Chirsman & N. J. Maretzi (Eds.), *Clinically applied anthropology* (pp. 297-324). London: D. Reidal.

Bobo, W. V., Voort, J. L. V., Croarkin, P. E., Leung, J. G., Tye, S. J., & Frye, M. A. (2016). Ketamine for treatment-resistant unipolar and bipolar major depression: Critical review and implications for clinical practice. *Depression and Anxiety*, *33*(8), 698-710. doi:10.1002/da.22505

Bockting, C. L. H., Schene, A. H., Spinhoven, P., Koeter, M. W. J., Wouters, L. F., Huyser, J., & Kamphuis, J. H. (2005). Preventing relapse/recurrence in recurrent depression with cognitive therapy: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 73*(4), 647-657. doi:10.1037/0022-006X.73.4.647

Bockting, C. L. H., Smid, N. H., Koeter, M. W. J., Spinhoven, P., Beck, A. T., & Schene, A. H. (2015). Enduring effects of preventive cognitive therapy in adults remitted from recurrent depression: A 10 year follow-up of a randomized controlled trial. *Journal of Affective Disorders, 185*, 188-194. doi:10.1016/j.jad.2015.06.048

Boerma, T. (2017, January). Message from the Director. *ICD-11 Update*. Retrieved from http://www.who.int/classifications/ICD11January2017Newsletter.pdf

Bolarinwa, O. A. (2015). Principles and methods of validity and reliability testing of questionnaires used in social and health science researches. *Nigerian Postgraduate Medical Journal*, *22*(4), 195-201. doi:10.4103/1117-1936.173959

Botha, F. B., & Dozois, D. J. A. (2015). The influence of emphasizing psychological causes of depression on public stigma. *Canadian Journal of Behavioural Science, 47*(4), 313-320. doi:10.1037/a0039611

Bowlby, J. (1969). *Attachment and loss: Vol. 1. Attachment.* New York, NY: Basic Books.

Bowlby, J. (1973). *Attachment and loss: Vol. 2. Separation: Anxiety and anger.* New York, NY: Basic Books.

Bowlby, J. (1980). *Attachment and loss: Vol. 3. Loss: Sadness and depression.* New York, NY: Basic Books.

Boyd, J. E., Adler, E. P., Otilingam, P. G., & Peters, T. (2014) Internalized stigma of mental illness (ISMI) scale: A multinational review. *Comprehensive Psychiatry, 55*(1), 221-231. doi:10.1016/j.comppsych.2013.06.005

Brennan, K. A., Clark, C. L., & Shaver, P. R. (1998). Self-report measurement of adult attachment: An integrative overview. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and* *close relationships* (pp. 46-76). New York: Guilford Press.

Briggs, S., & Cheek, J. (1986). The role of factor analysis in the development and evaluation of personality scales. *Journal of Personality, 54*(1), 106-148. doi:10.1111/j.1467-6494.1986.tb00391.x

Bromet, E., Andrade, L. H., Hwang, I., Sampson, N. A., Alonso, J., de Girolamo, G., … Kessler, R. C. (2011). Cross-national epidemiology of DSM-IV major depressive episode. *BMC Medicine*, *9*, 90-105. doi:10.1186/1741-7015-9-90

Brondino, N., Rocchetti, M., Fusar-Poli, L., Codrons, E., Correale, L., Vandoni, M., . . . Politi, P. (2017). A systematic review of cognitive effects of exercise in depression. *Acta Psychiatrica Scandinavica*, 135(4), 285-295. doi:10.1111/acps.12690

Brown, J. D. (2011, March). Likert items and scales of measurement? *SHIKEN: JALT Testing & Evaluation SIG Newsletter, 15*(1), 10-14. Retrieved from https://jalt.org/test/PDF/Brown34.pdf

Brown, C., Battista, D., Bruehlman, R., Sereika, S., Thase, M., & Dunbar-Jacob, J. (2005). Beliefs about antidepressant medications in primary care patients: Relationship to self-reported adherence. *Medical Care, 43*(12), 1203-7. doi:10.1097/01.mlr.0000185733.30697.f6

Brown, C., Battista, D. R., Sereika, S. M., Bruehlman, R. D., Dunbar-Jacob, J., & Thase, M. E. (2007). Primary care patients’ personal illness models for depression: Relationship to coping behavior and functional disability. *General Hospital Psychiatry, 29*(6), 492-500. doi:10.1016/j.genhosppsych.2007.07.007

Brown, C., Conner, K. O., Copeland, V. C., Grote, N., Beach, S., Battista, D., & Reynolds, C. F. (2010). Depression stigma, race, and treatment seeking behavior and attitudes. J*ournal of Community Psychology, 38*, 350-368. doi:10.1002/jcop.20368

Brown, G., Harris, T., & Hepworth, C. (1995). Loss, humiliation and entrapment among women developing depression: A patient and non-patient comparison. *Psychological Medicine, 25*(1), 7-21. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/7792364

Budd, R., James, D., & Hughes, I. (2008). Patients’ explanations for depression: A factor analytic study. *Clinical Psychology & Psychotherapy, 15*(1), 28-37. doi:10.1002/cpp.558

Buhrmester, M., Kwang, T., & Gosling, S. (2011). Amazon’s Mechanical Turk: A new source of inexpensive, yet high-quality, data? *Perspectives on Psychological Science, 6*(1), 3-5. doi:10.1177/1745691610393980

Bull, S. A, Hu, X. H., Hunkeler, E. M., Lee, J. Y., Ming, E. E, Markson, L. E, & Fireman, B. (2002). Discontinuation of use and switching of antidepressants: Influence of patient-physician communication, *Journal of the American Medical Association*, 288(11), 1403-1409. doi:10.1001/jama.288.11.1403

Burnett-Zeigler, I., Kim, H. M., Chiang, C., Kavanagh, J., Zivin, K., Rockefeller, K.,…Kales, H. C. (2014). The association between race and gender, treatment attitudes, and antidepressant treatment adherence. *International Journal of Geriatric Psychiatry, 29*(2), 169-177. doi:10.1002/gps.3984

Burton, C. (2003). Beyond somatisation: A review of the understanding and treatment of medically unexplained physical symptoms (MUPS). *British Journal of General Practice*, *53*(488), 231-239. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/14694702

Burton, C., Mcgorm, K., Weller, D., & Sharpe, M. (2011). Depression and anxiety in patients repeatedly referred to secondary care with medically unexplained symptoms: A case-control study. *Psychological Medicine*, 41(3), 555-563. doi:10.1017/S0033291710001017

Bushnell, J. (2004). Frequency of consultations and general practitioner recognition of psychological symptoms. *The British Journal of General Practice, 54*(508), 838-43. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1324917/

Buus, N., Johannessen, H., & Stage, K. B. (2012). Explanatory models of depression and treatment adherence to antidepressant medication: A qualitative interview study. *International Journal of Nursing Studies, 49*(10), 1220. doi:10.1016/j.ijnurstu.2012.04.012

Cabassa, L., Hansen, M., Palinkas, L., & Ell, K. (2008). Azucar y nervios: Explanatory models and treatment experiences of Hispanics with diabetes and depression. *Social Science & Medicine*, *66*(12), 2413-2424. doi:10.1016/j.socscimed.2008.01.054

Cameron, K. A., De Haes, H., & Visser, A. (2009). Theories in health communication research. *Patient Education and Counseling, 74*(3), 279-281. doi:10.1016/j.pec.2008.12.021

Cantazaro, A., & Wei, M. (2010). Adult attachment, dependence, self-criticism, and depressive symptoms: A test of a mediational model. *Journal of Personality, 78*(4), 1135-1162. doi:10.1111/j.1467-6494.2010.00645.x

Cardozo, B. L., Bilukha, O. O., Gotway, C. A., Wolfe, M. I., Gerber, M. L., & Anderson, M. (2005). Mental health of women in postwar Afghanistan. *Journal of Women's Health*, *14*(4), 285-293. doi:10.1089/jwh.2005.14.285

Carifio, L., & Perla, R. (2008). Resolving the 50 year debate around using and misusing Likert scales. *Medical Education, 42*, 1150-1152. doi:10.1111/j.1365-2923.2008.03172.x

Carley-Baxter, L. (2008). Respondent-interviewer rapport. In P. J. Lavrakas (Ed.), *Encyclopedia of survey research methods* (pp. 743-744). Thousand Oaks, CA: Sage. doi:10.4135/9781412963947.n481

Carney, P. A., Rhodes, L. A., Eliassen, M. S., Badger, L. W., Rand, E. H., Neiswender, C., … Dietrich, A. J. (1998). Variations in approaching the diagnosis of depression: A guided focus group study. *The Journal of Family Practice*, *46*(1), 73-82. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/9451373

Carpenter, C. J. (2010) A meta-analysis of the effectiveness of health belief model variables in predicting behavior, *Health Communication, 25*(8), 661-669, doi:10.1080/10410236.2010.521906

Carpenter-Song, E., Chu, E., Drake, R., Ritsema, M., Smith, B., & Alverson, H. (2010). Ethno-cultural variations in the experience and meaning of mental illness and treatment: Implications for access and utilization. *Transcultural Psychiatry*, 47(2), 224-251. doi:10.1177/1363461510368906

Carson, D. J., Gilmore, A., Perry, C., & Gronhaug, K. (2001). *Qualitative Marketing Research*. London: Sage. doi:10.4135/9781849209625

Carson, N., Katz, A., & Alegría, M. (2016). How patients and clinicians make meaning of physical suffering in mental health evaluations. *Transcultural Psychiatry, 53*(5), 595-611. doi:10.1177/1363461516660901

Carta, M. G., Angermeyer, M. C., Floris, F. F., Moro, M., Matschinger, H., & Holzinger, A. (2013). Perception of depressive symptoms by the Sardinian public: Results of a population study. *BMC Psychiatry, 13*, 57-65. doi:10.1186/1471-244X-13-57

Carter, F. I. (2008). *Exploration of siblings' explanatory models of autism* (Doctoral dissertation). Retrieved from ProQuest. (Order No. 3322093)

Casey, D. A. (2013). Do antidepressant medications work? *Pharmacy and Therapeutics, 38*(3), 162-163. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3638404/

Casini, F., Sighinolfi, C., Tedesco, P., Bandieri, P. V., Bologna, M., Colombini, N., … Menchetti, M. (2013). Primary care physicians' perspective on the management of anxiety and depressive disorders: A cross-sectional survey in Emilia Romagna Region. *BMC Family Practice, 14*(1), 75-. doi:10.1186/1471-2296-14-75

Cavanagh, J., Carson, A., Sharpe, M., & Lawrie, S. (2003). Psychological autopsy studies of suicide: A systematic review. *Psychological Medicine, 33*(3), 395-405. doi:10.1017/S0033291702006943

Cegala, D., McGee, D., & McNeilis, K. (1996). Components of patients' and doctors' perceptions of communication competence during a primary care medical interview. *Health Communication, 8*(1), 1-27. doi:10.1207/s15327027hc0801_1

Cepoiu, M., McCusker, J., Cole, M. G., Sewitch, M., Belzile, E., & Ciampi, A. (2007). Recognition of depression by non-psychiatric physicians: A systematic literature review and meta-analysis. *Journal of General Internal Medicine*, *23*(1), 25-36. doi:10.1007/s11606-007-0428-5

Chakraborty, K., Avasthi, A., Kumar, S., & Grover, S. (2009). Attitudes and beliefs of patients of first episode depression towards antidepressants and their adherence to treatment. *Social Psychiatry and Psychiatric Epidemiology, 44*(6), 482-488. doi:10.1007/s00127-008-0468-0

Chang, C., Hayes, R., Perera, G., Broadbent, M., Fernandes, A., Lee, W., . . . Scott, J. (2011). Life expectancy at birth for people with serious mental illness and other major disorders from a secondary mental health care case register in London. *PLoS ONE, 6*(5), e19590. doi:10.1371/journal.pone.0019590

Charles, H., Manoranjitham, S. D., & Jacob, K. S. (2007). Stigma and explanatory models among people with schizophrenia and their relatives in Vellore, South India*. International Journal of Social Psychiatry, 53*, 325-332. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/17703648

Chaudoir, S. R., Earnshaw, V. A., & Andel, S. (2013). “Discredited” versus “discreditable”: Understanding how shared and unique stigma mechanisms affect psychological and physical health disparities. *Basic and Applied Social Psychology, 35*, 75-87. doi:10.1080/01973533.2012.746612

Chesney, E., Goodwin, G., & Fazel, S. (2014). Risks of all-cause and suicide mortality in mental disorders: A meta-review. *World Psychiatry, 13*(2), 153-160. doi:10.1002/wps.20128

Clark, D. A., Beck, A. T., & Alford, B. A. (1999). *Scientific foundations of cognitive theory and therapy of depression.* New York: John Wiley.

Clarke, J., & Gawley, A. (2009). The triumph of pharmaceuticals: The portrayal of depression from 1980 to 2005. *Administration and Policy in Mental Health and Mental Health Services Research, 36*(2), 91-101. doi:10.1007/s10488-008-0199-2

Clason, D. L., & Dormody, T. J. (1994). Analyzing data measured by individual Likert-type items. *Journal of Agricultural Education,* (4), 31-35. Retrieved from http://pubs.aged.tamu.edu/jae/pdf/vol35/35-04-31.pdf

Cohen, J. (1988). *Statistical power analysis for the behavioural sciences.* Hillsdale, NJ: Lawrence Erlbaum.

Cohen, M. Z., Tripp-Reimer, T., Smith, C., Sorofman, B., & Lively, S. (1994). Explanatory models of diabetes: Patient practitioner variation. *Social Science & Medicine*, *38*(1), 59-66. doi:10.1016/0277-9536(94)90300-X

Colom, F. (2011) Keeping therapies simple: Psychoeducation in the prevention of relapse in affective disorders. *The British Journal of Psychiatry* 198: 338-340. doi:10.1192/bjp.bp.110.090209.

Conner, K., Copeland, V., Grote, N., Koeske, G., Rosen, D., Reynolds, C., & Brown, C. (2010). Mental health treatment seeking among older adults with depression: The impact of stigma and race. *The American Journal of Geriatric Psychiatry,18*(6), 531-43. doi:10.1097/JGP.0b013e3181cc0366

Cookson, J. (1993). Side-effects of antidepressants. *British Journal Of Psychiatry, 163* (Suppl. 20), 20-24. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/8352912

Cooney, G. M., Dwan, K., Greig, C. A., Lawlor, D. A., Rimer, J., Waugh, F. R., … Mead, G. E. (2013). Exercise for depression. *Cochrane Database of Systematic Reviews* (9). doi:10.1002/14651858.CD004366.pub6

Cooney, G., Dwan, K., & Mead, G. (2014). Exercise for depression. *Journal of the American Medical Association, 311*(23), 2432-2433. doi:10.1001/jama.2014.4930

Cooper, M. L., Shaver, P. R., & Collins, N. L. (1998). Attachment styles, emotion regulation, and adjustment in adolescence. *Journal of Personality and Social Psychology*, *74*(5), 1380-1397. doi:10.1037/0022-3514.74.5.1380

Corbin, J., & Strauss, A. (2008). *Basics of qualitative research: Techniques and procedures for developing grounded theory* (3rd ed.). Thousand Oaks, CA: Sage.

Cornford, C. S., Hill, A., & Reilly, J. (2007). How patients with depressive symptoms view their condition: A qualitative study. *Family Practice, 24*(4), 358-364. doi:10.1093/fampra/cmm032

Corrigan, P. (2004). How stigma interferes with mental health care. *American Psychologist, 59*(7), 614-625. doi:10.1037/0003-066X.59.7.614

Corrigan, P. (2016). Resolving mental illness stigma: Should we seek normalcy or solidarity? *The British Journal of Psychiatry, 208*(4), 314-5. doi:10.1192/bjp.bp.115.169664

Corrigan, P. W., Druss, B. G., & Perlick, D. A. (2014). The impact of mental illness stigma on seeking and participating in mental health care. *Psychological Science in the Public Interest, 15*(2), 37-70. doi:10.1177/1529100614531398

Corrigan, P. W., River, L. P., Lundin, R. K., Wasowski, K. U., Campoin, J., Mathisen, J., . . . Gagnon, C. (2000). Stigmatizing attributions about mental illness. *Journal of Community Psychology, 28*(1), 91-102. doi:10.1002/(SICI)1520-6629(200001)28:1<91::AID-JCOP9>3.0.CO;2-M

Corrigan P. W., Watson, A. C. (2002a). Understanding the impact of stigma on people with mental illness. *World Psychiatry*, 1(1), 16-20. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1489832

Corrigan, P. W., & Watson, A. C. (2002b). The paradox of self-stigma and mental illness. *Clinical Psychology: Science and Practice, 9*(1), 35-53. doi:10.1093/clipsy.9.1.35

Corrigan, P. W., Watson, A. C., & Barr, L. (2006). The self-stigma of mental illness: Implications for self-esteem and self-efficacy. *Journal of Social and Clinical Psychology, 25*(8), 875-884. doi:10.1521/jscp.2006.25.8.875

Coupland, N., Coupland, J., Giles, H., & Henwood, K. (1988). Accommodating the elderly: Invoking and extending a theory. *Language in Society, 17,* 1-41. Retrieved from http://www.jstor.org/stable/4167896

Crane, G. E. (1956). The psychiatric side-effects of iproniazid. *American Journal of Psychiatry*, *112*(7), 494-501. doi:10.1176/ajp.112.7.494

Cretchley, J., Gallois, C., Chenery, H., & Smith, A. (2010). Conversations between carers and people with schizophrenia: A qualitative analysis using Leximancer. *Qualitative Health Research, 20*(12), 1611-1628. doi:10.1177/1049732310378297

Crisp, A., Gelder, M., Goddard, E., & Meltzer, H. (2005). Stigmatization of people with mental illnesses: A follow-up study within the Changing Minds campaign of the Royal College of Psychiatrists. *World Psychiatry, 4*(2), 106-113. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/16633526

Cronbach, L. J. (1951). Coefficient alpha and the internal structure of tests. *Psychomerika, 16*(3), 297-334. doi:10.1007/bf02310555.

Crotty, M. (1998). *The foundations of social research: Meaning and perspective in the research process*. Sydney, NSW: Allen & Unwin.

Cuijpers, P., Berking, M., Andersson, G., Quigley, L., Kleiboer, A., & Dobson, K. (2013). A meta-analysis of cognitive-behavioural therapy for adult depression, alone and in comparison with other treatments. *The Canadian Journal of Psychiatry, 58*(7), 376-385. doi:10.1177/070674371305800702

Cuijpers, P., Van Straten, A., Bohlmeijer, E., Hollon, S., & Andersson, G. (2010). The effects of psychotherapy for adult depression are overestimated: A meta-analysis of study quality and effect size. *Psychological Medicine, 40*(2), 211-223. doi:10.1017/S0033291709006114

Cuijpers, P., Van Straten, A., Van Schaik, A., & Andersson, G. (2009). Psychological treatment of depression in primary care: A meta-analysis. *The British Journal of General Practice, 59*(559), E51-60. doi:10.3399/bjgp09X395139

Cullinan, V., Veale, A., & Vitale, A. (2016). Irish General Practitioner referrals to psychological therapies. *Irish Journal of Psychological Medicine, 33*(2), 73-80. doi:10.1017/ipm.2015.17

Cunningham, J., Sirey, J. A., & Bruce, M. L. (2007). Matching services to patients' beliefs about depression in Dublin, Ireland. *Psychiatric Services, 58*(5), 696-699. doi:10.1176/appi.ps.58.5.696

De Winter, J. C., & Dodou, D. (2010). Five-point likert items: *t*-test versus Mann-Whitney-Wilcoxon. *Practical Assessment, Research and Evaluation, 15*(11). Retrieved from http://pareonline.net/pdf/v15n11.pdf

Deacon, B. J., & Baird, G. L. (2009). The chemical imbalance explanation of depression: Reducing blame at what cost? *Journal of Social and Clinical Psychology, 28*(4), 415-443. doi:10.1521/jscp.2009.28.4.415

Dejman, M., Forouzan, A. S., Assari, S., Rasoulian, M., Jazayery, A., Malekafzali, H., … Ekblad, S. (2010). How Iranian lay people in three ethnic groups conceptualize a case of a depressed woman: An explanatory model. *Ethnicity & Health*, *15*(5), 475-493. doi:10.1080/13557858.2010.488262

Deledda, G., Moretti, F., Rimondini, M., & Zimmermann, C. (2012). How patients want their doctor to communicate: A literature review on primary care patients' perspective. *Patient Education and Counseling*, *90*(3), 297-306. doi:10.1016/j.pec.2012.05.005

Denscombe, M. (2010). *The good research guide for small-scale social research projects* (4th ed.). New York: McGraw Hill Open University Press

Diagnosis of depression. (2015). Retrieved March 30, 2017, from https://www.healthdirect.gov.au/diagnosis-of-depression

Dietrich, S., Beck, M., Bujantugs, B., Kenzine, D., Matschinger, H., & Angermeyer, M. (2004). The relationship between public causal beliefs and social distance toward mentally ill people. *Australian and New Zealand Journal of Psychiatry, 38*(5), 348-354. doi:10.1080/j.1440-1614.2004.01363.x

Diette G. B., & Rand C. (2007). The contributing role of health-care communication to health disparities for minority patients with asthma. *Chest, 132*(5), 802S-809S. doi:10.1378/chest.07-1909

Dinoff, A., Herrmann, N., & Lanctôt, K. L. (2017). Ceramides and depression: A systematic review. *Journal of Affective Disorders*, *213*, 35-43. doi:10.1016/j.jad.2017.02.008

Doblyte, S., & Jiménez-Mejías, E. (2017). Understanding help-seeking behavior in depression: A qualitative synthesis of patients' experiences. *Qualitative Health Research, 27*(1), 100-113. doi:10.1177/1049732316681282

Doran, John (2017, January 14). “Capitalist Realism Author Mark Fisher Dies.” *The Quietus.* Retrieved from http://thequietus.com/articles/21567-mark-fisher-dies-rip

Dunn, K., Forrest, J., Burnley, I., & McDonald, A. (2004). Constructing racism in Australia. *Australian Journal of Social Issues, 39*(4), 409-430. doi:10.1002/j.1839-4655.2004.tb01191.x

Dunn, O. J. (1964). Multiple comparisons using rank sums. *Technometrics, 6*(3):241-252. doi:10.1080/00401706.1964.10490181

Durà-Vilà, G., Dein, S., Littlewood, R., & Leavey, G. (2010). The dark night of the soul : Causes and resolution of emotional distress among contemplative nuns. *Transcultural Psychiatry,47*(4), 548-570.

Earnshaw, V.A., & Chaudoir, S.R. (2009). From conceptualizing to measuring HIV stigma: A review of HIV stigma mechanism measures. *AIDS and Behavior, 13*, 1160-1177. doi:10.1007/s10461-009-9593-3

Eaton, W., Shao, H., Nestadt, G., Lee, B., Bienvenu, O., & Zandi, P. (2008). Population-based study of first onset and chronicity in major depressive disorder. *Archives of General Psychiatry, 65*(5), 513-520. doi:10.1001/archpsyc.65.5.513

Eisinga, R., Grotenhuis, M., & Pelzer, T. (2013). The reliability of a two-item scale: Pearson, Cronbach, or Spearman-Brown? *International Journal of Public Health, 58*(4), 637-642. doi:10.1007/s00038-012-0416-3

Elliott, M., Maitoza, R., & Schwinger, E. (2012). Subjective accounts of the causes of mental illness in the USA. *International Journal of Social Psychiatry, 58*(6), 562-567. doi:10.1177/0020764011415207

Engel, G. (1977). The need for a new medical model: A challenge for biomedicine. *Science, 196*(4286), 129-136. Retrieved from http://www.jstor.org/stable/1743658

Engel, G. (1980). The clinical application of the biopsychosocial model. *The American Journal of Psychiatry, 137*(5), 535-544. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/7369396

Engstrom, E. J., & Kendler, K. S. (2015) Emil Kraepelin: Icon and reality. *The American Journal of Psychiatry, 172*(12), 1190-1196. doi:10.1176/appi.ajp.2015.15050665

Epstein, R. (2006). Making communication research matter: What do patients notice, what do patients want, and what do patients need? *Patient Education and Counseling, 60*(3), 272-278. doi:10.1016/j.pec.2005.11.003

Epstein, R. (2014). Realizing Engel’s biopsychosocial vision: Resilience, compassion, and quality of care. *The International Journal of Psychiatry in Medicine, 47*(4), 275-287. doi:10.2190/PM.47.4.b

Epstein, R. M., Franks, P., Fiscella, K., Shields, C. G., Meldrum, S. C., Kravitz, R. L., & Duberstein, P. R. (2005). Measuring patient-centered communication in patient-physician consultations: Theoretical and practical issues. *Social Science & Medicine, 61*(7), 1516-1528. doi:10.1016/j.socscimed.2005.02.001

Euba, R., Panihhidina, I., & Zamar, A. (2015). Treatment-resistant depression: The experience of the first rTMS Clinic in the UK. *Future Neurology*, *10*(3), 211-215. doi:10.2217/fnl.15.8.

Feighner, J., Robins, E., Guze, S., Woodruff, R., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry, 26*(1), 57-63. doi:10.1001/archpsyc.1972.01750190059011

Ferguson, P. (2006). Rush, Benjamin (1745-1813). In G. L. Albrecht (Ed.), *Encyclopedia of disability* (Vol. 5, pp. 1422-1423). Thousand Oaks, CA: Sage. doi:10.4135/9781412950510.n699

Fernandez Y Garcia, E., Franks, P., Jerant, A., Bell, R., & Kravitz, R. (2011). Depression treatment preferences of Hispanic individuals: Exploring the influence of ethnicity, language, and explanatory models. *Journal of the American Board of Family Medicine, 24*(1), 39-50. doi:10.3122/jabfm.2011.01.100118

Ferrari, A. J., Charlson, F. J., Norman, R. E., Patten, S. B., Freedman, G., Murray, C. J. L., … Whiteford, H. A. (2013). Burden of depressive disorders by country, sex, age, and year: findings from the global burden of disease study 2010. *PLoS Medicine*, *10*(11), e1001547. doi:10.1371/journal.pmed.1001547

Ferrari, A. J., Somerville, A. J., Baxter, A. J., Norman, R., Patten, S. B., Vos, T., & Whiteford, H. A. (2013). Global variation in the prevalence and incidence of major depressive disorder: a systematic review of the epidemiological literature. *Psychological Medicine*, *43*(3), 471-481. doi:10.1017/S0033291712001511

Field, A. (2003). Designing a questionnaire. Retrieved from http://www.discoveringstatistics.com/docs/designing_questionnaires.pdf

Field, A. (2009). *Discovering statistics using SPSS*. London: Sage.

Field, T., Beeson, E., & Jones, L. (2015). The new ABCs: A practitioner's guide to neuroscience-informed cognitive-behavior therapy. *Journal of Mental Health Counseling, 37*(3), 206-220. doi:10.17744/mehc.37.3.02

First, M. B. (2009). Harmonisation of ICD-11 and DSM-V: Opportunities and challenges. *The British Journal of Psychiatry, 195*(5), 382-90. doi:10.1192/bjp.bp.108.060822

Fisher, M. (2012, July 16). Why mental health is a political issue. *The Guardian*. Retrieved from https://www.theguardian.com/commentisfree/2012/jul/16/mental-health-political-issue

Fitton, F., & Acheson, H. W. K. (1979). The doctor-patient relationship. London: Her Majesty's Stationery Office.

Fonseca, M. J., Costa, P., Lencastre, L., & Tavares, F. (2013). A statistical approach to quantitative data validation focused on the assessment of students' perceptions about biotechnology. *SpringerPlus*, *2*, 496. doi:10.1186/2193-1801-2-496

Ford, A., Almeida, O.P., Flicker, L., Hirani, V., Mccaul, K., Singh, U., & Van Bockxmeer, F. (2015). B-vitamins and depression, *European Psychiatry, 30*, 1318, doi:10.1016/S0924-9338(15)31027-0

Foucault, M. (1961/1965). *Madness and civilization: A history of insanity in the age of reason* (R. Howard, Trans.). New York: Pantheon Books.

Fournier, J., DeRubeis, R., Hollon, S., Dimidjian, S., Amsterdam, J., Shelton, R., & Fawcett, J. (2010). Antidepressant drug effects and depression severity: A patient-level meta-analysis. *Journal of the American Medical Association, 303*(1), 47-53. doi:10.1001/jama.2009.1943

Freimuth, V. S., Massett, H. A., & Meltzer, W. (2006). A descriptive analysis of 10 years of research published in the Journal of Health Communication. *Journal of Health Communication, 11*, 11-20. doi:10.1080/10810730500461042

Fremon, B., Negrete, V. F., Davis, M., & Korsch, B. M. (1971). Gaps in doctor-patient communication: Doctor-patient interaction analysis. *Pediatric Research, 5*(7), 298-311. doi:10.1203/00006450-197107000-00003

Freud, S. (1917/1959). Mourning and melancholia (J. Riviere, Trans.). In E. Jones (Ed.), *Collected papers* (Vol. 4, pp. 60-83). New York: Basic Books.

Furnham, A., & Kuyken, W. (1991). Lay theories of depression. *Journal of Social Behavior and Personality, 6*(2), 329-342. Retrieved from https://search.proquest.com/docview/1292300075?accountid=14723

Gadermann, A. M., Guhn, M., & Zumbo, B. D. (2012). Estimating ordinal reliability for Likert-type and ordinal item response data: A conceptual, empirical, and practical guide. *Practical Assessment, Research & Evaluation*, *17*(3). Retrieved from http://pareonline.net/pdf/v17n3.pdf

Galasiński, D. (2008). *Men's discourses of depression*. Basingstoke, NY: Palgrave MacMillan.

Galasiński, D., & Kozlowska, O. (2010). Questionnaires and lived experience: Strategies of coping with the quantitative frame. *Qualitative Inquiry*, *16*(4), 271-284. doi:10.1177/1077800409354068

Gallois, C., & Giles, H. (2015). Communication accommodation theory. In K. Tracy, C. Ilie, & T. Sandel (Eds.), *The International Encyclopedia of Language and Social Interaction* (Vol. 1, pp. 159-176). Oxford: Wiley-Blackwell.

Gallois, C., Ogay, T., & Giles, H. (2005). Communication accommodation theory: A look back and a look ahead. In W. B. Gudykunst (Ed.), *Theorizing about intercultural communication* (pp. 121-148). Thousand Oaks, CA: Sage.

Gammell, D. J., & Stoppard, J. M. (1999). Women's experience of treatment of depression: Medicalization or empowerment? *Canadian Psychology, 40*, 112-128. doi:10.1037/h0086831

Ghane, S., Kolk, A. M., & Emmelkamp, P. M. G. (2010). Assessment of explanatory models of mental illness: Effects of patient and interviewer characteristics. *Social Psychiatry and Psychiatric Epidemiology*, *45*(2), 175-182. doi:10.1007/s00127-009-0053-1

Ghanean, H., Nojomi, M., & Jacobsson, L. (2011). Internalized stigma of mental illness in Tehran, Iran. *Stigma Research and Action, 1*(11), 11-17. doi:10.5463/sra.v1i1.10

Giles, H. (1973). Accent mobility: A model and some data. *Anthropological Linguistics, 15*(2), 87-105. Retrieved from http://www.jstor.org/stable/30029508

Giles, H., Coupland, J., & Coupland, N. (1991). Accommodation Theory: Communication, context, and consequence. In H. Giles, J. Coupland, & N. Coupland (Eds). *Contexts of Accommodation* (pp. 1-68). New York: Cambridge University Press.

Giles, H., Gasiorek, J., & Soliz, J. (2015). Accommodating new vistas. *Language and Communication, 41*(1), 1-5. doi:10.1016/j.langcom.2014.10.001

Giles, H., & Smith, P. (1979). Accommodation Theory: Optimal Levels of Convergence. In H. Giles & R. N. St. Clair (Eds.), *Language and Social Psychology* (pp. 45-65). Baltimore: University Park Press.

Giles, H., Taylor, D. M., & Bourhis, R. Y. (1973). Toward a theory of interpersonal accommodation through language: Some Canadian data. *Language in Society, 2*(2), 177-192. doi:10.1017/S0047404500000701

Givens, J. L., Datto, C. J., Ruckdeschel, K., Knott, K., Zubritsky, C., Oslin, D. W., … Barg, F. K. (2006). Older patients' aversion to antidepressants: A qualitative study. *Journal of General Internal Medicine*, *21*(2), 146-151. doi:10.1111/j.1525-1497.2005.00296.x

Gjerde, P. F., Onishi, M. S., & Carlson, K. (2004). Personality characteristics associated with romantic attachment: A comparison of interview and self-report methodologies. *Personality and Social Psychology Bulletin, 30*(11), 1402-1415. doi:10.1177/0146167204264291

Glanz, K. & Bishop, D. B. (2010). The role of behavioral science theory in development and implementation of public health interventions. *Annual Review of Public Health, 31*, 399-418. doi:10.1146/annurev.publhealth.012809.103604

Goffman, E. (1986). *Stigma: Notes on the management of spoiled identity.* New York: Simon & Schuster. Retrieved from http://books.google.com/books (Original work published 1963)

Goldman, L., Nielsen, S., & Champion, N. (1999). Awareness, diagnosis, and treatment of depression. *Journal of General Internal Medicine, 14*(9), 569-580. doi:10.1046/j.1525-1497.1999.03478.x

Goldstein, W. N., & Anthony, R. N. (1988). The diagnosis of depression and the DSMs. *American Journal of Psychotherapy*, 42(2), 180-196. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/?term=3041844

Goldstein, B., & Rosselli, F. (2003). Etiological paradigms of depression: The relationship between perceived causes, empowerment, treatment preferences, and stigma. *Journal of Mental Health, 12,* 551-563. doi:10.1080/09638230310001627919

González, H. M., Tarraf, W., West, B. T., Chan, D., Miranda, P. Y., & Leong, F. T. (2010). Antidepressant use among Asians in the United States. *Depression and Anxiety*, *27*(1), 46-55. doi:10.1002/da.20636

Goodman, M., & Hess, B. (1999). Could implantable cardioverter defibrillators provide a human model supporting the learned helplessness theory of depression? *General Hospital Psychiatry,21*(5), 382-385. doi:10.1016/S0163-8343(99)00030-4

Goodnick, P. J., & Hernandez, M. (2000). Treatment of depression in comorbid medical illness. *Expert Opinion on Pharmacotherapy*, *1*(7), 1367-1384. doi:10.1517/14656566.1.7.1367

Gray, D. E. (2014). *Doing research in the real world* (3rd ed.). London: Sage.

Green, S. B., & Salkind, N. J. (2008). *Using SPSS for Window and Macintosh: Analyzing and understanding data* (5th ed.). Upper Saddle River, NJ: Pearson Prentice Hall.

Greenberg, S. A., Shuman, D. W., & Meyer, R. G. (2004). Unmasking forensic diagnosis. *International Journal of Law and Psychiatry,27*(1), 1-15. doi: 10.1016/j.ijlp.2004.01.001

Grob, G. N. (2011). The attack of psychiatric legitimacy in the 1960s: Rhetoric and reality. *Journal of the History of Behavioural Sciences, 47,* 398-416. doi:10.1002/jhbs.20518

Grosso, G., Micek, A., Marventano, S., Castellano, S., Mistretta, A., Pajak, A., & Galvano, F. (2016). Dietary n-3 PUFA, fish consumption and depression: A systematic review and meta-analysis of observational studies. *Journal of Affective Disorders, 205*, 269-281. doi:10.1016/j.jad.2016.08.011

Grover, S., Kumar, V., Chakrabarti, S., Hollikatti, P., Singh, P., Tyagi, S., … P., K. (2012). Explanatory models in patients with first episode depression: A study from North India. *Asian Journal of Psychiatry*, *5*(3), 251-257. doi:10.1016/j.ajp.2012.07.003

Groves, R. M., Fowler, F. J., Jr., Couper, M. P., Lepkowski, J. M., Singer, E., & Tourangeau, R. (2009). *Survey methodology* (2nd ed.). Hoboken, NJ: Wiley.

Gruenberg, A. M., Goldstein, R. D., & Pincus, H. A. (2008). Classification of depression: Research and diagnostic criteria: DSM-IV and ICD-10. In J. Licinio & M. Wong (Eds.), *Biology of depression: From novel insights to therapeutic strategies* (pp. 1-12). Weinheim: Wiley-VCH. doi: 10.1002/9783527619672.ch1

Gustad, L., Laugsand, L., Janszky, I., Dalen, H., & Bjerkeset, O. (2014). Symptoms of depression and risk of heart failure. The HUNT 2 study. Poster session presented at annual meeting of the European Society of Cardiology (EuroHeartCare 2014), Stavanger, Norway. *European Journal of Cardiovascular Nursing,13*, S16-S17. doi: 10.1177/1474515114521363

Guthrie, G. (2010). *Basic research methods: An entry to social science research*. New Delhi: Sage India.

Ha, J. F., & Longnecker, N. (2010). Doctor-patient communication: A review. *Ochsner Journal, 10*(1), 38-43. Retrieved from http://www.ochsnerjournal.org/doi/pdf/10.1043/TOJ-09-0040.1

Hahn, T., Marquand, A. F., Ehlis, A., Dresler, T., Kittel-Schneider, S., Jarczok, T. A. …Fallgatter, A. J. (2011). Integrating neurobiological markers of depression. *Archives of General Psychiatry*, *68*(4), 361-368. doi:10.1001/archgenpsychiatry.2010.178

Haidet, P., O'Malley, K. J., Sharf, B. F., Gladney, A. P., Greisinger, A. J., & Street, R. L. (2008). Characterizing explanatory models of illness in healthcare: Development and validation of the CONNECT instrument. *Patient Education and Counseling, 73*(2), 232-239. doi:10.1016/j.pec.2008.07.007

Hallenbeck, J. (2003). The explanatory model #26. *Journal of Palliative Medicine*, *6*(6), 931. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/14733686

Halter, M. (2004). The stigma of seeking care and depression. *Archives of Psychiatric Nursing, 18*(5), 178-184. doi:10.1016/j.apnu.2004.07.005

Hamilton, D. L. , & Sherman, J. W. (1994). Stereotypes. In R. S. Wyer Jr. & T. K. Srull (Eds.), *Handbook of social cognition* (Vol. 1, pp. 1-68). Hillsdale, NJ: Lawrence Erlbaum.

Hampson, S., Glasgow, R., Toobert, D., & Matthews, K. A. (1990). Personal models of diabetes and their relations to self-care activities. *Health Psychology, 9*(5), 632-646. doi:10.1037/0278-6133.9.5.632

Harman, J. S., Veazie, P. J., & Lyness, J. M. (2006). Primary care physician office visits for depression by older Americans. *Journal of General Internal Medicine, 21*(9), 926-930. doi:10.1007/bf02743139

Harmon, G., Lefante, J., & Krousel-Wood, M. (2006). Overcoming barriers: The role of providers in improving patient adherence to antihypertensive medications. *Current Opinion in Cardiology, 21*(4), 310-315. doi:10.1097/01.hco.0000231400.10104.e2

Haslam, N., & Kvaale, E. P. (2015). Biogenetic explanations of mental disorder: The mixed-blessings model. *Current Directions in Psychological Science*, *24*(5), 399-404. doi:10.1177/0963721415588082

Hauenstein, E. J. (2003). No comfort in the rural South: Women living depressed. *Archives of Psychiatric Nursing*, *17*, 3-11. doi:10.1053/apnu.2003.6

Hawton, K., Casañas i Comabella, C., Haw, C., & Saunders, K. (2013). Risk factors for suicide in individuals with depression: A systematic review. *Journal of Affective Disorders, 147*, 17-28. doi:10.1016/j.jad.2013.01.004

Hayward, P. A., & Bright, J. (1997). Stigma and mental illness: A review and critique. *Journal of Mental Health, 6*(4), 345-354. doi:10.1080/09638239718671

Hazan, C., & Shaver, P. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality and Social Psychology*, *52*(3), 511-524. doi:10.1037/0022-3514.52.3.511

Heeren, T., & D'Agostino, R. (1987). Robustness of the two independent samples *t*-test when applied to ordinal scaled data. *Statistics in Medicine, 6*, 79-90. doi:10.1002/sim.4780060110

Heider, F. (1958). *The psychology of interpersonal relations*. New York: John Wiley.

Helman, C. G. (1985). Communication in primary care: The role of patient and practitioner explanatory models. *Social Science & Medicine, 20*(9), 923-931. doi:10.1016/0277-9536(85)90348-x

Herting, G., Axelrod, J., & Whitby, L. (1961). Effect of drugs on the uptake and metabolism of H^3-norepinephrine. *The Journal of Pharmacology and Experimental Therapeutics, 134*, 146-153. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/14042299

Herman, S., Archambeau, O. G., Deliramich, A. N., Kim, B. S. K., Chiu, P. H., & Frueh, B. C. (2011). Depressive symptoms and mental health treatment in an ethnoracially diverse college student sample. *Journal of American College Health, 59*(8), 715-720.

Hickie, I. B., Davenport, T. A., Scott, E. M., Hadzi-Pavlovic, D., Naismith, S. L., & Koschera, A. (2001). Unmet need for recognition of common mental disorders in Australian general practice. *The Medical Journal of Australia*, *175 Suppl.*, S18-24. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/11556431

Highet, N., Luscombe, G., Davenport, T., Burns, J., & Hickie, I. (2006). Positive relationships between public awareness activity and recognition of the impacts of depression in Australia. *Australian and New Zealand Journal of Psychiatry, 40*(1), 55-58. doi:10.1080/j.1440-1614.2006.01742.x

Hinton, P. R., Brownlow, C., McMurray, I., & Cozens, B. (2004). *SPSS explained*. East Sussex, UK: Routledge.

Hiroto, D. S. & Seligman, M. E. P. (1975). Generality of learned helplessness in man. *Journal of Personality and Social Psychology, 31*(2), 311-327. doi:10.1037/h0076270

Hirschfeld, R. M. (2000). History and evolution of the monoamine hypothesis of depression. *The Journal of Clinical Psychiatry*, *61* (Suppl. 6), 4-6. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/10775017

Ho, E., Au, P. & Amerasinghe, D. (2015). *Suicide in Asian communities: An exploratory study in New Zealand – 2015*. Planning and Funding, Auckland District Health Board. Retrieved from https://www.fmhs.auckland.ac.nz/assets/fmhs/soph/sch/cahre/publications/Suicide%20in%20Asian%20communities%202015.pdf

Hodge, D. R., & Gillespie, D. (2003). Phrase completions: An alternative to Likert scales. *Social Work Research, 27,* 45-55. doi:10.1093/swr/27.1.45

Hoffmann, E. A. (2007). Open-ended interviews, power, and emotional labor. *Journal of Contemporary Ethnography, 36*(3), 318346. doi:10.1177/0891241606293134

Holmes, J. (2000). Fitting the biopsychosocial jigsaw together. *The British Journal of Psychiatry, 177*, 93-94. doi:10.1192/bjp.177.2.93

Holzinger, A., Matschinger, H., Schomerus, G., Carta, M., & Angermeyer, M. (2011). The loss of sadness: The public’s view. *Acta Psychiatrica Scandinavica, 123*(4), 307-313. doi:10.1111/j.1600-0447.2010.01669.x

Horowitz, C. R., Rein, S. B., & Leventhal, H. (2004). A story of maladies, misconceptions and mishaps: Effective management of heart failure. *Social Science & Medicine, 58*(3), 631-643. doi:10.1016/S0277-9536(03)00232-6

Horvath, A. O., Del Re, A. C., Fluckiger, C., & Symonds, D. (2011). Alliance in individual psychotherapy. *Psychotherapy, 48*, 9-16. doi:10.1037/a0022186

Horwitz, A., & Wakefield, J. (2007). *The loss of sadness: How psychiatry transformed normal sorrow into depressive disorder.* Oxford: Oxford University Press.

Houts, A. (2000). Fifty years of psychiatric nomenclature: Reflections on the 1943 War Department Technical Bulletin, Medical 203. *Journal of Clinical Psychology, 56*(7), 935-67. doi:10.1002/1097-4679(200007)56:7<935::AID-JCLP11>3.0.CO;2-8

Hudson, L., & Ozanne, J. (1988). Alternative ways of seeking knowledge in consumer research. *Journal of Consumer Research,14*(4), 508-521. Retrieved from http://www.jstor.org/stable/2489157

Hunt, K., Sweeting, H., Keoghan, M., & Platt, S. (2006). Sex, gender role orientation, gender role attitudes, and suicidal thoughts in three generations: A general population study. *Social Psychiatry and Psychiatric Epidemiology, 41*(8), 641-647. doi:10.1007/s00127-006-0074-y

Hyden, L. C. (1997). Illness and narrative. *Sociology of Health and Illness, 19*(1), 48-69. doi:10.1111/j.1467-9566.1997.tb00015.x

Ihara, K., Muraoka, Y., Oiji, A., & Nadaoka, T. (1998). Prevalence of mood disorders according to DSM-III-R criteria in the community elderly residents in Japan. *Environmental Health and Preventive Medicine, 3*(1), 44-49. doi:10.1007/BF02931238

International Advisory Group for the Revision of ICD-10 Mental and Behavioural Disorders. (2008). *Summary Report of the 3rd Meeting of the International Advisory Group for the Revision of ICD-10 Mental and Behavioural Disorders*. Geneva: World Health Organization. Retrieved from http://www.who.int/mental_health/evidence/icd_summary_report_march_2008.pdf

Iselin, M., & Addis, M. (2003). Effects of etiology on perceived helpfulness of treatments for depression. *Cognitive Therapy and Research, 27*(2), 205-222. doi:10.1023/A:1023513310243

Iversen, L. (2005). The monoamine hypothesis of depression. In J. Licinio & M.-L. Wong (Eds.), *Biology of depression: From novel insights to therapeutic strategies* (pp. 71-86). Weinham: Wiley-VCH. doi: 10.1002/9783527619672.ch5

Jadhav, S., Weiss, M. G., & Littlewood, R. (2001). Cultural experience of depression among white Britons in London. *Anthropology & Medicine, 8*(1), 47-69. doi:10.1080/13648470120063989

Jakobsson, U. (2004). Statistical presentation and analysis of ordinal data in nursing research. *Scandinavian Journal of Caring Sciences, 18*(4), 437-440. doi:10.1111/j.1471-6712.2004.00305.x

Jamieson, S. (2004). Likert scales: How to (ab)use them. *Medical Education, 38*, 1217-1218. doi:10.1111/j.1365-2929.2004.02012.x

Janz, N. K., & Becker, M. H. (1984). The health belief model: A decade later. *Health Education & Behavior, 11*,1-47. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/6392204

Jensen, J. D., King, A. J., Guntzviller, L. M., & Davis, L. A. (2010). Patient-provider communication and low-income adults: Age, race, literacy, and optimism predict communication satisfaction. *Patient Education and Counseling, 79*(1), 30-35. doi:10.1016/j.pec.2009.09.041

Johnson, D. P., & Whisman, M. A. (2013). Gender differences in rumination: A meta-analysis. *Personality and Individual Differences, 55*(4), 367-374. doi:10.1016/j.paid.2013.03.019

Johnston, B. (2013). The role of patient experience and its influence on adherence to antidepressant treatment. *Journal of Psychosocial Nursing & Mental Health Services, 51*(12), 29-37. doi:10.3928/02793695-20130930-04

Johnston, O., Kumar, S., Kendall, K., Peveler, R., Gabbay, J., & Kendrick, T. (2007). Qualitative study of depression management in primary care: GP and patient goals, and the value of listening. *The British Journal of General Practice, 57*(544), 872-879. doi:10.3399/096016407782318026

Jones, L., & Watson, B. M. (2012). Developments in health communication in the 21st Century. *Journal of Language and Social Psychology*, *31*(4), 415-436. doi:10.1177/0261927X12446612

Jorm, A. F., Christensen, F., & Griffiths, K. M. (2005). Public beliefs about causes and risk factors for mental disorders. *Social Psychiatry and Psychiatric Epidemiology, 40*(9), 764-767. doi:10.1007/s00127-005-0940-z

Jorm, A. F., & Griffiths, K. M. (2008). The public's stigmatizing attitudes towards people with mental disorders: How important are biomedical conceptualizations? *Acta Psychiatrica Scandinavica, 118*, 315-321. doi:10.1111/j.1600-0447.2008.01251.x

Jorm, A. F., & Oh, E. (2009). Desire for social distance from people with mental disorders: A review. *Australian and New Zealand Journal of Psychiatry, 43*, 183-200. doi:10.1080/00048670802653349

Jorm, A. F., & Wright, A. (2008). Influences on young people's stigmatising attitudes towards peers with mental disorders: National survey of young Australians and their parents. *The British Journal of Psychiatry, 192*(2), 144-9. doi:10.1192/bjp.bp.107.039404

Kanter, J., Rusch, L., & Brondino, M. (2008). Depression self-stigma: A new measure and preliminary findings. *Journal of Nervous and Mental Disease, 196*(9), 663-670. doi:10.1097/NMD.0b013e318183f8af

Karasz, A. (2005). Cultural differences in conceptual models of depression. *Social Science & Medicine, 60*(7), 1625-1635. doi:10.1016/j.socscimed.2004.08.011

Karasz, A., Dowrick, C., Byng, R., Buszewicz, M., Ferri, L., Olde Hartman, T., . . . Reeve, J. (2012). What we talk about when we talk about depression: Doctor-patient conversations and treatment decision outcomes. *The British Journal of General Practice, 62*(594), e55-63. doi:10.3399/bjgp12X616373

Karyotaki, E., Smit, Y., Holdt Henningsen, K., Huibers, M. J. H., Robays, J., de Beurs, D., & Cuijpers, P. (2016). Combining pharmacotherapy and psychotherapy or monotherapy for major depression? A meta-analysis on the long-term effects. *Journal of Affective Disorders*, *194*, 144-152. doi:10.1016/j.jad.2016.01.036

Katon, W., & Schulberg, H. (1992). Epidemiology of depression in primary care. *General Hospital Psychiatry, 14*(4), 237-247. doi:10.1016/0163-8343(92)90094-Q

Keeley, R. D., West, D. R., Tutt, B., & Nutting, P. A. (2014). A qualitative comparison of primary care clinicians' and their patients' perspectives on achieving depression care: Implications for improving outcomes. *BMC Family Practice*. doi:10.1186/1471-2296-15-13

Kemp, J. J., Lickel, J. J., & Deacon, B. J. (2014). Effects of a chemical imbalance causal explanation on individuals' perceptions of their depressive symptoms. *Behaviour Research and Therapy, 56*, 47-52. doi:10.1016/j.brat.2014.02.009

Kenny, D. A., Veldhuijzen, W., Van Der Weijden, T., LeBlanc, A., Lockyer, J., Légaré, F. & Campbell, C. (2010). Interpersonal perception in the context of doctor-patient relationships: A dyadic analysis of doctor-patient communication. *Social Science & Medicine, 70*(5), 763-768. doi: 10.1016/j.socscimed.2009.10.065

Kessing, L. V. (2004). Severity of depressive episodes according to ICD-10: Prediction of risk of relapse and suicide. *The British Journal Of Psychiatry, 184*, 153-156. doi:10.1192/bjp.184.2.153

Kessler, R. C., Amminger, G. P., Aguilar-Gaxiola, S., Alonso, J., Lee, S., & Ustun, T. B. (2007). Age of onset of mental disorders: A review of recent literature. *Current Opinion in Psychiatry*, *20*(4), 359-364. doi:10.1097/YCO.0b013e32816ebc8c

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., . . . Wang, P. S. (2003). The epidemiology of Major Depressive Disorder: Results from the national comorbidity survey replication (NCS-R). *Journal of the American Medical Association, 289*(23), 3095-3105. doi:10.1001/jama.289.23.3095

Kessler, R. C., & Bromet, E. J. (2013). The epidemiology of depression across cultures. *Annual Review of Public Health*. doi:10.1146/annurev-publhealth-031912-114409

Khan, A., Faucett, J., Lichtenberg, P., Kirsch, I., & Brown, W. A. (2012). A systematic review of comparative efficacy of treatments and controls for depression. *PLoS ONE*, *7*(7), 1-11. doi:10.1371/journal.pone.0041778

Kiloh, L., Andrews, G., & Neilson, M. (1988). The long-term outcome of depressive illness. *The British Journal of Psychiatry, 153*, 752-757. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/3256374

Kim, J-N., Park, S-C., Yoo, S-W., & Shen, H. (2010). Mapping health communication scholarship: Breadth, depth, and agenda of published research in Health Communication. *Health Communication, 25*(6-7), 487-503. doi:10.1080/10410236.2010.507160

Kinavey, C. (2006). Explanatory models of self-understanding in adolescents born with spina bifida. *Qualitative Health Research, 16*(8), 1091-1107. doi:10.1177/1049732306292092

King, M., Dinos, S., Shaw, J., Watson, R., Stevens, S., Passetti, F., … Serfaty, M. (2007). The stigma scale: Development of a standardised measure of the stigma of mental illness. *The British Journal of Psychiatry, 190*(3), 248-254. doi:10.1192/bjp.bp.106.024638

King, R. (2002). Books reconsidered: Cognitive therapy of depression. *The Australian and New Zealand Journal of Psychiatry,36*(2), 272-275. doi:10.1046/j.1440-1614.2002.t01-4-01015.x

Kirk, L., Haaga, D., Solomon, A., & Brody, F. (2000). Perceptions of depression among never-depressed and recovered-depressed people. *Cognitive Therapy and Research, 24*(5), 585-594. doi:10.1023/A:1005518229707

Kiropoulos, L., & Bauer, I. (2011). Explanatory models of depression in Greek-born and Italian-born immigrants living in Australia: Implications for service delivery and clinical practice. *Asia-Pacific Psychiatry, 3*(1), 23-29. doi:10.1111/j.1758-5872.2011.00108.x

Kirsch, I. (2010). *The emperor's new drugs: Exploding the antidepressant myth.* New York: Basic Books.

Kirsch, I., & Sapirstein, G. (1998). Listening to Prozac but hearing placebo: A meta-analysis of antidepressant medication. *Prevention & Treatment, 1*(2), doi: /10.1037//1522-3736.1.0002a

Kleinman, A. (1980). *Patients and healers in the context of culture: An exploration of the borderland between anthropology, medicine, and psychiatry*. Berkeley: University of California Press.

Kleinman, A. (2004). Culture and depression. *The New England Journal of Medicine, 351*(10), 951-953. doi:10.1056/NEJMp048078

Kleinman, A., & Benson, P. (2006) Anthropology in the clinic: The problem of cultural competency and how to fix it. *PLoS Med, 3*(10), 1673-1676. doi:10.1371/ journal.pmed.0030294.

Kleinman, A., Eisenberg, L., & Good, B. (1978). Culture, illness, and care: Clinical lessons from anthropologic and cross-cultural research. *Annals of Internal Medicine, 88*(2), 251-258. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/626456

Kline, N. S. (1958). Clinical experience with iproniazid (marsilid). *Journal of Clinical and Experimental Psychopathology*, *19*(2, Suppl. 1), 72-79. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/13549569

Koenig, H. G., & Larson, D. B. (2001). Religion and mental health: Evidence for an association. *International Review of Psychiatry*, *13*(2), 67-78. doi:10.1080/09540260124661

Kokanovic, R., Bendelow, G.,Philip, B. (2013). Depression: The ambivalence of diagnosis. *Sociology of Health and Illness, 35*(3), 377-390. doi:10.1111/j.1467-9566.2012.01486.x

Kokanovic, R., Butler, E., Halilovich, H., Palmer, V., Griffiths, F., Dowrick, C., & Gunn, J. (2013). Maps, models, and narratives: The ways people talk about depression. *Qualitative Health Research, 23*(1), 114-125. doi:10.1177/1049732312467231

Kokanovic, R., Dowrick, C., Butler, E., Herrman, H., & Gunn, J. (2008). Lay accounts of depression amongst Anglo-Australian residents and East African refugees. *Social Science and Medicine*, *66*(2), 454-466. doi:10.1016/j.socscimed.2007.08.019

Körner, H., Newman, C. E., Mao, L., Kidd, M. R., Saltman, D. C., & Kippax, S. (2011). Discourses of depression of Australian general practitioners working with gay men. *Qualitative Health Research*, *21*(8), 1051-1064. doi:10.1177/1049732311404030

Korsch, B. M. (1989). Current issues in communication research, *Health Communication, 1*(1), 5-9. doi:10.1207/s15327027hc0101_1

Korsch, B. M., Gozzi, E. K., & Francis, V. (1968). Gaps in doctor-patient communication. 1. Doctor-patient interaction and patient satisfaction. *Pediatrics, 42*(5), 855-871. Retrieved from http://pediatrics.aappublications.org/content/pediatrics/42/5/855.full.pdf

Kraepelin, E. (1921). *Manic-depressive insanity and paranoia*. Edinburgh: ES Livingstone.

Kravitz, R. L., Paterniti, D. A., Epstein, R. M., Rochlen, A. B., Bell, R. A. Cipri, C. . . . Duberstein, P. (2011). Relational barriers to depression help-seeking in primary care. *Patient Education and Counseling, 82*(2), 207-213. doi:10.1016/j.pec.2010.05.007

Krug, E. G., Mercy, J. A., Dahlberg, L. L., & Zwi, A. B. (2002) The world report on violence and health. *The Lancet*, *360*(9339), 1083-1088. doi:10.1016/S0140-6736(02)11133-0

Kruger, T. H. C., & Wollmer, M. A. (2015). Depression – An emerging indication for botulinum toxin treatment. *Toxicon*, *107*, 154-157. doi:10.1016/j.toxicon.2015.09.035

Kuhn, R. (1958). The treatment of depressive states with G 22355 (imipramine hydrochloride). *The American Journal of Psychiatry*, *115*(5), 459-464. doi:10.1176/ajp.115.5.459

Kvaale, E. P., Haslam, N., & Gottdiener, W. H. (2013). The 'side effects' of medicalization: A meta-analytic review of how biogenetic explanations affect stigma. *Clinical Psychology Review, 33*(6), 782-794. doi:10.1016/j.cpr.2013.06.002

Kvale, S. (2007). *Doing interviews*. London: Sage.

Knyazev, G. G., Kuznetsova, V. B., Savostyanov, A. N., & Dorosheva, E. A. (2017). Does collectivism act as a protective factor for depression in Russia? *Personality and Individual Differences*, *108*, 26-31. doi:10.1016/j.paid.2016.11.066

Lacasse, J. R., & Leo, J. (2005). Serotonin and depression: A disconnect between the advertisements and the scientific literature. *PLoS Medicine, 2*(12), e392-e392. doi:10.1371/journal.pmed.0020392

Lafrance, M. N. (2007). A bitter pill: A discursive analysis of women's medicalized accounts of depression. *Journal of Health Psychology, 12*, 127-140. doi:10.1177/1359105307071746

Lagacé, M., Tanguay, A., Lavallée, M. L., Laplante, J., & Robichaud, S. (2012). The silent impact of ageist communication in long term care facilities: Elders' perspectives on quality of life and coping strategies. *Journal of Aging Studies, 26*(3), 335-342. doi:10.1016/j.jaging.2012.03.002

Laing, R. D. (1969). *The divided self.* London: Random House.

Lakhan, S. E., & Callaway, E. (2010). Deep brain stimulation for obsessive-compulsive disorder and treatment-resistant depression: Systematic review. *BMC Research Notes, 3*, doi:10.1186/1756-0500-3-60-20202203

Latalova, K., Kamaradova, D., & Prasko, J. (2014) Perspectives on perceived stigma and self-stigma in adult male patients with depression. *Neuropsychiatric Disease and Treatment, 10*, 1399-1405. doi:10.2147/NDT.S54081

Lau, R. R., & Hartman, K. A. (1983). Common sense representations of common illnesses. *Health Psychology*, *2*(2), 167-185. doi:10.1037/0278-6133.2.2.167

Lawrence, V., Murray, J., Banerjee, S., Turner, S., Sangha, K., Byng, R., … Macdonald, A. (2006). Concepts and causation of depression: A cross-cultural study of the beliefs of older adults. *The Gerontologist*, *46*(1), 23-32. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/16452281

Laws, M. (2016). Explanatory models and illness experience of people living with HIV. *AIDS and Behavior*, *20*(9), 2119-2129. doi:10.1007/s10461-016-1358-1

Lebowitz, M. S. (2014). Biological conceptualizations of mental disorders among affected individuals: A review of correlates and consequences. *Clinical Psychology: Science and Practice, 21*(1), 67-83. doi:10.1111/cpsp.12056

Lebowitz, M. S., Ahn, W., & Nolen-Hoeksema, S. (2013). Fixable or fate? Perceptions of the biology of depression. *Journal of Consulting and Clinical Psychology, 81*, 518-527. doi:10.1037/a0031730

Lebowitz, M. S., Ahn, W., & Oltman, K. (2015). Sometimes more competent, but always less warm: Perceptions of biologically oriented mental-health clinicians. *International Journal of Social Psychiatry, 61*(7), 668-676. doi:10.1177/0020764015573086

Lee, A., & Hankin, B. (2009). Insecure attachment, dysfunctional attitudes, and low self-esteem predicting prospective symptoms of depression and anxiety during adolescence. *Journal of Clinical Child & Adolescent Psychology*, *38*(2), 219-231. doi:10.1080/15374410802698396

Lefaucheur, J.-P. P., Andre-Obadia, N., Antal, A., Ayache, S. S., Baeken, C., Benninger, D. H., … Garcia-Larrea, L. (2014). Evidence-based guidelines on the therapeutic use of repetitive transcranial magnetic stimulation (rTMS). *Clinical Neurophysiology*, *125*(11), 2150-2206. doi:S1388-2457(14)00296-X [pii]\r10.1016/j.clinph.2014.05.021

Lemon, T., & Smith, R. (2014). Consultation content not consultation length improves patient satisfaction. *Journal of Family Medicine and Primary Care, 3*(4), 333-339. doi:10.4103/2249-4863.148102

Leo, J., & Lacasse, J. (2008). The media and the chemical imbalance theory of depression. *Society, 45*(1), 35-45. doi:10.1007/s12115-007-9047-3

Leventhal, A. M., & Martell, C. R. (2006). *The myth of depression as disease: Limitations and alternatives to drug treatment*. Westport, CT: Praeger.

Leventhal, H., Diefenbach, M., & Leventhal, E. A. (1992). Illness cognition: Using common sense to understand treatment adherence and affect cognition interactions. Cognitive Therapy Research, 16: 143-163. doi:10.1007/BF01173486

Leventhal, H., Meyer, D., & Nerenz, D. (1980). The common-sense representations of illness danger. In S. Rachman (Ed.), *Medical Psychology* (Vol. 2, pp. 7-30). New York: Pergamon.

Leventhal, H., & Nerenz, D. (1985). The assessment of illness cognition. In P. Karoly (Ed.), *Measurement strategies in health psychology* (pp. 517-555). New York: John Wiley.

Leventhal, H., Nerenz, D., & Steele, D. (1984). Illness representations and coping with health threats. In A. Baum, & J. Singer (Eds.), *A Handbook of Psychology and Health* (Vol. 4, pp. 219-252). Hillsdale, NJ: Erlbaum.

Leximancer (2011). *Leximancer manual* (Version 4). Available from www.leximancer.com/site-media/lm/science/Leximancer_Manual_Version_4_0.pdf

Ley, P. (1983). Patients' understanding and recall in clinical communication failure. In D. Pendleton & J. Hasler, (Eds.) *Doctor-patient communication* (pp. 89-107). London: Academic Press.

Linden, M., Nather, J., & Wilms, H. U. (1988). Definition, significance and measurement of disease concepts of patients: The Disease Concept Scale for schizophrenic patients. *Fortschritte der Neurologie-Psychiatrie*, *56*(2), 35-43. doi:10.1055/s-2007-1001215

Link, B. G., & Phelan, J. C. (2001). Conceptualising stigma. *Annual Review of Sociology, 27*, 363-385. doi:10.1146/annurev.soc.27.1.363

Link, B.G., Struening, E.L., Neese-Todd, S., Asmussen, S., & Phelan, J.C. (2001). Stigma as a barrier to recovery: The consequences of stigma for the self-esteem of people with mental illnesses. *Psychiatric Services, 52*, 1621-1626. doi:10.1176/appi.ps.52.12.1621

Livingston, J. D., & Boyd, J. E. (2010). Correlates and consequences of internalized stigma for people living with mental illness: A systematic review and meta-analysis. *Social Science & Medicine, 71*(12), 2150-2161. doi: 10.1016/j.socscimed.2010.09.030

Lloyd, K. R., Jacob, K. S., Patel, V., St. Louis, L., Bhugra, D., & Mann, A. H. (1998). The development of the Short Explanatory Model Interview (SEMI) and its use among primary-care attenders with common mental disorders. *Psychological Medicine, 28*(5), 1231-1237. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/9794030

Lopez, A. D., & Murray, C. J. L. (1996). *The Global Burden of Disease: A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020*. Cambridge, MA: Harvard School of Public Health on behalf of the World Health Organization and the World Bank.

Lynch, J. M., Askew, D. A., Mitchell, G. K., & Hegarty, K. L. (2012). Beyond symptoms: Defining primary care mental health clinical assessment priorities, content and process. *Social Science and Medicine*, *74*(2), 143-149. doi:10.1016/j.socscimed.2011.08.043

Mahalik, J., & Rochlen, R. (2006). Men’s likely responses to clinical depression: What are they and do masculinity norms predict them? *Sex Roles, 55*(9), 659-667. doi:10.1007/s11199-006-9121-0

Maj, M. (2008). Depression, bereavement, and “understandable” intense sadness: Should the DSM-IV approach be revised? *American Journal of Psychiatry, 165*(11), 1373-1375. doi:10.1176/appi.ajp.2008.08071047

Makanjuola, V., Esan, Y., Oladeji, B., Kola, L., Appiah-Poku, J., Harris, B., … Gureje, O. (2016). Explanatory model of psychosis: Impact on perception of self-stigma by patients in three sub-saharan African cities. *Social Psychiatry and Psychiatric Epidemiology*, *51*(12), 1645-1654. doi:10.1007/s00127-016-1274-8

Malach, M., & Imperato, P. J. (2004). Depression and acute myocardial infarction. *Preventive Cardiology, 7*(2), 83-92. doi:10.1111/j.1520-037X.2004.3164.x

Mamdani, M., & Wilby, K. (2013). International variation in drug utilization: Antidepressant utilization in North America, Greece, and Ireland. *Journal of Health Specialties, 1*(2), 78-83. doi:10.4103/1658-600X.114689

Marcus M., Yasamy, T. M., Van Ommeren, M., Chisholm, D., & Saxena, S. (2012). *Depression: A global public health concern.* Retrieved from WHO website: http://www.who.int/mental_health/management/depression/who_paper_depression_wfmh_2012.pdf

Mathers, C., & Loncar, D. (2006). Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Medicine, 3*(11), E442. doi:10.1371/journal.pmed.0030442

Mausner-Dorsch, H., & Eaton, W. W. (2000). Psychosocial work environment and depression: Epidemiologic assessment of the demand-control model. *American Journal of Public Health, 90*(11), 1765-1770. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1446399/

Mayes, R., Bagwell, C., & Erkulwater, J. L. (2009). Medicating children: ADHD and pediatric mental health. Cambridge, MA: Harvard University Press.

McAllister, I., & Moore, R. (1989). *Ethnic prejudice in Australian society: Patterns, intensity and explanations*. Barton, ACT: Office of Multicultural Affairs.

McCoyd, J. L., & Kerson, T. S. (2006). Conducting intensive interviews using email: A serendipitous comparative opportunity. *Qualitative Social Work, 5,* 389-406. doi:10.1177/1473325006067367

McPherson, S., & Armstrong, D. (2009). Negotiating ‘depression’ in primary care: A qualitative study. *Social Science & Medicine*, *69*(8), 1137-1143. doi:10.1016/j.socscimed.2009.05.032

McPherson, S., & Armstrong, D. (2012). General practitioner management of depression: A systematic review. *Qualitative Health Research, 22*(8), 1150-1159. doi:10.1177/1049732312448540

Medical Board of Australia. (2014). *Good medical practice: A code of conduct for doctors in Australia.* Retrieved from http://www.medicalboard.gov.au/Codes-Guidelines-Policies/Code-of-conduct.aspx

Meggiolaro, E., Berardi, M., Andritsch, E., Nanni, M., Sirgo, A., Samorì, E., . . . Grassi, L. (2016). Cancer patients’ emotional distress, coping styles and perception of doctor-patient interaction in European cancer settings. *Palliative and Supportive Care, 14*(3), 204-211. doi:10.1017/S1478951515000760

Mehta, S. & Farina, A. (1997). Is being “sick” really better? Effect of the disease view of mental disorder on stigma, *Journal of Social and Clinical Psychology,*16(4), 405-419. doi:/10.1521/jscp.1997.16.4.405

Melander, H., Ahlqvist-Rastad, J., Meijer, G., & Beermann, B. (2003). Evidence b(i)ased medicine--selective reporting from studies sponsored by pharmaceutical industry: Review of studies in new drug applications. *BMJ (Clinical Research Ed.)*, *326*(7400), 1171-1173. doi:10.1136/bmj.326.7400.1171

Meyer, D., Leventhal, H., & Gutmann, M. (1985). Common-sense models of illness: The example of hypertension. *Health Psychology, 4*(2), 115-135. doi:10.1037/0278-6133.4.2.115

Mihai, A., Ricean, A., & Voidazan, S. (2014). No significant difference in depression rate in employed and unemployed in a pair-matched study design. *Frontiers in Public Health*, 2, 93. doi:10.3389/fpubh.2014.00093

Mikesell, L. (2013). Medicinal relationships: Caring conversation. *Medical Education,47*(5), 443-52. doi:10.1111/medu.12104

Mikulincer, M., & Shaver, P. R. (2003). The attachment behavioral system in adulthood: Activation, psychodynamics, and interpersonal processes. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 35, pp. 53-152). San Diego, CA: Elsevier Academic Press. doi:10.1016/S0065-2601(03)01002-5

Mikulincer, M., & Shaver, P. R. (2012). An attachment perspective on psychopathology. *World Psychiatry, 11*(1), 11-15. doi:10.1016/j.wpsyc.2012.01.003

Miller, W. R., Roselini, R. A., & Seligman, M. E. (1977). Learned helplessness and depression. In J. D. Maser & M. E. Seligman (Eds.), *Psychopathology: Experimental models* (pp. 104-130). San Francisco, CA: Freeman.

Mitchell, A. J., Vaze, A., & Rao, S. (2009). Clinical diagnosis of depression in primary care: a meta-analysis. *The Lancet*, *374*(9690), 609-619. doi:10.1016/S0140-6736(09)60879-5

Mittman, R. & Cain, M. (2001). The future of the internet in health care: A five-year forecast. In R. E. Rice & J. E. Katz (Eds.), *The Internet and health communication: Experiences and expectations* (pp. 47-74). Thousand Oaks, CA: Sage. doi:10.4135/9781452233277.n2

Moffitt, T. E., Arseneault, L., Jaffee, S. R., Kim-Cohen, J., Koenen, K. C., … Viding, E. (2008). DSM-V conduct disorder: Research needs for an evidence base. *Journal of Child Psychology and Psychiatry*, *49*(1), 3-33. doi:10.1111/j.1469-7610.2007.01823.x

Moffitt, T. E., Caspi, A., Taylor, A., Kokaua, J., Milne, B. J., Polanczyk, G., & Poulton, R. (2010). How common are common mental disorders? Evidence that lifetime prevalence rates are doubled by prospective versus retrospective ascertainment. *Psychological Medicine*, *40*(6), 899-909. doi:10.1017/S0033291709991036

Mokkink, L. B., Terwee, C. B., Patrick, D. L., Alonso, J., Stratford, P. W., Knol, D. L., … De Vet, H. C. W. (2010). The COSMIN study reached international consensus on taxonomy, terminology, and definitions of measurement properties for health-related patient-reported outcomes, *Journal of Clinical Epidemiology, 63*(7), 737-745, doi:10.1016/j.jclinepi.2010.02.006.

Mola, E., De Bonis, J. A., & Giancane, R. (2008). Integrating patient empowerment as an essential characteristic of the discipline of general practice/family medicine. *European Journal of General Practice, 2008, 14*(2), 89-94. doi:10.1080/13814780802423463

Montejo-González, A. L., Llorca, G., Izquierdo, J. A., Ledesma, A., Bousoño, M., Calcedo, A., . . . Vicens, E. (1997). SSRI-induced sexual dysfunction: Fluoxetine, paroxetine, sertraline, and fluvoxamine in a prospective, multicenter, and descriptive clinical study of 344 patients. *Journal of Sex & Marital Therapy, 23*(3), 176-94. doi:10.1080/00926239708403923

Montgomery, S., Henry, J., Mcdonald, G., Dinan, T., Lader, M., Hindmarch, I., . . . Nutt, D. (1994). Selective serotonin reuptake inhibitors: Meta-analysis of discontinuation rates. *International Clinical Psychopharmacology, 9*(1), 47-53. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/8195583

Moussavi, S., Chatterji, S., Verdes, E., Tandon, A., Patel, V., & Ustun, B. (2007). Depression, chronic diseases, and decrements in health: Results from the world health surveys. *The Lancet, 370*(9590), 851-858. doi:10.1016/S0140-6736(07)61415-9

Mueller, T., Leon, A., Keller, M., Solomon, D., Endicott, J., Coryell, W., . . . Maser, J. (1999). Recurrence after recovery from Major Depressive Disorder during 15 years of observational follow-up. *American Journal of Psychiatry,156*(7), 1000-1006. doi:10.1176/ajp.156.7.1000

Muller, J., Pryor, W., Gibbons, J., & Orgain, E. (1955). Depression and anxiety occurring during rauwolfia therapy. *Journal of the American Medical Association, 159*(9), 836-839. doi:10.1001/jama.1955.02960260006002

Mumford, D. (1995). Cultural issues in assessment and treatment. *Current Opinion in Psychiatry, 8*(2), 134-137. doi:10.1097/00001504-199503000-00016

Narli, S. (2010). An alternative evaluation method for Likert-type attitude scales: Rough set data analysis. *Scientific Research and Essays, 5*(6), 519-528. Retrieved from http://www.academicjournals.org/article/article1380627186_Narli.pdf

Neitzke, A. (2016). An illness of power: Gender and the social causes of depression. *Culture, Medicine, and Psychiatry, 40*(1), 59-73. doi:10.1007/s11013-015-9466-3

Nelson, C., & Fife, E. (2013). RateMyProfessors.com: A validity exercise. *Communication Teacher, 27*(1), 6-10. doi:10.1080/17404622.2012.730621

Nestler, E. J., Barrot, M., Dileone, R. J., Eisch, A. J., Gold, S. J., & Monteggia, L. M. (2002). Neurobiology of Depression. *Neuron*, *34*(1), 13-25. doi:10.1016/S0896-6273(02)00653-0

Neuman, W. L. (2014). *Social research methods: Qualitative and quantitative approaches* (7th ed.). Boston, MA: Pearson Education.

Ng, Q. X., Venkatanarayanan, N., & Ho, C. Y. X. (2017). Clinical use of Hypericum perforatum (St John's Wort) in depression: A meta-analysis. *Journal of Affective Disorders*, *210*, 211-221. doi:10.1016/j.jad.2016.12.048

Nolen-Hoeksema, S. (1987). Sex differences in unipolar depression: Evidence and theory. *Psychological Bulletin, 101*, 259-282. doi:10.1037/0033-2909.101.2.259

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*(4), 569-582. doi:10.1037/0021-843X.100.4.569

Nosè, M., Mazzi, M., Esposito, A., Bianchini, E., Petrosemolo, M., Ostuzzi, P., . . . Barbui, M. (2012). Adverse effects of antipsychotic drugs: Survey of doctors' versus patients' perspective. *Social Psychiatry and Psychiatric Epidemiology,47*(1), 157-164. doi:10.1007/s00127-010-0320-1

Novick, G. (2008). Is there a bias against telephone interviews in qualitative research? *Research in Nursing and Health*, *31*(4), 391-398. doi:10.1002/nur.20259

Nunnally, J. C., & Bernstein, I. H. (1994). The assessment of reliability. In J. Vaicunas & J. R. Belser (Eds.), *Psychometric theory* (3rd ed., pp. 248-292). New York: McGraw-Hill.

Nussbaum, J. F., & Fisher, C. L. (2009). A communication model for the competent delivery of geriatric medicine. *Journal of Language and Social Psychology, 28*(2), 190-208. doi:10.1177/0261927X08330609

Nutt, D. J. (2008). Relationship of neurotransmitters to the symptoms of major depressive disorder. *Journal of Clinical Psychiatry, 69* (Suppl. E1), 4-7. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/18494537

Nuyen, J., Volkers, A. C., Verhaak, P. F. M., Schellevis, F. G., Groenewegen, P. P., & Van Den Bos, G. A. M. (2005). Accuracy of diagnosing depression in primary care: The impact of chronic somatic and psychiatric co-morbidity. *Psychological Medicine*, *35*(8). doi:10.1017/S0033291705004812

Ogden, J., Boden, J., Caird, R., Chor, C., Flynn, M., Hunt, M., . . . Ogden, V. (1999). "You're depressed"; "No I'm not": GPs' and patients' different models of depression. *British Journal of General Practice, 49*(439), 123-124. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1313347/

Okello, E. S., & Neema, S. (2007). Explanatory models and help-seeking behavior: Pathways to psychiatric care among patients admitted for depression in Mulago hospital, Kampala, Uganda. *Qualitative Health Research*, *17* (1), 14-25. doi:10.1177/1049732306296433

Olfson, M., & Marcus, S. C. (2010). National trends in outpatient psychotherapy. *American Journal of Psychiatry, 167*, 1456-1463. doi:10.1176/appi.ajp.2010.10040570

Olfson, M., Marcus, S. C., Druss, B., Elinson, L., Tanielian, T., & Pincus, H. A. (2002). National trends in the outpatient treatment of depression. *Journal of the American Medical Association, 287*(2), 203-209. doi:10.1001/jama.287.2.203

Opdenakker, R. (2006). Advantages and disadvantages of four interview techniques in qualitative research. *Qualitative Social Research, 7*, 11. Retrieved from http://www.qualitative-research.net/fqs-texte/ 4-06/06-4-11-e.htm.

Östman, M. (2008). Severe depression and relationships: The effect of mental illness on sexuality. *Sexual and Relationship Therapy, 23*(4), 355-363. doi:10.1080/14681990802419266

Palitsky, D. O., Mota, N. C., Afifi, T., Downs, A., & Sareen, J. (2013). The association between adult attachment style, mental disorders, and suicidality: Findings from a population-based study. *The Journal of Nervous and Mental Disease*, *201*(7), 579-586. doi:10.1097/NMD.0b013e31829829ab

Pallant, J. (2013). *SPSS survival manual: A step by step guide to data analysis using IBM SPSS* (5th ed.). Crows Nest, NSW: Allen & Unwin.

Palmer, V., Johnson, C., Furler, J., Densley, K., Potiriadis, M., & Gunn, J. (2014). Written plans: An overlooked mechanism to develop recovery-oriented primary care for depression? *Australian Journal of Primary Health, 20*(3), 241-249. doi:10.1071/PY12128

Parker, G. (2004). Evaluating treatments for the mood disorders: Time for the evidence to get real. *Australian and New Zealand Journal of Psychiatry, 38(6),* 408-414. doi:10.1080/j.1440-1614.2004.01386.x

Parker, G. (2005). Beyond major depression. *Psychological Medicine, 35*, 467-474, doi:10.1017/S0033291704004210

Paul, M. (2015). New blood test a milestone for diagnosing depression. *WhatDoctorsKnow.Com*. Retrieved March 29, 2017, from http://www.chicagotribune.com/lifestyles/health/sns-201504280000--tms--premhnstr--k-h20150429-20150429-story.html

Paykel, E. S. (2002). Mood disorders: Review of current diagnostic systems. *Psychopathology, 35*(2–3), 94-99. doi:10.1159/000065126

Pearson, A., Saini, P., Da Cruz, D., While, D., Swinson, N., Williams, A., . . . Miles, C. (2009). Primary care contact prior to suicide in individuals with mental illness. *British Journal of General Practice, 59*(568), 825-832. doi:10.3399/bjgp09X472881

Pendleton, D. (1985). Towards more effective medical practice. *Journal of Applied Communication Research, 13*(2), 96-102. doi:10.1080/00909888509388426

Pendleton, D., Brouwer, H., & Jaspars, J. (1983). Communication difficulties: The doctor's perspective. *Journal of Language and Social Psychology, 2*(1), 17-36. doi:10.1177/0261927X8300200102

Penn-Edwards, S. (2010). Computer aided phenomenography: The role of Leximancer computer software in phenomenographic investigation. *Qualitative Report, 15*(2), 252-267. Retrieved from http://www.nova.edu/ssss/QR/QR15-2/penn-edwards.pdf

Pereira, B., Andrew, G., Pednekar, S., Pai, R., Pelto, P., & Patel, V. (2007). The explanatory models of depression in low income countries: Listening to women in India. *Journal of Affective Disorders, 102* (1): 209-218. doi:10.1016/j.jad.2006.09.025

Pescosolido, B. A., Martin, J. K., Long, J. S., Medina, T. R., Phelan, J. C., & Link, B. G. (2010). "A disease like any other"? A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. *American Journal of Psychiatry*, *167*(11), 1321-1330. doi:10.1176/appi.ajp.2010.09121743

Petersen, A. J., & Madsen, O. (2017). Depression: Diagnosis and suffering as process. *Nordic Psychology, 69*(1), 19-32. doi:10.1080/19012276.2016.1267913

Peterson, C., Maier, S. F., & Seligman, M. E. P. (1993). *Learned helplessness: A theory for the age of personal control.* New York: Oxford University Press.

Peterson, C., & Seligman, M. E. (1984). Causal explanations as a risk factor for depression: Theory and evidence. *Psychological Review, 91*(3), 347-374. doi:10.1037/0033-295X.91.3.347

Peterson, R. (1994). A meta-analysis of Cronbach's coefficient alpha. *Journal of Consumer Research, 21*(2), 381-391. Retrieved from http://www.jstor.org/stable/2489828

Piccinelli, M., & Wilkinson, G. (2000). Gender differences in depression: Critical review. *The British Journal of Psychiatry, 177*, 486-492. doi:10.1192/bjp.177.6.486

Pies, R. (2014). The bereavement exclusion and DSM-5: An update and commentary. *Innovations in Clinical Neuroscience, 11*(7-8), 19-22. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4204469/

Platt, F., & Keating, K. (2007). Differences in physician and patient perceptions of uncomplicated UTI symptom severity: Understanding the communication gap. *International Journal of Clinical Practice, 61*(2), 303-308. doi:10.1111/j.1742-1241.2006.01277.x

Pollock, K., & Grime, J. (2002). Patients' perceptions of entitlement to time in general practice consultations for depression: Qualitative study. *BMJ (Clinical Research Ed.)*, *325*(7366), 687. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/12351362

Poulakou-Rebelakou, E., Tsiamis, C., Panteleakos, G., & Ploumpidis, D. (2009). Lycanthropy in Byzantine times (AD 330—1453). *History of Psychiatry, 20*(4), 468-479. doi:10.1177/0957154X08338337

Quinn, D., & Chaudoir, S. (2009). Living with a concealable stigmatized identity: The impact of anticipated stigma, centrality, salience, and cultural stigma on psychological distress and health. *Journal of Personality and Social Psychology, 97*(4), 634-651. doi:10.1037/a0015815

Rasch, D., & Guiard, V. (2004). The robustness of parametric statistical methods. P*sychology Science, 46*(2), 175-208. Retrieved from http://www.pabst-publishers.de/psychology-science/2-2004/ps_2_2004_175-208.pdf

Read, J., Cartwright, C., Gibson, K., Shiels, C. & Magliano, L. (2015). Beliefs of people taking antidepressants about the causes of their own depression. *Journal of Affective Disorders,174*, 150-156. doi:10.1016/j.jad.2014.11.009

Reavley, N., & Jorm, A. (2011). *National Survey of Mental Health Literacy and Stigma.* Department of Health and Ageing, Canberra. Retrieved from http://pmhg.unimelb.edu.au/__data/assets/pdf_file/0007/636496/National_Mental_Health_Literacy_Survey_Monograph.pdf

Redei, E. E., Andrus, B. M., Kwasny, M. J., Seok, J., Cai, X., Ho, J., & Mohr, D. C. (2014). Blood transcriptomic biomarkers in adult primary care patients with major depressive disorder undergoing cognitive behavioral therapy. *Translational Psychiatry*, *4*(9), e442. doi:10.1038/tp.2014.66

Rholes, W. S., Simpson, J. A., Kohn, J. L., Wilson, C. L., Martin, A. M., Tran, S., & Kashy, D. A. (2011). Attachment orientations and depression: A longitudinal study of new parents. *Journal of Personality and Social Psychology*, *100*(4), 567-586. doi:10.1037/a0022802

Richards, D. (2011). Prevalence and clinical course of depression: A review. *Clinical Psychology Review, 31*(7), 1117-1125. doi:10.1016/j.cpr.2011.07.004

Richards, S., & Perri, M. (2002). *Depression: A primer for practitioners*. Thousand Oaks, CA: Sage. doi:10.4135/9781452229393

Ritsher, J. B., Otilingam, P. G., & Grajales, M. (2003). Internalized stigma of mental illness: Psychometric properties of a new measure. *Psychiatry Research, 121*(1), 31-49. doi:10.1016/j.psychres.2003.08.008

Rivera, J. (Producer), & Docter, P., & Del Carmen, R. (Directors). (2015). *Inside Out* [Motion Picture]. United States: Walt Disney Pictures Pixar Animation Studios.

Robinson, W. D., Geske, J. A., Prest, L. A., & Barnacle, R. (2005). Depression treatment in primary care. *The Journal of the American Board of Family Practice, 18*(2), 79-86. doi:10.3122/jabfm.18.2.79

Rogers, A., May, C., & Oliver, D. (2001). Experiencing depression, experiencing the depressed: The separate worlds of patients and doctors. *Journal of Mental Health, 10*(3), 317-333. doi: 10.1080/09638230125545

Rosenstock, I. (1966). Why people use health services. *The Milbank Memorial Fund Quarterly, 44*(3), 94-127. doi:10.2307/3348967

Rossom, R. C., Shortreed, S., Coleman, K. J., Beck, A., Waitzfelder, B. E., Stewart, C., . . . Simon, G. E. (2016). Antidepressant adherence across diverse populations and healthcare settings. *Depression and Anxiety, 33*(8), 765-74. doi:10.1002/da.22532

Roter, D. L., Erby, L. H., Adams, A., Buckingham, C. D., Vail, L., Realpe, A., . . . Hall, J. A. (2014). Talking about depression: An analogue study of physician gender and communication style on patient disclosures. *Patient Education and Counseling, 96*(3), 339-345. doi:10.1016/j.pec.2014.05.006

Rounsaville, B. J., Alarcon, R. D., Andrews, G., Jackson, J. S., Kendell, R. E., Kendler, K. (2002). Basic nomenclature issues for DSM-V. In D. J. Kupfer, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V* (pp. 1-30). Washington, DC: American Psychiatric Association. Retrieved from http://apsychoserver.psych.arizona.edu/jjbareprints/psyc621/rounsaville_et_al_research_agenda_dsm-v_2002.pdf

Ruhe, H. G., Mason, N. S., & Schene, A. H. (2007). Mood is indirectly related to serotonin, norepinephrine and dopamine levels in humans: A meta-analysis of monoamine depletion studies. *Molecular Psychiatry*, *12*(4), 331-359. doi:10.1038/sj.mp.4001949

Rusch, L. C., Kanter, J. W., & Brondino, M. J. (2009). A comparison of contextual and biomedical models of stigma reduction for depression with a nonclinical undergraduate sample. *Journal of Nervous and Mental Disease, 197*, 104-110. doi:10.1097/NMD .0b013e318192416f

Rüsch, N., Angermeyer, M., & Corrigan, P. W. (2005). Mental illness stigma: Concepts, consequences, and initiatives to reduce stigma. *European Psychiatry, 20,* 529-539. doi:10.1016/j.eurpsy.2005.04.004

Safer, D. J., & Zito, J. M. (2007). Do antidepressants reduce suicide rates? *Public Health, 121*(4), 274-277. doi:10.1016/j.puhe.2006.09.024

Saito, M., Iwata, N., Kawakami, N., Matsuyama, Y., Ono, Y., Nakane, Y., … Kikkawa, T. (2010). Evaluation of the DSM-IV and ICD-10 criteria for depressive disorders in a community population in Japan using item response theory. *International Journal of Methods in Psychiatric Research*, *19*(4), 211-222. doi:10.1002/mpr.320

Salmon, P., Peters, S., & Stanley, I. (1999). Patients' perceptions of medical explanations for somatisation disorders: Qualitative analysis. *British Medical Journal*, *318*(7180), 372-376. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC27727/

Sax, L. J., Gilmartin, S. K., Lee, J. J., & Hagedorn, L. S. (2003). Using web surveys to reach community college students: An analysis of response rates and response bias. *Community College Journal of Research and Practice, 32*(9), 712-729. doi:10.1080/10668920802000423

Schnittker, J. (2008). An uncertain revolution: Why the rise of a genetic model of mental illness has not increased tolerance. *Social Science & Medicine, 67*(9), 1370-1381. doi:10.1016/j.socscimed.2008.07.007

Schomerus, G., Matschinger, H., & Angermeyer, M. C. (2009). The stigma of psychiatric treatment and help-seeking intentions for depression*.* *European Archives of Psychiatry and Clinical Neuroscience, 259*(5), 298-306. doi:10.1007/s00406-009-0870-y

Schomerus, G., Schwahn, C., Holzinger, A., Corrigan, P. W., Grabe, H. J., Carta, M. G., & Angermeyer, M. C. (2012). Evolution of public attitudes about mental illness: A systematic review and meta-analysis. *Acta Psychiatrica Scandinavica*, *125*(6), 440-452. doi:10.1111/j.1600-0447.2012.01826.x

Schotte, C. K. W., Van Den Bossche, B., De Doncker, D., Claes, S., & Cosyns, P. (2006). A biopsychosocial model as a guide for psychoeducation and treatment of depression. *Depression and Anxiety*, *23*, 312-324. doi:10.1002/da.20177

Schouten, B. C., & Meeuwesen, L. (2006). Cultural differences in medical communication: A review of the literature. *Patient Education and Counseling, 64*(1), 21-34. doi:10.1016/j.pec.2005.11.014

Schreiber, R., & Hartrick, G. (2002). Keeping it together: How women use the biomedical explanatory model to manage the stigma of depression. *Issues in Mental Health Nursing, 23*(2), 91-91. doi:10.1080/016128402753542749

Schumann, I., Schneider, A., Kantert, C., Löwe, B., & Linde, K. (2012). Physicians' attitudes, diagnostic process and barriers regarding depression diagnosis in primary care: A systematic review of qualitative studies. *Family Practice, 29*(3), 255-263. doi:10.1093/fampra/cmr092

Schwalbe, M. L., & Wolkomir, M. (2002). Interviewing men. In J. F. Gubrium and J. A. Holstein (Eds.), *Handbook of interview research* (pp. 203-219). Thousand Oaks, CA: Sage.

Schwenk, T. L., Evans, D. K., Laden, S., & Lewis, L. (2004). Treatment outcome and physician-patient communication in primary care patients with chronic, recurrent depression. *American Journal of Psychiatry, 161*(10), 1892-1901. doi:10.1176/ajp.161.10.1892

Sedgwick, P. (2015). A comparison of parametric and non-parametric statistical tests. *British Medical Journal, 350*, h2053. doi:10.1136/bmj.h2053

Seligman, M. E. P. (1972). Learned helplessness. *Annual Review of Medicine, 23*, 407-12. doi:10.1146/annurev.me.23.020172.002203

Seligman, M. E. P. (1975). *Helplessness: On depression, development, and death*. San Francisco: W. H. Freeman.

Sharf, B. F., & Street, R. L., Jr. (1997). The patient as a central construct: Shifting the emphasis. *Health Communication, 9*(1), 1-11. doi:10.1207/s15327027hc0901_1

Sharf, J., Primavera, L. H., & Diener, M. J. (2010). Dropout and therapeutic alliance: A meta-analysis of adult individual psychotherapy. *Psychotherapy, 47*, 637-645. doi:doi:10.1037/a0021175

Shaw, I. (2002). How lay are lay beliefs? *Health, 6*(3), 287-299. doi:10.1177/136345930200600302

Shore, P., Silver, S., & Brodie, B. (1955). Interaction of reserpine, serotonin, and lysergic acid diethylamide in brain. *Science, 122*(3163), 284-5. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/13246633

Shryock, R. H. (1945). The psychiatry of Benjamin Rush. *The American Journal of Psychiatry, 101*(4), 429-432. doi:10.1176/ajp.101.4.429

Simard, L.M., Taylor, D.M. & Giles, H. (1976). Attribution processes and interpersonal accommodation in a bilingual setting. *Language and Speech, 19*(4), 374-387. doi:10.1177/002383097601900408

Simon, G. E., Chisholm, D., Treglia, M., & Bushnell, D. (2002). Course of depression, health services costs, and work productivity in an international primary care study. *General Hospital Psychiatry, 24*(5), 328-335. doi:10.1016/S0163-8343(02)00201-3

Simpson, J. A., Rholes, W. S., Campbell, L., Tran, S., & Wilson, C. L. (2003). Adult attachment, the transition to parenthood, and depressive symptoms. *Journal of Personality and Social Psychology*, *84*, 1172-1187. doi:10.1037/0022-3514.84.6.1172

Simpson, J. A., Rholes, W. S., & Nelligan, J. S. (1992). Support seeking and support giving within couples in an anxiety-provoking situation: The role of attachment styles. *Journal of Personality and Social Psychology, 62*(3), 434-446. doi:10.1037/0022-3514.62.3.434

Singh, I., & Rose, N. (2009). Biomarkers in psychiatry. *Nature, 460*(7252), 202-7. doi:10.1038/460202a.

Sirey, J. A., Bruce, M. L., Alexopoulos, G. S., Perlick, D. A., Friedman, S. J., & Meyers, B. S. (2001). Stigma as a barrier to recovery: Perceived stigma and patient-rated severity of illness as predictors of antidepressant drug adherence. *Psychiatric Services, 52*, 1615-1620. doi:10.1176/appi.ps.52.12.1615

Sirey, J., Bruce, M., Alexopoulos, G., Perlick, D., Raue, P., Friedman, S., & Meyers, B. (2001). Perceived stigma as a predictor of treatment discontinuation in young and older outpatients with depression. *American Journal of Psychiatry, 158*(3), 479-481. doi:10.1176/appi.ajp.158.3.479

Skultans, V. (2003). From damaged nerves to masked depression: Inevitability and hope in Latvian psychiatric narratives. *Social Science & Medicine, 56*(12), 2421-2431. doi:10.1016/S0277-9536(02)00245-9

Smith, A. E. & Humphreys, M. S. (2006). Evaluation of unsupervised semantic mapping of natural language with Leximancer concept mapping. *Behavior Research Methods, 38*, 262-279 doi:10.3758/BF03192778

Smith, C. A., Hay, P. P. J., & MacPherson, H. (2010). Acupuncture for depression. *Cochrane Database of Systematic Reviews* (1). doi:10.1002/14651858.CD004046.pub3

Snyder, C. R. (1995). Conceptualizing, measuring, and nurturing hope. *Journal of Counseling and Development*, *73*(1), 355-360. doi:10.1002/j.1556-6676.1995.tb01764.x

Sodergren, S., & Hyland, M. (2000). What are the positive consequences of illness? *Psychology & Health, 15*(1), 85-97. doi:10.1080/08870440008400290

Soliz, J., & Giles, H. (2014). Relational and identity processes in communication: A contextual and meta-analytical review of communication accommodation theory. In E. Cohen (Ed.), *Communication Yearbook 38* (pp. 106-143). Thousand Oaks, CA: Sage.

Spijker, J., De Graaf, R., Bijl, R. V., Beekman, A. T. F., Ormel, J., & Nolen, W. A. (2002). Duration of major depressive episodes in the general population: Results from The Netherlands Mental Health Survey and Incidence Study (NEMESIS). *The British Journal of Psychiatry, 181*, 208-213. doi:10.1192/bjp.181.3.208

Spitzer, R., Endicott, J., & Robins, E. (1978). Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry,35*(6), 773-782. doi:10.1001/archpsyc.1978.01770300115013

Staub, M. E. (2011). *Madness is civilization: When the diagnosis was social, 1948 -1980.* Chicago: University of Chicago Press.

Stephenson, C. P., Karanges, E., & McGregor, I. S. (2013). Trends in the utilisation of psychotropic medications in Australia from 2000 to 2011. *The Australian and New Zealand Journal of Psychiatry, 47*(1), 74-87. doi:10.1177/0004867412466595.

Stevelink, S. A., Wu, I. C., Voorend, C. G., & Van Brakel, W. H. (2012). The psychometric assessment of internalized stigma instruments: A systematic review. *Stigma Research and Action*, *(2)*2, 100-118. doi:10.5463/SRA.v1i1.11

Stewart, M. (2001). Towards a global definition of patient centred care: The patient should be the judge of patient-centred care. *British Medical Journal, 322*(7284), 444-445. doi:10.1136/bmj.322.7284.444

Stewart, M., Brown, J. B., Donner, A., McWhinney, I. R., Oates, J., Weston, W. W., & Jordan, J. (2000). The impact of patient-centered care on outcomes. *Journal of Family Practice, 49*(9), 796-804. Retrieved from http://www.mdedge.com/jfponline/article/60893/impact-patient-centered-care-outcomes

Stewart, M. A. (1995). Effective physician-patient communication and health outcomes: A review. *Canadian Medical Association Journal, 152*(9), 1423-1433. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1337906/

Street, R. L., Jr. (2001). Active patients as powerful communicators. In W. P. Robinson & H. Giles (Eds.), *The New Handbook of Language and Social Psychology* (pp. 541-560). New York: John Wiley.

Street, R. L., Jr. (2003). Communication in medical encounters: An ecological perspective. In T. Thompson, A. Dorsey, K. Miller, & R. Parrott (Eds.), *Handbook of Health Communication* (pp. 63-89). Mahwah, NJ: Lawrence Erlbaum.

Street, R. L., Jr., & Haidet, P. (2011). How well do doctors know their patients? Factors affecting physician understanding of patients' health beliefs. *Journal of General Internal Medicine, 26*(1), 21-27. doi:10.1007/s11606-010-1453-3

Street, R. L., Krupat, L., Bell, E., Kravitz, R., & Haidet, A. (2003). Beliefs about control in the physician-patient relationship. *Journal of General Internal Medicine, 18*(8), 609-616. doi:10.1046/j.1525-1497.2003.20749.x

Street, R. L., Makoul, G., Arora, N. K., & Epstein, R. M. (2009). How does communication heal? Pathways linking clinician-patient communication to health outcomes. *Patient Education and Counseling, 74*(3), 295-301. doi:10.1016/j.pec.2008.11.015

Street, H., Sheeran, P., & Orbell, S. (1999). Conceptualizing depression: An integration of 27 theories. *Clinical Psychology & Psychotherapy, 6*(3), 175-193. doi:10.1002/(SICI)1099-0879(199907)6:3<175::AID-CPP200>3.0.CO;2-Q

Sturges, J. E., & Hanrahan, K. J. (2004). Comparing telephone and face-to-face qualitative interviewing: A research note. *Qualitative Research, 4*, 107-118. doi:10.1177/1468794104041110

Styron, W. (1990). *Darkness visible: A memoir of madness.* New York: Random House.

Substance Abuse and Mental Health Services Administration. (2012). *Results from the 2011 National Survey on Drug Use and Health: Mental health findings* (NSDUH Series H-45, HHS Publication No. [SMA] 12-4725). Rockville, MD: Author.

Sumathipala, A., Siribaddana, S., Hewege, S., Sumathipala, K., Prince, M., & Mann, A. (2008). Understanding the explanatory model of the patient on their medically unexplained symptoms and its implication on treatment development research: A Sri Lanka study. *BMC Psychiatry*, *8*(8), 54. doi:10.1186/1471-244X-8-54

Sweet, L. (2002). Telephone interviewing: Is it compatible with interpretive phenomenological research? *Contemporary Nurse, 12*, 58-63. doi:10.5172/conu.12.1.58

Swenson, S. L., Zettler, P., & Lo, B. (2006). 'She gave it her best shot right away': Patient experiences of biomedical and patient-centered communication. *Patient Education and Counseling, 61*(2), 200-211. doi:10.1016/j.pec.2005.02.019

Szasz, T. (1960). The myth of mental illness. *American Psychologist, 15*(2), 113-118. doi:10.1037/h0046535

Tajfel, H., & Turner, J. C. (1986). The social identity theory of intergroup behaviour. In S. Worchel & W. G. Austin (Eds.), *Psychology of Intergroup Relations* (pp. 7-24). Chicago, IL: Nelson-Hall.

Tawney, R. H. (1931). *Equality*. London: Allen & Unwin.

Teasdale, J., Segal, Z., Williams, J., Ridgeway, V., Soulsby, J., Lau, M., & Kendall, Philip C. (2000). Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *Journal of Consulting and Clinical Psychology, 68*(4), 615-623. doi:10.1037/0022-006X.68.4.615

Terwee, C. B., Bot, S. D., De Boer, M. R., Van Der Windt, D.A., Knol, D. L., Dekker, J., … De Vet, H. C. W. (2007). Quality criteria were proposed for measurement properties of health status questionnaires. *Journal of Clinical Epidemiology, 60*, 34-42. doi:10.1016/j.jclinepi.2006.03.012

Thachuk, A. K. (2011). Stigma and the politics of biomedical models of mental illness. *International Journal of Feminist Approaches to Bioethics, 4*(1), 140-163. doi:10.2979/intjfemappbio.4.1.140

The advantages and disadvantages of questionnaires. (n.d.). Retrieved from http://libweb.surrey.ac.uk/library/skills/Introduction%20to%20Research%20and%20Managing%20Information%20Leicester/page_51.htm

Thomas, R. K. (2006). *Health Communication*. Dordrecht: Springer. doi:10.1007/b136859

Thomas-MacLean, R., & Stoppard, J. M. (2004). Physicians' constructions of depression: Inside/outside the boundaries of medicalization. *Health*, *8*(3), 275-293. doi:10.1177/1363459304043461

Thompson, T. L. (1984) The invisible helping hand: The role of communication in the health and social service professions. *Communication Quarterly, 32*(2), 148-163, doi:10.1080/01463378409369545

Thompson, T. L., Cusella, L. P., & Southwell, B. G. (2014). Method matters. In B. B. Whaley (Ed.) *Research methods in health communication: Principles and application* (pp. 3-19). New York: Routledge.

Thompson, T. L., & Zorn, M. (2010). Welcome to the 100th issue of health communication! *Health Communication, 25*(6-7), 483-486. doi:10.1080/10410236.2010.497986

Thompson, V. L. S., Bazile, A., & Akbar, M. (2004). African Americans' perceptions of psychotherapy and psychotherapists. *Professional Psychology: Research and Practice*, *35*(1), 19-26. doi:10.1037/0735-7028.35.1.19

Trujols, J., Feliu-Soler, A., de Diego-Adeliño, J., Portella, M. J., Cebrià, Q., Soler, J., … Perez, V. (2013). A psychometric analysis of the Clinically Useful Depression Outcome Scale (CUDOS) in Spanish patients. *Journal of Affective Disorders*. doi:10.1016/j.jad.2013.08.007

Trull, T. J. (2004). *Clinical psychology* (7th ed.). Belmont, CA: Wadsworth.

Tucker, J. R., Hammer, J. H., Vogel, D. L., Bitman, R. L., Wade, N. G., & Maier, E. J. (2013). Disentangling self-stigma: are mental illness and help-seeking self-stigmas different? *Journal of Counseling Psychology, 60*, 520-531. doi:10.1037/a0033555

Tuckett, D. (1982). *Final report on the patient project*. London: Health Education Council.

Turner, E., Matthews, A., Linardatos, E., Tell, R., & Rosenthal, R. (2008). Selective publication of antidepressant trials and its influence on apparent efficacy. *The New England Journal of Medicine, 358*(3), 252-260. doi:10.1056/NEJMsa065779

Tursi, M., Baes, C., Camacho, F. R., Tofoli, S. M., & Juruena, M. F. (2013). Effectiveness of psychoeducation for depression: A systematic review. *Australian & New Zealand Journal of Psychiatry, 47*(11), 1019-1031. doi:10.1177/0004867413491154

Üstün, T. B., Ayuso-Mateos, J. L., Chatterji, S., Mathers, C., & Murray, C. J. L. (2004). Global burden of depressive disorders in the year 2000. *The British Journal of Psychiatry*, *184*(5), 386 LP-392. doi:10.1192/bjp.184.5.386

Uzun, S., Kozumplik, O., Topic, R., & Jakovljevic, M. (2009). Depressive disorders and comorbidity: Somatic illness vs. side effect. *Psychiatria Danubina*, *21*(3), 391-398. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/19794363

Van Praag, H. M. (2002). Why has the antidepressant era not shown a significant drop in suicide rates? *Crisis, 23* (2), 77-82. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/12500893

Van Vliet, L. M., Van Der Wall, E., Albada, A., Spreeuwenberg, P. M. M., Verheul, W., & Bensing, J. M. (2012). The validity of using analogue patients in practitioner-patient communication research: Systematic review and meta-analysis. *Journal of General Internal Medicine, 27*(11), 1528-1543. doi:10.1007/s11606-012-2111-8

Van Voorhees, B. W., Fogel, J., Houston, T. K., Cooper, L. A., Wang, N.-Y., & Ford, D. E. (2005). Beliefs and attitudes associated with the intention to not accept the diagnosis of depression among young adults. *Annals of Family Medicine*, *3*(1), 38-46. doi:10.1370/afm.273

Veldhuijzen, W., Mogendorff, K., Ram, P., Van Der Weijden, T.T., Elwyn, G., & Ven Der Vleuten, C.P.M. (2013). How doctors move from generic goals to specific communicative behavior in real practice consultations. *Patient Education and Counseling, 90*(2), 170-176. doi:10.1016/j.pec.2012.10.005

Vetere, A., & Myers, L. B. (2002). Repressive coping style and adult romantic attachment style: Is there a relationship? *Personality and Individual Differences, 32*(5), 799-807. doi:10.1016/S0191-8869(01)00083-6

Vogel, D., & Wade, N. G. (2009). Stigma and help-seeking. *The Psychologist, 22,* 20-23. Retrieved from http://thepsychologist.bps.org.uk/volume-22/edition-1/stigma-and-help-seeking

Vögele, C. (2015). Behavioral medicine. In J. D. Wright (Ed.), *International Encyclopedia of the Social & Behavioral Sciences* (2nd ed., pp. 463-469). USA: Elsevier. doi:10.1016/B978-0-08-097086-8.14060-7

Vogt, W. P. (2005). *Dictionary of statistics & methodology: A nontechnical guide for the social sciences* (3rd ed.). Thousand Oaks, CA: Sage.

Von Korff, M. V., Gruman, J., Schaefer, J., Curry, S. J., & Wagner, E. H. (1997). Collaborative management of chronic illness. *Annals of Internal Medicine, 127*(12), 1097-1102. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/9412313

Waite, R., & Killian, P. (2008). Health beliefs about depression among African American women. *Perspectives in Psychiatric Care, 44*(3), 185-195. doi:10.1111/j.1744-6163.2008.00173.x

Watson, B., & Gallois, C. (1998). Nurturing communication by health professionals toward patients: A communication accommodation theory approach. *Health Communication, 10*(4), 343-355. doi:10.1207/s15327027hc1004_3

Watson, B., & Gallois, C. (1999). Communication accommodation between patients and health professionals: Themes and strategies in satisfying and unsatisfying encounters. *International Journal of Applied Linguistics, 9*(2), 167-180. doi:10.1111/j.1473-4192.1999.tb00170.x

Watson, B. M., & Gallois, C. (2002). Patients' interactions with health providers: A linguistic category model approach. *Journal of Language and Social Psychology, 21*(1), 32-52. doi:10.1177/0261927X02021001003

Watson, B. M., & Gallois, C. (2004). Emotional expression as a sociolinguistic strategy: Its importance in medical interactions. In S. H. Ng, C. N. Canddlin, & C. Y. Chiu (Eds.), *Language matters: Communication, identity, and culture* (pp. 63-84). Hong Kong: City University of Hong Kong Press.

Watson, B., & Gallois, C. (2007). Language, discourse, and communication about health and illness: Intergroup relations, role, and emotional support. In A. Weatherall, B. M. Watson, & C. Gallois (Eds.), *Language, discourse and social psychology* (pp. 108-130). New York: Palgrave Macmillan.

Watson, B. M., Hewett, D. G., & Gallois, C. (2012). Intergroup communication and healthcare. In H. Giles (Ed.), *The handbook of intergroup communication* (pp. 293-305). New York: Routledge.

Watson, B., Jones, L., & Cretchley, J. (2014). Time as a key topic in health professionals' perceptions of clinical handovers. *Global Qualitative Nursing Research, 1*, 1-11. doi:10.1177/2333393614550162

Watson, B. M., Jones, L., & Hewett, D. G. (2016). Accommodating health. In H. Giles (Ed.), *Communication accommodation theory: Negotiating personal relationships and social identities across contexts* (pp. 152-168). Cambridge, UK: Cambridge University Press. doi:10.1017/CBO9781316226537

Watson, M., Smith, A., & Watter, S. (2005). Leximancer concept mapping of patient case studies. In R. Khosla, R. J. Howlett, & L. C. Jain (Eds.), *Knowledge-Based Intelligent Information And Engineering Systems* (Part III, vol. 3683, pp. 1232-1238). Germany: Springer.

Webb, E. J., Campbell, D. T., Schwartz, R., & Sechrest, L. (2000). *Unobtrusive measures* (Rev. ed.). Thousand Oaks, CA: Sage.

Weber, M. (1947). *The theory of social and economic organization*. New York: Oxford University Press.

Wei, M., Heppner, P. P., & Mallinckrodt, B. (2003). Perceived coping as a mediator between attachment and psychological distress: A structural equation modeling approach. *Journal of Counseling Psychology*, *50*(4), 438-447. doi:10.1037/0022-0167.50.4.438

Weiner, B. (1972). *Theories of motivation: From mechanism to cognition.* Chicago: Markham.

Weiner, B. (1974). *Achievement motivation and attribution theory.* Morristown, NJ: General Learning Press.

Weissman, M. M., Bland, R. C., Canino, G. J., Faravelli, C., Greenwald, S., Hwu, H.-G., … Yeh, E.-K. (1996). Cross-national epidemiology of major depression and bipolar disorder. *Journal of the American Medical Association*, *276*(4), 293-299. doi:10.1001/jama.1996.03540040037030

Whitley, R. (2012). The antipsychiatry movement: Dead, diminishing, or developing? *Psychiatric Services*, *63*(10), 1039-1041. doi:10.1176/appi.ps.201100484

Whooley, M., Avins, A., Miranda, A., & Browner, L. (1997). Case-finding instruments for depression. *Journal of General Internal Medicine, 12*(7), 439-445. doi:10.1046/j.1525-1497.1997.00076.x

Wijeratne, C., & Sachdev, P. (2008). Treatment-resistant depression: Critique of current approaches. *Australian & New Zealand Journal of Psychiatry*, *42*(9), 751-762. doi:10.1080/00048670802277206

Williams, A. (1999). Communication accommodation theory and miscommunication: Issues of awareness and communication dilemmas. *International Journal of Applied Linguistics, 9*(2), 151-165. doi:10.1111/j.1473-4192.1999.tb00169.x

Williams, B., Cattell, D., Greenwood, M., LeFevre, S., Murray, I., & Thomas, P. (1999). Exploring 'person-centredness': User perspectives on a model of social psychiatry. *Health & Social Care in the Community, 7*(6), 475-482. doi:10.1046/j.1365-2524.1999.00215.x

Williams, B., & Healy, D. (2001). Perceptions of illness causation among new referrals to a community mental health team: "Explanatory model" or "exploratory map"? *Social Science & Medicine, 53*(4), 465-476. doi:10.1016/s0277-9536(00)00349-x

Wilson, G. T. (2008). *Behavior therapy.* In R. J. Corsini & D. Wedding (Eds.), *Current psychotherapies* (8th ed., pp 63-106). Belmont, CA: Thomson Brooks/Cole.

Wittchen, H. U., Hofler, M., & Meister, W. (2001). Prevalence and recognition of depressive syndromes in German primary care settings: poorly recognized and treated? *International Clinical Psychopharmacology*, *16*(3), 121-135. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/11354234

Wittink, M. N., Barg, F. K., & Gallo, J. J. (2006). Unwritten rules of talking to doctors about depression: Integrating qualitative and quantitative methods. *Annals of Family Medicine, 4*(4), 302-309. doi:10.1370/afm.558

Woolley, D., & Shaw, E. (1954). A biochemical and pharmacological suggestion about certain mental disorders. *Proceedings of the National Academy of Sciences of the United States of America, 40*(4), 228-231. Retrieved from http://www.jstor.org/stable/89043

World Health Organization. (2008). *The Global Burden of Disease 2004 update*. Retrieved from WHO website http://www.who.int/healthinfo/global_burden_disease/GBD_report_2004update_full.pdf

Wright, A. (1999). Through a glass darkly: Understanding depression. *British Journal of General Practice, 49*(439), 91-92. Retrieved from http://bjgp.org/content/bjgp/49/439/91.full.pdf

Wright, J. H., & Davis, D. (1994). The therapeutic relationship in cognitive-behavioral therapy: Patient perceptions and therapist responses. *Cognitive and Behavioral Practice, 1*(1), 25-45. doi:10.1016/S1077-7229(05)80085-9

Wulsin, L., Vaillant, G., & Wells, V. (1999). A systematic review of the mortality of depression. *Psychosomatic Medicine, 61*(1), 6-17. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/10024062

Zimmerman, M., Chelminski, I., McGlinchey, J. B., & Posternal, M. A. (2008). A clinically useful depression outcome scale. *Comprehensive Psychiatry, 49*(2), 131-140. doi:10.1016/j.comppsych.2007.10.006

Zimmerman, M., & Martinez, J. H. (2012). Web-based assessment of depression in patients treated in clinical practice: reliability, validity, and patient acceptance. *The Journal of Clinical Psychiatry*, *73*(3), 333-338. doi:10.4088/JCP.10m06519

Zivin, K., Ilgen, M., Pfeiffer, P., Welsh, D., Mccarthy, J., Valenstein, M., . . . Kales, H. (2012). Early mortality and years of potential life lost among veterans affairs patients with depression. *Psychiatric Services, 63*(8), 823-826. doi:10.1176/appi.ps.201100317

Appendices

Appendix A
Background – Depression Criteria

Throughout the 2007 Survey Of Mental Health And Wellbeing (SMHWB), the criteria used to define a depressive episode were based on the 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10) (Australian Bureau of Statistics, 2007, p. 65). A Depressive Episode lasts for at least two weeks and is characterised by the presence of a number of the following symptoms:

- depressed mood
- loss of interest in activities
- lack of energy or increased fatigue
- loss of confidence or self esteem
- feelings of self-reproach or excessive guilt
- thoughts of death or suicide, or suicide attempts
- diminished ability to concentrate, think or make decisions
- change in psychomotor activity; agitation or retardation
- sleep disturbance
- change in appetite

The 3 different types of Depressive Episode, based on the number of symptoms the person experienced were:

- Severe Depressive Episode: All of the first three symptoms from the above list and additional symptoms from the remainder of the list to give a total of at least eight
- Moderate Depressive Episode: At least two of the first three symptoms from the above list and additional symptoms from the remainder of the list to give a total of at least six
- Mild Depressive Episode: At least two of the first three symptoms from the above list and additional symptoms from the remainder of the list to give a total of at least four.

Hierarchy rules have been applied to all of the Depressive Episodes. To meet criteria for the ‘with hierarchy’ versions, the person cannot have met criteria for either Hypomanic or Manic episodes in their lifetime. The three types of Depressive Episode collected by the 2007 SMHWB are also mutually exclusive. A person cannot be diagnosed with Moderate Depressive Episode if the criteria for a Severe Depressive Episode have already been met and a diagnosis of a Mild

Depressive Episode is considered only when the other two types of depression have been excluded. This criteria is applied regardless of whether the ‘with hierarchy’ or ‘without hierarchy’ versions of the disorder is used.

Appendix B

Patient Characteristics: Comorbid Psychiatric Conditions

Alphabetical list of conditions from Patient Survey 1 participants' responses to the question: Do you have a previous history of mental distress (e.g., you feel you have experienced psychological symptoms in the past)?

- Agoraphobia
- Anxiety
- Asperger Syndrome
- Borderline Personality Disorder
- Dissociative Identity Disorder
- Eating disorder
- Generalised Anxiety Disorder
- Obsessive Compulsive Disorder
- Panic attacks
- Panic Disorder
- Post Traumatic Stress Disorder
- Social Phobia
- Tourette Syndrome

Appendix C1

Patient Survey: Patient's Explanatory Model for Their Own Depression

These statements are designed to gauge how you understand your depression and how to treat it. Feel free to comment or provide additional information should you wish to do so.

	Strongly disagree	Moderately disagree	Slightly disagree	Slightly agree	Moderately agree	Strongly agree
1. I have depression because something in my body is not functioning efficiently (e.g., brain chemistry imbalance).	○	○	○	○	○	○
2. I have depression because of circumstances (e.g., death of a loved one, relationship breakup, loss of a limb, abuse, etc.).	○	○	○	○	○	○
3. I believe that choices I made led to my depression.	○	○	○	○	○	○
4. If I take the right actions, I have the power to decrease my depression symptoms.	○	○	○	○	○	○
5. Complementary medicines (e.g., vitamins, fish oil, St John's Wort) will help decrease my depression symptoms.	○	○	○	○	○	○
6. My depression symptoms can lessen if I start activities/practices such as exercise, learning, prayer, meditation etc (activities/practices referred to must not involve taking any drugs or complementary medicines).	○	○	○	○	○	○

7. My depression symptoms can lessen by using other remedies in addition to what my doctor recommends.						
8. Alternative providers (for example, counselors, psychologists, etc.) can provide remedies that will reduce my depression symptoms.	○	○	○	○	○	○
9. Depression has changed my priorities.	○	○	○	○	○	○
10. Depression currently plays a major role in my life.	○	○	○	○	○	○
11. I believe that depression will change the future course of my life.	○	○	○	○	○	○
12. There could be some positive outcomes arising from my experience of depression (e.g., increased empathy, greater clarity about one's priorities).	○	○	○	○	○	○
13. I believe it is necessary to take antidepressants to prevent my depression symptoms from becoming worse.	○	○	○	○	○	○
14. It seems to me that the negative side effects from antidepressants can do me more harm than good.	○	○	○	○	○	○
15. I feel some doctors can often be too quick to prescribe antidepressants.	○	○	○	○	○	○
16. It is important that my doctor and I work together to improve my recovery from depression.	○	○	○	○	○	○

Appendix C2

Doctor's survey: Doctor's Explanatory Model for Their Patient's Depression

These questions are designed to capture your views regarding your patient's depression and how to treat it. Feel free to comment or provide additional information you wish to do so.

	Strongly disagree	Moderately disagree	Slightly disagree	Slightly agree	Moderately agree	Strongly agree
1. This patient has depression because something in his/her body is not functioning efficiently (e.g., brain chemistry imbalance).	○	○	○	○	○	○
2. This patient has depression because of circumstances (e.g., death of a loved one, relationship breakup, loss of a limb, abuse, etc.)	○	○	○	○	○	○
3. Choices that this patient made led to his/her depression.	○	○	○	○	○	○
4. If this patient takes the right actions, he/she has the power to decrease his/her depression symptoms.	○	○	○	○	○	○
5. Complementary medicines (e.g., vitamins, fish oil, St John's Wort) will help decrease this patient's depression symptoms.	○	○	○	○	○	○
6. This patient's depression symptoms can lessen if he/she starts activities/practices such as exercise, learning, prayer, meditation etc (activities/practices referred to must not involve taking any drugs/complementary medicines).	○	○	○	○	○	○
7. This patient's depression symptoms can lessen by using other remedies in addition to what I recommend.	○	○	○	○	○	○

8. Alternative providers (for example, counselors, psychologists, etc.) can provide remedies that will reduce this patient's depression symptoms.	○	○	○	○	○	○
9. Depression has changed this patient's priorities.	○	○	○	○	○	○
10. Depression currently plays a major role in this patient's life.	○	○	○	○	○	○
11. Depression will change the future course of this patient's life.	○	○	○	○	○	○
12. There could be some positive outcomes arising from this patient's experience of depression (e.g., increased empathy, greater clarity about one's priorities).	○	○	○	○	○	○
13. It is necessary for this patient to take antidepressants to prevent his/her depression symptoms from becoming worse.	○	○	○	○	○	○
14. The negative side effects from antidepressants can do more harm than good to this patient.	○	○	○	○	○	○
15. Some doctors can often be too quick to prescribe antidepressants.	○	○	○	○	○	○
16. It is important that this patient and I work together to improve his/her recovery from depression.	○	○	○	○	○	○

Appendix C3

Doctor survey: Doctor's Perception of Patient's Explanatory Model for Their Depression (GP-Perceived Patient EM)

These questions are designed to elicit how you think your patient views his/her depression and how to treat it. Feel free to comment or provide additional information should you wish to do so.

	Strongly disagree	Moderately disagree	Slightly disagree	Slightly agree	Moderately agree	Strongly agree
1. I think this patient thinks that he/she has depression because something in his/her body is not functioning efficiently (e.g., brain chemistry imbalance).	○	○	○	○	○	○
2. I think this patient thinks that he/she has depression because of circumstances (e.g., death of a loved one, relationship breakup, loss of a limb, abuse, etc.)	○	○	○	○	○	○
3. I think this patient thinks that choices he/she made led to his/her depression.	○	○	○	○	○	○
4. I think this patient thinks that if he/she takes the right actions, he/she has the power to decrease his/her depression symptoms.	○	○	○	○	○	○
5. I think this patient thinks that complementary medicines (e.g., vitamins, fish oil, St John's Wort) will help decrease his/her depression symptoms.	○	○	○	○	○	○

6. I think this patient thinks that his/her depression symptoms can lessen if he/she starts activities/practices such as exercise, learning, prayer/meditation etc (activities/practices referred to must not involve taking any drugs/complementary medicines).	○	○	○	○	○	○
7. I think this patient thinks that his/her depression symptoms can lessen by using other remedies in addition to what I recommend.	○	○	○	○	○	○
8. I think this patient thinks that alternative providers (for example, counselors, psychologists, etc.) can provide remedies that will reduce his/her depression symptoms.	○	○	○	○	○	○
9. I think this patient thinks that depression has changed his/her priorities.	○	○	○	○	○	○
10. I think this patient thinks that depression currently plays a major role in his/her life.	○	○	○	○	○	○
11. I think this patient thinks that depression will change the future course of his/her life.	○	○	○	○	○	○
12. I think this patient thinks that there could be some positive outcomes arising from his/her experience of depression (e.g., increased empathy, greater clarity about one's priorities).	○	○	○	○	○	○
13. I think this patient thinks that it is necessary for him/her to take antidepressants to prevent his/her depression symptoms from becoming worse.	○	○	○	○	○	○
14. I think this patient thinks that the negative side effects from antidepressants can do more harm than good to him/her.	○	○	○	○	○	○
15. I think this patient thinks that some doctors can often be too quick to prescribe antidepressants.	○	○	○	○	○	○
16. I think this patient thinks that it is important that he/she and I work together to improve his/her recovery from depression.	○	○	○	○	○	○

Appendix D

Patient survey: Modified Internalised Stigma of Mental Illness (ISMI) Depression Scale

These statements are designed to gauge how you feel about yourself because of your depression. Feel free to comment or provide additional information should you wish to do so.

	Strongly disagree	Moderately disagree	Slightly disagree	Slightly agree	Moderately agree	Strongly agree
1. I feel isolated and/or out of place in the world because I have depression.	○	○	○	○	○	○
2. It is possible for people without depression to understand me.	○	○	○	○	○	○
3. I am ashamed that I have depression.	○	○	○	○	○	○
4. I do not feel embarrassed about seeking professional help for depression.	○	○	○	○	○	○
5. I would feel embarrassed if others knew I was seeking professional help for depression.	○	○	○	○	○	○
6. I am not disappointed in myself for having depression.	○	○	○	○	○	○
7. I feel inferior to others who don't have depression.	○	○	○	○	○	○
8. It is wrong for me to think that I should be able to "pull myself together".	○	○	○	○	○	○

9. I think I should be stronger (i.e., able to cope with things).	○	○	○	○	○	○
10. Because I have depression, I can be violent towards others.	○	○	○	○	○	○
11. Having depression does not mean I need others to make decisions for me.	○	○	○	○	○	○
12. I believe I can live a rewarding life despite having depression.	○	○	○	○	○	○
13. Having depression means I am not suited for a long-term relationship or marriage.	○	○	○	○	○	○
14. Having depression does not stop me from being able to contribute to society.	○	○	○	○	○	○
15. I would not see myself as weak if I took antidepressants.	○	○	○	○	○	○
16. I feel people discriminate against me (e.g., they think I can't achieve much in life) because I have depression.	○	○	○	○	○	○
17. I feel ignored or taken less seriously by others because I have depression	○	○	○	○	○	○
18. Nobody would be interested in getting close to me because I have depression.	○	○	○	○	○	○
19. Nobody can get close to me because I have depression.	○	○	○	○	○	○

20. I don’t talk about myself much because I don’t want to burden others with my depression.	○	○	○	○	○	○
21. I stay away from social situations in order to protect my family or friends from embarrassment.	○	○	○	○	○	○
22. I avoid getting close to people who don’t have depression because I believe they will reject me.	○	○	○	○	○	○
23. Living with depression has made me a tough survivor.	○	○	○	○	○	○

Appendix E

Patient Survey: Perception of GP-Patient Communication Effectiveness

These statements are designed to gauge how well you feel you communicate with the GP that you see for depression. Feel free to comment or provide additional information should you wish to do so.

	Strongly disagree	Moderately disagree	Slightly disagree	Slightly agree	Moderately agree	Strongly agree
1. This GP and I have developed a good relationship (e.g., satisfying, supportive).	○	○	○	○	○	○
2. I felt I could relate to this GP because we have some things in common.	○	○	○	○	○	○
3. This GP treated me as an individual person.	○	○	○	○	○	○
4. This GP listened attentively to me.	○	○	○	○	○	○
5. I felt able to ask questions of this GP.	○	○	○	○	○	○
6. I felt this GP was empathetic (i.e., able to put himself/herself in my shoes).	○	○	○	○	○	○
7. This GP gave me as much time as I needed to explain my feelings and situation.	○	○	○	○	○	○
8. This GP treated me as an equal.	○	○	○	○	○	○
9. I discussed the issues that I wanted to with this GP.	○	○	○	○	○	○
10. This GP asked me clear questions.	○	○	○	○	○	○
11. This GP was reassuring.	○	○	○	○	○	○

12. I was able to actively participate in discussion with this GP.	○	○	○	○	○	○
13. This GP provided the right amount of info.	○	○	○	○	○	○
14. I felt I was able to express my needs clearly.	○	○	○	○	○	○
15. I felt this GP understood my needs.	○	○	○	○	○	○
16. This GP checked my understanding of the topics discussed	○	○	○	○	○	○

Appendix F

Patient Survey: Modified Clinically Useful Depression Outcome Scale

These are statements about symptoms of depression. For each item please indicate how well it describes you during the past week, including today. Feel free to comment or provide additional information should you wish to do so. During the past week, including today…

Symptoms	Not at all true (0 days)	Sometimes (1-2 days)	Half the time (3-4 days)	Most of the time (5-6 days)	Every day
1. I have felt sad or depressed.	○	○	○	○	○
2. I have not been as interested in my usual activities	○	○	○	○	○
3. My appetite has been poor	○	○	○	○	○
4. My appetite has been greater than usual	○	○	○	○	○
5. I have had difficulty sleeping	○	○	○	○	○
6. I have been sleeping too much	○	○	○	○	○
7. I have felt very fidgety, making it difficult to sit still	○	○	○	○	○
8. I have felt physically slowed down, like my body was stuck in mud	○	○	○	○	○
9. My energy level has been low	○	○	○	○	○
10. I have felt guilty	○	○	○	○	○
11. I have thought I was a failure	○	○	○	○	○
12. I have had problems concentrating	○	○	○	○	○
13. I have had more difficulties making decisions than usual	○	○	○	○	○
14. I have thought that the future looked hopeless	○	○	○	○	○
15. I have wished I was dead.	○	○	○	○	○
16. I have thought about killing myself.	○	○	○	○	○

17. Overall how much have symptoms of depression interfered with or caused difficulties in your life during the past week?

○ Not at all
○ A little bit
○ Moderately
○ Quite a bit
○ Extremely

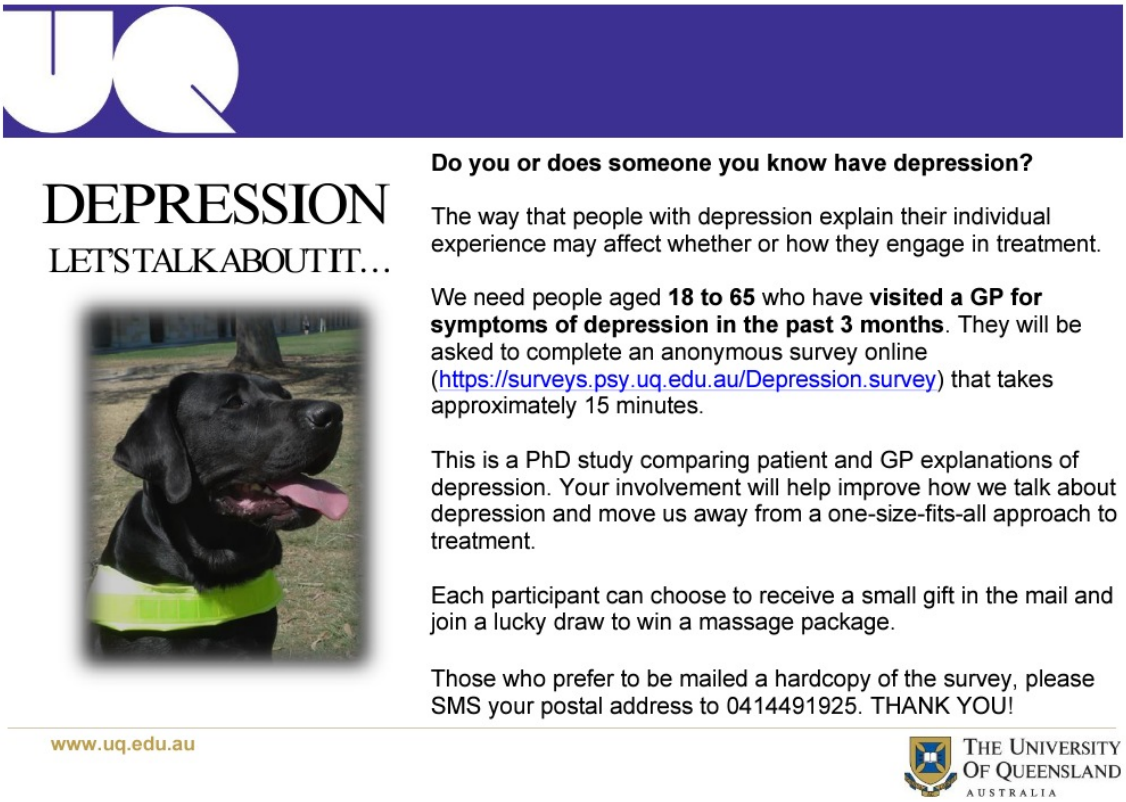
18. How would you rate your overall quality of life during the past week?

○ very good, my life could hardly be better
○ pretty good, most things are going well
○ the good and bad parts are about equal
○ pretty bad, most things are going poorly
○ very bad my life could hardly be worse

Appendix G

Recruitment: Flyers for Patient Surveys

Sample survey promotion flyer: Formatted on A4 (landscape) for clinics, A5 for pharmacies, community centres, and so forth.



UQ

DEPRESSION

LET'S TALK ABOUT IT…

Do you or does someone you know have depression?

The way that people with depression explain their individual experience may affect whether or how they engage in treatment.

We need people aged **18 to 65** who have **visited a GP for symptoms of depression in the past 3 months**. They will be asked to complete an anonymous survey online (https://surveys.psy.uq.edu.au/Depression.survey) that takes approximately 15 minutes.

This is a PhD study comparing patient and GP explanations of depression. Your involvement will help improve how we talk about depression and move us away from a one-size-fits-all approach to treatment.

Each participant can choose to receive a small gift in the mail and join a lucky draw to win a massage package.

Those who prefer to be mailed a hardcopy of the survey, please SMS your postal address to 0414491925. THANK YOU!

www.uq.edu.au

THE UNIVERSITY OF QUEENSLAND
AUSTRALIA

Appendix H1

Participant Information Sheet: Patient Survey

Depression: How do people with depression and their doctors explain it, and does it matter?

Purpose of study

This study examines patient and general practitioner (GP) explanations for depression. We are interested in explanations for depression because communication between patients and doctors, patient's experiences of stigma and their treatment outcome could be affected by differing explanations. This study is being conducted by Miss Jen Lee Teh as part of the requirements for the Doctor of Philosophy (PhD) degree at the University of Queensland under the supervision of Dr Bernadette Watson (Senior Lecturer, UQ School of Psychology) and Dr David King (Senior Lecturer, UQ School of Medicine).

Participation and withdrawal

We welcome your participation if you are between 18 and 65 years old and have visited a GP for what you consider to be depressive symptoms (with or without diagnosis of depression) in the past 3 months. We will approach your GP to request his/her participation if you provide the necessary information. GPs who join this study will be asked for their understandings of depression models and not clients' personal information as that is confidential. Your answers to this survey will also be kept confidential but you can choose to share them with your GP upon your GP's request if he/she is in the project.

Participation in this study is completely voluntary and you are free to withdraw from this study at any time without any negative consequences. If you wish to withdraw, simply stop completing the survey.

What is involved

Participants will indicate the extent of their agreement with statements on depression, communication and stigma. They will also rate the severity of their depressive symptoms and prioritise which areas they want to see improvement in. The survey questions are not exhaustive so you are welcome to give additional comments if you wish.

After 6 months, there will be a follow-up survey. The first survey will take less than 20 minutes and the follow-up will take 15 minutes.

Get a free gift and stand to win a 90-minute spa package!

All participants who finish the first survey will receive a small gift in the mail for their efforts if they provide a postal address. Participants who complete the follow-up surveys can join a lucky draw to win a 90-minute spa package. There are four packages up for grabs from Ripple Massage & Spa, a national mobile massage, beauty and day spa service that operates in 17 locations including Brisbane, Ipswich, Gold Coast, Sunshine Coast, Melbourne, Yarra Valley, Mornington Peninsula, Dandenongs, Great Ocean Road and Hobart.

Risks

There are minimal risks from participating in this study. It is possible that in reflecting on and answering questions about your depression, you may experience negative emotions and feelings. If you should find any question to be invasive or offensive, you are free to skip the question.

If you feel yourself becoming distressed, we recommend that you stop answering the survey. You may return to the survey at a later time, or withdraw your participation. The researcher may get in touch with your GP if, in the course of conducting this research, she assesses that there is a risk that you may hurt yourself. If you need support, please contact:

Lifeline 13 11 14
Suicide Callback Service 1300 659 467
beyondblue **1300 22 4636**
SANE Australia 1800 18 SANE (7263)

Ethics Clearance and Contacts

This study has been cleared in accordance with the ethical review processes of the University of Queensland and within the guidelines of the National Statement on Ethical Conduct in Human Research. You are of course, free to discuss your participation in this study with the researcher (contactable by email j.teh@uq.edu.au or mobile 0414491925). If you would like to speak to an officer of the University not involved in the study, you may contact the University of Queensland Ethics Officer (07 3365 3924, e-mail: humanethics@research.uq.edu.au).

If you would like to learn the outcome of this project, you can contact the researcher at the email above after July 2016, and a summary of the study's findings will be sent to you.

Thank you for participating.

Appendix H2

Participant Information Sheet: Patient Interview

How do people with depression and their doctors explain it, and does it matter?

The purpose of the study

This study examines patient and general practitioner (GP) explanations for depression. We are interested in explanations for depression because communication between patients and doctors, patient's experiences of stigma and their treatment outcome could be affected by differing explanations.

This study is being conducted by Jen Lee Teh (Miss) as part of the requirements for the Doctor of Philosophy (PhD) degree at the University of Queensland under the supervision of Dr Bernadette Watson (Senior Lecturer, UQ School of Psychology).

Participation and withdrawal

If you have gone to a GP to seek help for what you consider to be depressive symptoms and you are between 18 and 65 years old, we welcome your participation. Participation in this study is completely voluntary and you are free to withdraw from this study at any time without prejudice or penalty. If you wish to withdraw, simply stop the interview. If you do withdraw, the materials that you have completed to that point will be deleted and will not be included in the study.

What is involved

Participants are asked to meet with researcher for in-person interview at a location that suits them with the option to be audiotaped OR they can provide e-mail address for questions to be e-mailed. It will take about 45 minutes to 1 hour to answer the interview questions in person. Alternatively, participants can choose to be interviewed over e-mail.

Risks

There are minimal risks associated with involvement in this study. It is possible that in reflecting on and answering questions about your depression you may experience negative emotions and feelings. If you should find any question or procedure to be invasive or offensive, you are free to omit answering or participating in that aspect of the study. If you feel yourself becoming distressed, we recommend that you stop the interview. You may reschedule, or withdraw your participation altogether.

The researcher may get in touch with your GP if, in the course of conducting this research, she assesses that there is a risk that you may hurt yourself.

If you need to seek support, sources include:

Lifeline	13 11 14

Suicide Callback Service 1300 659 467
beyondblue Information and Referral line: 1300 22 4636
SANE Australia: 1800 18 SANE (7263)

Confidentiality and security of data

All data collected in this study will be stored confidentially. Only members of the research team will have access to identified data. All data will be coded in a de-identified manner and subsequently analysed and reported in such a way that responses will not be able to be linked to any individual. The data you provide will only be used for this study's specific research purposes.

Ethics Clearance and Contacts

This study has been cleared in accordance with the ethical review processes of the University of Queensland and within the guidelines of the National Statement on Ethical Conduct in Human Research. You are, of course, free to discuss your participation in this study with the researcher (contactable on j.teh@uq.edu.au or mobile +61414491925). If you would like to speak to an officer of the University not involved in the study, you may contact the University of Queensland Ethics Officer on 3365 3924, e-mail: humanethics@research.uq.edu.au.

If you would like to learn the outcome of the study in which you are participating, you can contact me at the email above after July 2016, and I will send you an Abstract of the study and findings.

Thank you for your participation in this study.

Appendix I

Recruitment: Invitation for Doctor Surveys

Sample letter to GPs of study participants – formatted for email or standard mail

Dear Dr ____,

My name is Jen and I am conducting PhD research that compares depression explanations between GPs and patients, with the aim of improving communication and treatment outcomes. One of your patients, ____, recently completed our survey and consented to us asking you to join the study (please see attached .jpeg for screenshot of ____'s consent on the online survey). It would greatly help to maximise ____'s data if you could take the doctor's survey.

You can read more about the doctor's survey here https://surveys.psy.uq.edu.au/GPdepression.survey (the link brings you to the participant information sheet, which flows on to the questions). Other GPs who have completed it have done so in less than 10 minutes.

As mentioned earlier, my study is about depression explanations – how would a GP explain the condition to that particular patient (or others like him/her)? My co-advisor, Dr David King, who is a GP like yourself, said it was important to let GPs know that there is no "right" answer, as we are not testing GPs on how much they know of their patients. We are only interested to see if the lens through which patient and GP use are the same/different, and how communication comes into the picture if they are the same/different.

I very much look forward to your participation. Thank you and please let me know if you have any questions.

Yours sincerely,

Jen

Appendix J
Participant Information Sheet: Doctor Survey

How do doctors and patients use depression explanatory models (EMs)? The role of EMs in communication effectiveness, patient stigma levels and treatment satisfaction

Principal researcher

Jen Lee Teh (Ms) – PhD candidate, University of Queensland (UQ) School of Psychology (Advisors: Dr Bernadette Watson – Senior Lecturer, UQ School of Psychology; Dr David King – Senior Lecturer, UQ School of Medicine)

Purpose of study

This survey is part of a PhD research project investigating the importance of aligning depression conceptualisations or explanatory models (EMs) between patients and their general practitioners (GPs). One key project aim is to help GPs better understand the expectations of their depressed patients in order to improve GP-patient communication effectiveness, levels of patient self-stigma (self-stigma is the stigma that depression patients internalise and feel about themselves) and treatment outcomes.

Criteria for participation

One of your patients who consulted you in the past 3 months for his/her depressive symptoms is part of this project and your participation is requested so as to compare your explanatory model as a GP with your patient's model. Your answers can be kept confidential or you can choose to make your answers available to your patient upon his/her request.

Expected duration of participation

10 minutes to answer survey questions (can be done online OR over the phone OR the researcher can bring a hardcopy of the survey to meet you at a time and location of your convenience). If you have a hardcopy of the survey with you already, please complete it and mail it back to **Jen Lee Teh, School of Psychology, UQ St Lucia, QLD 4072.**

Description of involvement and any foreseeable risks

In the first segment, you will read statements about depression explanatory models and indicate the extent of your agreement. The second segment will ask you to rate communication effectiveness with your patient. There are no risks associated with involvement in this study. This research focuses on how GPs and patients explain depression. Confidential information about the patient is not required.

IMPORTANT: Your participation in this project is voluntary and you may withdraw at any time without prejudice or penalty.

If you have questions, feedback or would like to discuss your participation in this study, please contact Miss Jen Lee Teh (j.teh@uq.edu.au or mobile +61414491925). This study has been cleared by a human ethics committee at the University of Queensland in accordance with the National Health and Medical Research Council's guidelines. If you would like to speak to an officer of the University not involved in the study, you may contact the Ethics Officer on 3365 3924. If you would like to learn the outcome of this study, you can contact the researcher at the email above after July 2016, and a summary of the study's findings will be sent to you.

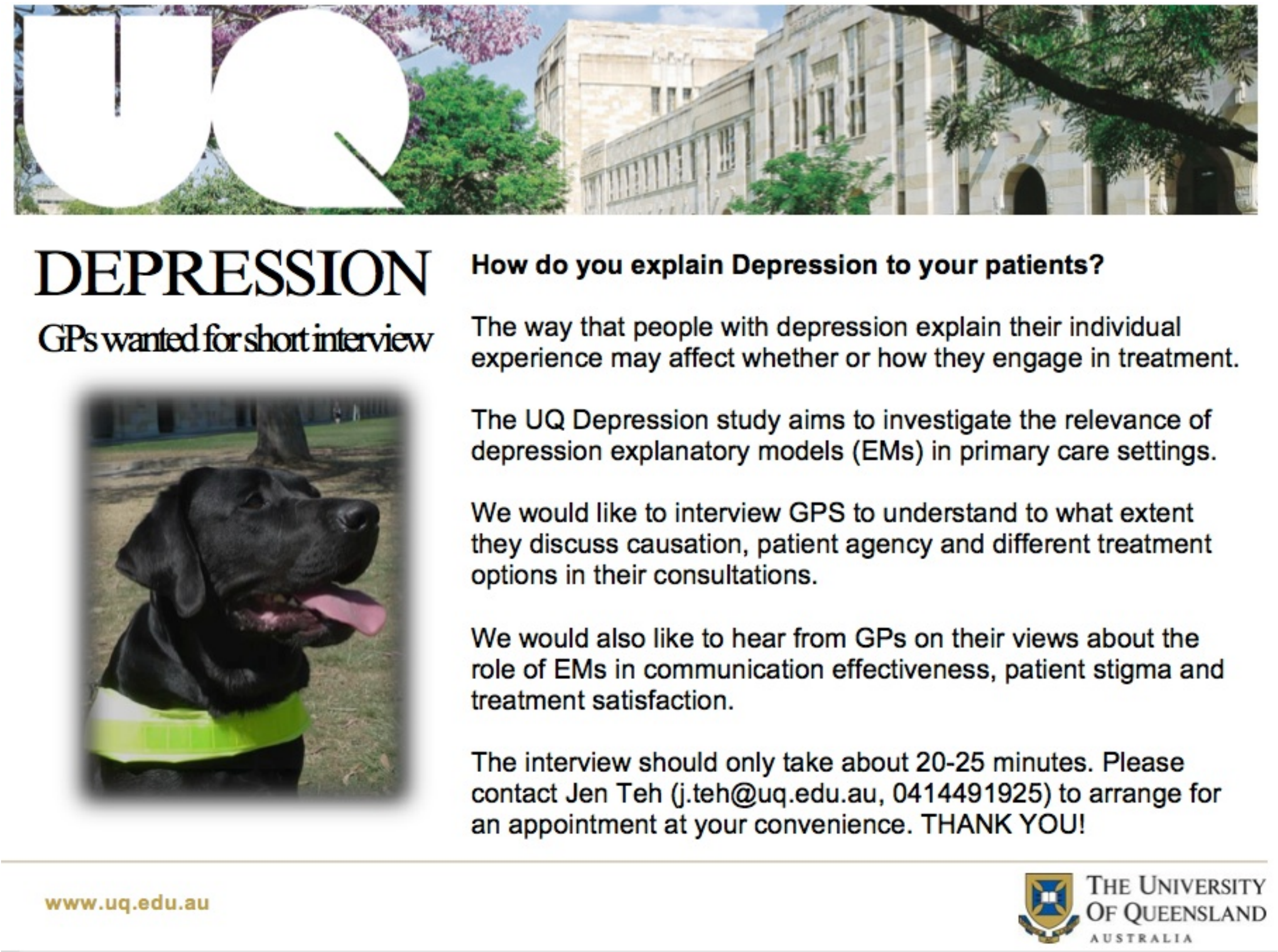
Thank you for your participation. Please provide your initials, your patient's initials and date of birth so that we can match the data collected. You will be asked at the end of the survey whether you want to share your answers with your patient.

Your initials ____ Your patient's initials ____

Appendix K

Recruitment: Flyer for Doctor Interviews

Sample flyer – disseminated via e-mail



UQ

DEPRESSION

GPs wanted for short interview

How do you explain Depression to your patients?

The way that people with depression explain their individual experience may affect whether or how they engage in treatment.

The UQ Depression study aims to investigate the relevance of depression explanatory models (EMs) in primary care settings.

We would like to interview GPS to understand to what extent they discuss causation, patient agency and different treatment options in their consultations.

We would also like to hear from GPs on their views about the role of EMs in communication effectiveness, patient stigma and treatment satisfaction.

The interview should only take about 20-25 minutes. Please contact Jen Teh (j.teh@uq.edu.au, 0414491925) to arrange for an appointment at your convenience. THANK YOU!

www.uq.edu.au

THE UNIVERSITY
OF QUEENSLAND
AUSTRALIA

Appendix L

Participant Information Sheet: Doctor Interview

Depression: How do people with depression and their doctors explain it, and does it matter?

The purpose of the study

This study examines patient and general practitioner (GP) explanations for depression. One key project aim is to help GPs better understand the expectations of their depressed patients in order to improve GP-patient communication effectiveness, levels of patients self-stigma (self-stigma is the stigma that depression patients internalise and feel about themselves) and treatment outcomes.

This study is being conducted by Jen Lee Teh (Miss) as part of the requirements for the Doctor of Philosophy (PhD) degree at the University of Queensland under the supervision of Dr Bernadette Watson (Senior Lecturer, UQ School of Psychology).

Participation and withdrawal

Participation in this study is completely voluntary and you are free to withdraw from this study at any time without prejudice or penalty. If you wish to withdraw, simply stop the interview. If you do withdraw, the materials that you have completed to that point will be deleted and will not be included in the study.

What is involved

Participants are asked to meet with researcher for in-person interview at a location that suits them with the option to be audiotaped. It will take about 30 minutes to answer the interview questions.

Risks

There are no risks associated with involvement in this study. This research focuses on how GPs and patients explain depression. Confidential information about patients is not required.

Confidentiality and security of data

All data collected in this study will be stored confidentially. Only members of the research team will have access to identified data. All data will be coded in a de-identified manner and subsequently analysed and reported in such a way that responses will not be able to be linked to any individual. The data you provide will only be used for this study’s specific research purposes.

Ethics Clearance and Contacts

This study has been cleared in accordance with the ethical review processes of the University of Queensland and within the guidelines of the National Statement on Ethical Conduct in Human Research. You are, of course, free to discuss your participation in this study with the researcher (contactable on j.teh@uq.edu.au or mobile +61414491925). If you would like to speak to an officer of the University not involved in the study, you may contact the University of Queensland Ethics Officer on 3365 3924, e-mail: humanethics@research.uq.edu.au.

If you would like to learn the outcome of the study in which you are participating, you can contact me at the email above after July 2016, and I will send you an Abstract of the study and findings.

Thank you for your participation in this study.

Appendix M
Interview Consent Form

Name of Project:
Depression: How do people with depression and their doctors explain it, and does it matter?

Principal researcher
Jen Lee Teh (Ms) – Research Higher Degree student, University of Queensland (UQ) School of Psychology

Advisors
Dr Bernadette Watson – Senior Lecturer, UQ School of Psychology
Dr David King – Senior Lecturer, UQ School of Medicine

Date:

I hereby agree to be involved in the above research project as a participant. I have read the research information sheet pertaining to this research project and understand the nature of the research and my role in it.

I understand that my participation in this study is completely voluntary and that I am free to withdraw from this study at any time without prejudice or penalty.

I am aware of the risks (if any) that may arise from this project. I understand that the researcher will safeguard the confidentiality and security of data from this project.

Signature of research subject:

Appendix N

Supplementary Results: Scatter Plots of Patient Endorsement of Biomedical Causation Of Depression and Self-Stigma Subscales

X-axis: Agreement to the statement “I have depression because something in my body is not functioning efficiently (e.g., brain chemistry imbalance).” 1 = *strongly disagree*; 6 = *strongly agree*
Y-axis: Participant’s average score for items in the self-stigma subscale

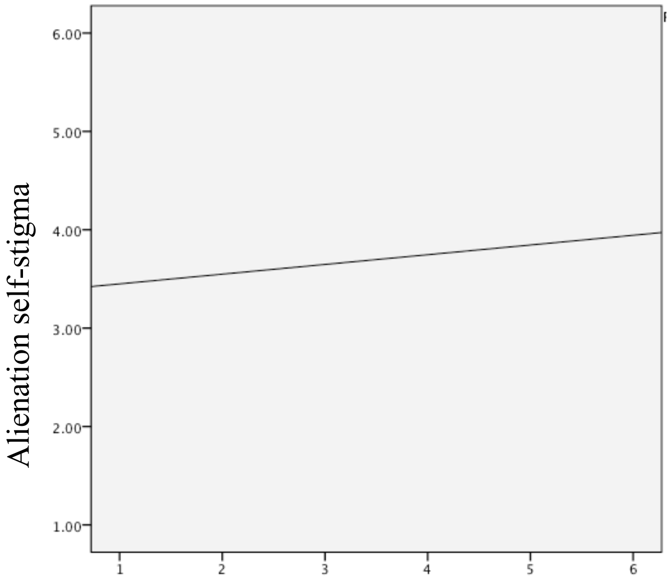


Figure N1. Alienation subscale scores and responses to biomedical causation item

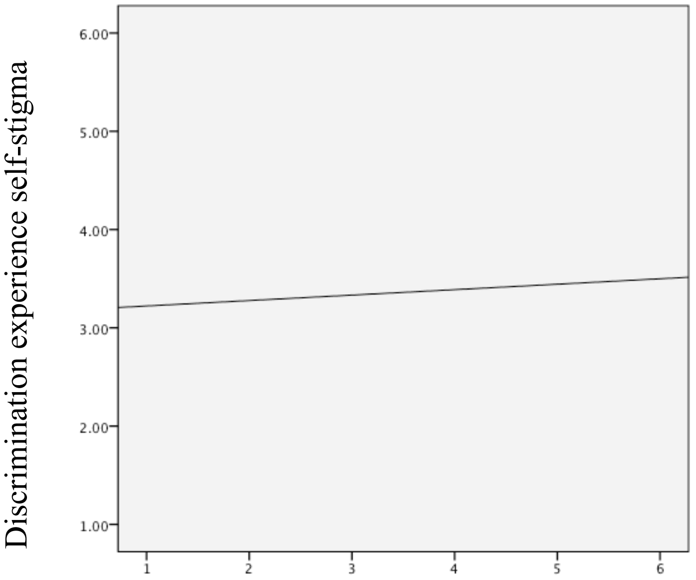


Figure N2. Discrimination experience subscale scores and responses to biomedical causation item

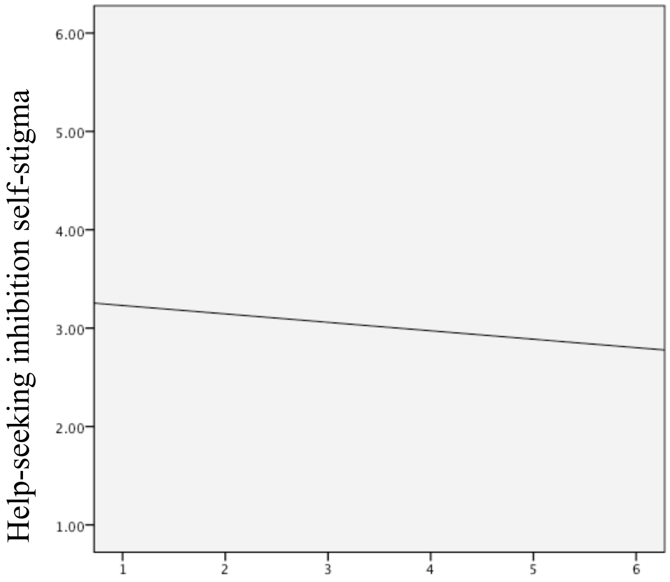


Figure N3. Help-seeking inhibition subscale scores and responses to biomedical causation item

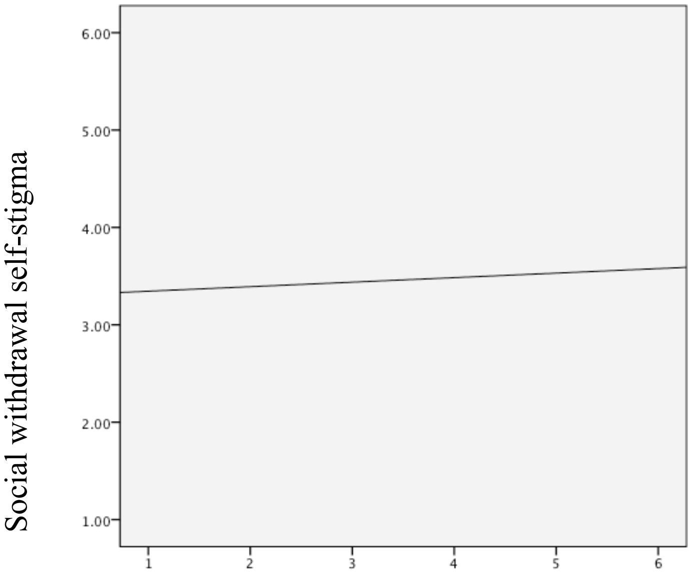


Figure N4. Social withdrawal subscale scores and responses to biomedical causation item

Appendix O

Patient Survey: Patient’s Perception of Whether Their Doctor Shares Their View

1. Do you think your GP views your depression in the same way as you do, in terms of the origin/cause of your depression (e.g., your depression is caused by brain chemistry imbalance)?

Please elaborate on your answer if possible.

○ Yes because...(If you can, please explain why) ___________________________________

○ No because...(If you can, please explain why) _____________________________________

○ I am not sure/I don’t know because...(If you can, please explain why) ____________________

2. Do you think your GP views your depression in the same way as you do, in terms of how much your actions contributed to your condition?

Please elaborate on your answer if possible.

○ Yes because...(If you can, please explain why) ___________________________________

○ No because...(If you can, please explain why) _____________________________________

○ I am not sure/I don’t know because...(If you can, please explain why) ____________________

3. Do you think your GP views your depression in the same way as you do, in terms of how big a part you play in reducing your symptoms?

Please elaborate on your answer if possible.

○ Yes because...(If you can, please explain why) ___________________________________

○ No because...(If you can, please explain why) _____________________________________

○ I am not sure/I don’t know because...(If you can, please explain why) ____________________

4. Do you think your GP views your depression in the same way as you do, in terms of effectiveness of non-medical therapies (e.g., fish oil supplements or activities such as exercise)?

Please elaborate on your answer if possible.

○ Yes because...(If you can, please explain why) ___________________________________

○ No because...(If you can, please explain why) _____________________________________

○ I am not sure/I don’t know because...(If you can, please explain why) ____________________

5. Do you think your GP views your depression in the same way as you do, in terms of the impact depression has on your life?

Please elaborate on your answer if possible.

○ Yes because...(If you can, please explain why) ____________________________________

○ No because...(If you can, please explain why) _____________________________________

○ I am not sure/I don’t know because...(If you can, please explain why) ____________________

Appendix P

Supplementary Results: Correlation Coefficients (r_s) Between Preference for GP-Patient Cooperation and Communication Accommodation Theory (CAT) Survey Subscales

Characteristics	Emotional expression	Discourse management	Interpretability	Inter-personal control
Gender				
Female (*n* = 168 or 169)	.41**	.45**	.41**	.37**
Male (*n* = 56 or 57)	.46**	.55**	.53**	.64**
Age (years)				
18-24 (*n* = 75)	.29*	.28*	.27*	.26*
25-39 (*n* = 94 or 95)	.49**	.57**	.51**	.49**
40-54 (*n* = 41)	.32*	.39*	.41**	.34*
Culture/Ethnicity				
Asian (*n* = 36 or 37)	.50**	.62**	.49**	.37*
European (*n* = 162 or 163)	.37**	.40**	.40**	.38**
Education				
≥ Bachelor (*n* = 137 or 138)	.46**	.51**	.45**	.39**
Cert/Dip/Adv Dip (*n* = 46)	.24 (*ns*)	.35*	.38**	.38**
Up to Year 12 (*n* = 36)	.48**	.45**	.39*	.50**
Diagnosed with depression				
Yes (*n* = 168 or 169)	.39**	.45**	.41**	.42**
No (*n* = 50 or 51)	.37**	.43**	.45**	.40**
Earliest depression onset				
≤ 1 yr ago (*n* = 38 or 39)	.31 (.053)	.41*	.37*	.34*
> 1 and ≤ 5 yrs ago (*n* = 48)	.52**	.47**	.42**	.57**
> 5 and ≤ 10 yrs ago (*n* = 50)	.32*	.42**	.37**	.41**
>10 yrs (*n* = 76 or 77)	.51**	.54**	.56**	.46**
Psychiatric co-morbidity				
Yes (*n* = 187 or 188 or 189)	.43**	.50**	.45**	.46**
No (*n* = 43)	.45**	.39**	.42**	.29 (.060)
Current physical health				
Poor (*n* = 45)	.39*	.45**	..48**	.36*
Fair (*n* = 110 or 111)	.54**	.53**	.51**	.55**
Good (*n* = 62 or 63)	.21 (*ns*)	.35**	.27*	.23 (.075)
Impact on quality of life				
Severely (*n* = 33 or 34)	.35*	.39*	.48**	.31 (.077)
Moderately (*n* = 68 or 69)	.53**	.59**	.52**	.54**
Slightly (*n* = 69)	.35**	.34**	.34**	.35**
Not at all (*n* = 45 or 46)	.35*	.45**	.37*	.40**

Note. p-values between .05 and .10 are in parentheses. *p < .05, **p < .01.

Appendix Q

Question Guide for Semi-structured Interviews with General Practitioners (GPs)

Overall Purpose

A. To understand to what extent explanatory models are discussed in primary care settings
B. To understand the role of explanatory models in GP-patient communication, patient self-stigma and outcomes (i.e., why EMs are important)

Good morning/afternoon Dr ____. Thank you for your time today. Would you mind if I recorded the interview? In case my note taking is not quick enough. So my study investigates the importance of aligning how depression is conceptualised between patients and their GPs. These conceptualizations are called explanatory models (EMs). The theory of explanatory model comes from psychiatrist and anthropologist Arthur Kleinman – and he proposed that individuals and groups can have very different notions of health and disease that may have an impact on treatment. This can be for any disease but my research focuses on explanatory models in depression.

1. Aim – to find out how much GP values patient's perspective (baseline)

Example question: When you diagnose and treat someone with depression, how important is it for you to know whether the patient has an explanatory model or pre-existing notions about their condition?

2a. Aim – to find out how keen a GP is to discuss explanatory models with patients (psychoeducation)

Example question: How much effort do you put into discussing with your patient the cause and prognosis of their depression?

2b. Aim – to find out degree of variation in GP's explanation of a patient's depression (consistency)

Example question: Do you use the same words/concepts to explain a patient's depression or does your explanation vary?

(If need to clarify say "If it varies, in what way and due to what factors?")

3a. Aim – to find out how GP explores patient's EM and why (specific aspects)

Kleinman suggests asking certain questions to learn how patients see their illness:- Here are some sample questions as applied to my study on depression.

[Questions A-E below was printed out on an A5 piece of paper in large font]

Let me give you a minute to read the questions and then I will ask you what you think of them as questions to elucidate your patient's EM.

A. What do you think triggered/made you more susceptible to/caused your depression? (e.g., finding out if is it due to biophysical or psychosocial reasons)

B. How has depression affected your life?

C. How have you coped with this in the past/what have you found useful/What do you think you can do to alleviate it?

D. What treatment options would you like to try/do you think would be beneficial for you?

E. Where would you like to be in 3 months' time/What are the preferred outcomes you hope to achieve?

Example question: Do you use questions like this to collect information from the patient. Can you give details? Why or why not?

3b. Aim – to find out what GPs consider to be key information for effective treatment (any emphasis)

Example question: Out of the five questions above, which, if any, do you tend to emphasise? Why?

3c. Aim – to find out <u>barriers</u> that GPs face when trying to understand patient EM

Example question: What are some challenges, in any, in getting patients to answer about such aspects of their EMs?

4. Aim – to find out if explanatory model differences cause problems

Example question: What problems/difficulties may arise when you and your patient have different ideas about their depression? **Can you think of an example/anecdote that you can share?**

5. Aim – to find out if GP thinks accurate knowledge of patient EM affects communication process

Example question: To what extent, if at all, does having accurate knowledge of your patient’s EM influence how you talk to that patient?

If there is confusion over whether this question is asking for the same thing as Q3b, clarify that Q3b is about content and Q5 is about process of communication. If needed give scenario of telling someone they were not successful in job application. Content may be the same, but how they are told can differ. For example, “You didn’t get it” versus “There was a lot of stiff competition and very limited vacancies.”

6a. Aim – to find out effect of self-stigma on therapeutic relationship

Example question:

Let me now ask about self-stigma that is, negative beliefs patients internalise about themselves for having depression. What would you say is the effect of patient self-stigma, if any, on the relationship you have with your patient?

6b. Aim – to find out GP’s view on whether accurate knowledge of patient EM can help reduce patient self-stigma

Example question: To what extent, if at all, does having accurate knowledge of your patient’s EM help to reduce his or her self-stigma?

7. Aim – to find out GP’s view on whether accurate knowledge of patient EM affects patient recovery

Example question: To what extent, if at all, would having accurate knowledge of your patient’s EM aid in your patient’s recovery?

8. Aim – to find out if GP has any other perspectives to share about EMs

Example question: I have come to the end of my questions. Please let me know if the interview has brought up anything else that you would like to discuss?