Destigmatising Schizophrenia: An Investigation into the Effects of Different Causal

Explanations upon Stigma

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Abstract

While many people with mental illnesses are stigmatised, those with schizophrenia are the most severely stigmatised group (Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000; Marie & Miles, 2008; Pescosolido et al., 1999). A vast body of psychology research has been devoted to investigating how education – particularly education about the causes of schizophrenia – can reduce this stigma that is attached to schizophrenia. While there is great support for the notion that education in general can reduce stigma (e.g. Costin & Kerr, 1962; Griffiths, Christensen, Jorm, Evans, & Groves, 2004; Ritterfeld & Jin, 2006), there is still disagreement regarding exactly which set of causal factors the general public should be educated about – biogenetic or psychosocial? Until now, only three previous studies (Lincoln, Arens, Berger, & Rief, 2008; Schlier, Schmick, & Lincoln, 2014; Walker & Read, 2002) have experimentally compared teaching a purely biogenetic causal explanation to teaching a purely psychosocial causal explanation. The results of this research appear to be somewhat contradictory leading to the need for another, more robustly designed experiment. In the present research, two experiments were conducted in which participants' level of stigma was measured after they were given a biogenetic causal explanation of schizophrenia, a psychosocial explanation, or given no causal explanation. It was predicted that participants given a causal explanation would show reduced levels of stigma compared to participants given no causal information, and that there would be a significant difference in the stigma reduction effectiveness between types of causal explanation. Contrary to these expectations, the results of Experiment One showed no reduction in stigma when participants were given a causal explanation compared to no causal explanation, and revealed no significant differences in stigma reduction efficacy between the biogenetic and psychosocial causal explanations. Experiment Two utilised the same basic paradigm as Experiment One but with the addition of more convincing causal explanations and a manipulation check. The results of Experiment

Two gave evidence that both a biogenetic and psychosocial causal explanation successfully reduces discrimination compared to giving no information on the causes of schizophrenia. In addition, a purely biogenetic causal explanation was also found to successfully reduce belief in other stereotypes compared to a psychosocial causal explanation or no causal explanation. Thus, I conclude that stigma can be effectively reduced by providing education about the causes of schizophrenia, and that a biogenetic causal explanation is a more effective stigma reduction tool as it reduces multiple types of stigma. Strengths, limitations, implications and future directions are discussed.

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Destigmatising Schizophrenia: An Investigation into the Effects of Different Causal Explanations upon Stigma

Schizophrenia is a severely debilitating psychiatric disorder with a range of symptoms that affect an individual's cognitions, behaviour, and emotions (American Psychiatric Association, 2013). The symptoms of schizophrenia, as defined by the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), include delusions, hallucinations, disorganised speech (including incoherence and frequent derailment), disorganised or catatonic behaviour (essentially, any type of psychomotor disturbance), and a range of negative symptoms (e.g. flat affect, lack of motivation, or poverty of speech). Fortunately, while the symptoms of schizophrenia can be severely debilitating, antipsychotic medications and regular therapy enable many patients to successfully manage their symptoms, with 80% of patients achieving remission (Robinson et al., 1999).

However, for people living with schizophrenia, managing the symptoms of their disorder is not the only battle they will face, as the majority are stigmatised simply for having a psychiatric illness (Heginbotham, 1998; Link, Struening, Neese-Todd, Asmussen, & Phelan, 2001; Rabkin, 1974). Stigma can be defined as the 'social mark' located on members of a group (who have a particular social identity or defining feature in common) that leads to those individuals being devalued, discredited, and marginalised (Goffman, 1963; Major & O'Brien, 2005). These stigmatising marks "may be visible or invisible, controllable or uncontrollable, and linked to appearance (e.g., a physical deformity), behaviour (e.g., child abuser), or group membership (e.g., African American)" (Major & O'Brien, 2005, p. 395). In the context of the current research, stigma can be conceived of as a socially constructed mark attached to people living with or showing symptoms of schizophrenia which leads to healthy individuals devaluing and mistreating patients.

While stigma affects individuals with a large range of mental illnesses, it seems that people with schizophrenia are particularly vulnerable to stigma. Research has found that schizophrenia is the most negatively viewed mental illness and that people with schizophrenia are affected to a much greater extent than people with mood disorders such as anxiety and depression (Crisp et al., 2000; Marie & Miles, 2008; Pescosolido et al., 1999). Supporting research by Griffiths et al., 2006, found that people are more likely to hold stigmatising attitudes regarding people with schizophrenia than people suffering from depression, and are more willing to engage in a relationship with someone displaying signs of depression compared to an individual with schizophrenia (Marie & Miles, 2008). For many people with schizophrenia, the stigma suffered in connection with their illness is an issue just as concerning as the management of symptoms.

The Three Components of Stigma

In the literature, stigma is conceived of as being made up of three distinct yet inextricably related components: stereotypical belief, prejudice, and discrimination (Corrigan & Watson, 2002; Corrigan, 2000; Pilgrim, 2005). The first component of this model of stigma, stereotypical belief, is the cognitive component (Corrigan & Watson, 2002; Corrigan, 2000; Pilgrim, 2005). A stereotype can be defined as a standardised idea regarding the characteristics of a particular person or group that is often based on an over-generalisation and oversimplification of inaccurate or incomplete information (Corrigan & Watson, 2002; Crisp et al., 2000; Hilton & von Hippel, 1996; Link & Phelan, 2001). For example, people suffering from depression are often stereotyped as lazy.

When an individual accepts stereotypes as true and incorporates these stereotypes into their belief system, this can lead them to be prejudiced (Devine, 1989; Hilton & von Hippel, 1996). Prejudice, the second component of stigma is the evaluative component; it is the negative emotional reactions that result from the endorsement of negative stereotypes

(Corrigan & Watson, 2002). Thus, prejudice is the direct result of holding to stereotypical beliefs. For example, an individual who believes the stereotype that peoples with depression are lazy may, upon seeing or thinking about such a person, react with feelings of anger.

In turn, prejudice can lead to discrimination, the behavioural component of stigma. Discrimination involves the stigmatiser taking negative action against the out-group or a member of the out-group as a result of their emotional reaction (Corrigan, 2004) (Croke, Major , & Steele, 1998, as cited in Corrigan & Watson, 2002). For example, an individual who has reacted in anger towards a person with depression may then restrict or completely withhold help from that person, or at least desire to do so. Thus, while the term ‘stigma’ simply refers to a socially constructed label, the results of stigma can be harmful as it causes the stigmatiser to believe false or wildly exaggerated things about the stigmatised individuals, to react negatively towards them, and to mistreat them.

Types of Stigma

The model of stigma as discussed above still shows a simplified picture of what stigma actually looks like in the real world. Each component of stigma can be expressed in many ways; there are a variety of stereotypes to believe, a wide range of negative emotional reactions, and a multitude of discriminatory behaviours. Here, we shall discuss examples of how stigma against people with schizophrenia is displayed across each of the three components. Since the subject of the present research is the stigma attached to schizophrenia specifically, the following discussion will primarily focus on those stigmas relating to schizophrenia.

Stereotypes. There are many stereotypes regarding individuals with schizophrenia, but three main stereotypes stand out as the stereotypes most often believed: that people with schizophrenia are unpredictable, dangerous, and prone to criminal behaviour (Angermeyer & Dietrich, 2006; Link, Phelan, Bresnahan, Stueve, & Pescosolido, 1999; Yildiz, Yazici,

Çetinkaya, Bilici, & Elçim, 2010). Previous studies have found that the majority of people (61% to 74%) believe individuals with schizophrenia to be dangerous (Crisp et al., 2000; Peluso & Blay, 2011; Phelan, Link, Stueve, & Pescosolido, 2000). This is a far greater degree of fear than is warranted by the rates of violence that individuals with schizophrenia actually perpetrate, meaning that this is a dangerous and unfair stereotype based on an exaggeration rather than a useful characterisation based on fact (Marie & Miles, 2008).

In addition, people with schizophrenia are often stereotyped as evil, crazy, unintelligent, hard to talk to, and unable to live independent lives, unable to care for offspring, and unable to have meaningful and fulfilling relationships (Angermeyer & Matschinger, 2003a; Arboleda-Flórez, 2008; Crisp et al., 2000; James, 1998; Miller & Mason, 2002; Pescosolido et al., 1999).

Prejudice. Prejudice includes any negative emotional reaction that a stigmatiser experiences in response to seeing, interacting with, or thinking about a person with schizophrenia. Previous research by Angermeyer and Matschinger (1997), and Corrigan (2000), has established that, when faced with people with mental illness, there are three main types of emotional reactions: fear, anger, and pity. Each of these prejudicial reactions is related to specific stereotypes about people with mental illness (Corrigan, 2000). The first, fear, has been shown to result directly from believing that people with mental illness are dangerous (Angermeyer & Matschinger, 1996; Corrigan, 2000; Link & Cullen, 1986). In turn, fear often leads to avoidance behaviours such as deliberately excluding members of the out-group from social circles (Corrigan, 2000). Anger is also known to result from the belief that people with mental illness are dangerous, but in this case it is mediated by a second belief – that people with mental illness retain personal control (Betancourt & Blair, 1992; Corrigan, 2000; Corrigan et al., 2000; Graham, Hudley, & Williams, 1992). In other words, while the fear reactions stem directly from the belief that a person with a mental illness is

dangerous, anger results from the belief that the person is dangerous but that they are able to control their behaviour. Fear reactions often lead to the stigmatiser acting in a punishing or controlling manner towards members of the out-group (e.g. forced treatment; Corrigan, 2000).

The third common emotional reaction to people with mental illness, pity, results from belief in the stereotype that people with mental illness are unable to care for themselves (Corrigan, 2000). This type of emotional reaction is different to the other two, however since it is generally regarded to be a positive reaction in the literature and is known to result in helping behaviour (Corrigan, 2000).

Discrimination. As the behavioural component of stigma, discrimination is the most visible element of stigma and, since it is the only element of stigma that directly affects the stigmatised individual in any way, it is also likely to be the most harmful component. Discrimination presents in two main forms: desire for social distance and desire for control. The first of these, desire for social distance from individuals with schizophrenia is an attitude shared by the majority of the general population. For example, 63% of a US survey sample responded that they wished to maintain a high degree of social distance from people with schizophrenia (Phelan et al., 2000). Similarly, Angermeyer, Holzinger, and Matschinger (2009) found that 60% of respondents would not accept a person with schizophrenia as a member of their same social circle and 65% answered that they would not accept a person with schizophrenia as a tenant.

According to a survey by the Canadian Mental Health Association, the most common ways in which desire for social distance affects life for patients is through exclusion from social and family life, and exclusion from employment opportunities and housing (Canadian Mental Health Association, 1994, as cited in Arboleda-Flórez, 2008). Social exclusion, a discriminatory action often built on fear, is probably the most prevalent of these three with

one survey of patients revealing 50% had experienced social exclusion (Tarrier, Khan, Cater, & Picken, 2007). Social exclusion is the most emotionally harmful form of discrimination as it threatens an individual's self-esteem and basic need to belong (Williams & Zadro, 2005) and can increase the likelihood of admission to mental health care facilities (Webber & Huxley, 2004). Unfortunately misunderstandings and prejudicial emotions such as these mean that many people living with schizophrenia face long-term unemployment (Corrigan, 2004; Pilgrim, 2005) as, far too often, mentally ill individuals are judged based on their psychiatric history, and are not accepted into employment positions that they are otherwise qualified for (Pilgrim, 2005).

English government report found that individuals suffering from mental illness are almost three times more likely to be unemployed than individuals with a physical disability (Department for Education and Employment, 1998). Surveys such as those conducted by Wahl (1999) have found that 53% of respondents had been turned down for a job for which they were qualified after their history of mental illness was revealed. In addition, experimental research such as that by Farina and Felner (1973) found that employment interviewers were less friendly and a job was less likely to be offered when job applicants admitted to having a history of mental illness.

Despite the obvious financial implications, chronic unemployment also perpetuates the individual's feelings of demoralisation and increases their chance of relapse (Warner, 1985). Finally, as Shore (1986) points out, people with schizophrenia in particular face an additional potential hardship since they frequently develop symptoms during critical higher-learning and job-development years (i.e. 18 to 35 years of age) and thus are less likely to complete training or education programs and thus will be less skilled and less able to find work (U.S. Department of Health and Human Services, 1986).

In addition to social exclusion and exclusion from employment, discrimination also presents in the desires of healthy individuals who attempt to control or at least desire control over individuals with schizophrenia. For example, many people believe that individuals living with schizophrenia should be either coerced into or forced to undertake treatment even if they do not consent (Pescosolido et al., 1999; Schnittker, 2008). Similarly, as a result of the belief that people with schizophrenia are dangerous, many individuals believe that patients should be incarcerated in mental health institutions against their will (Pescosolido et al., 1999; Schnittker, 2008). Other ways in which non-sufferers desire or attempt to gain control over individuals with schizophrenia includes limiting their ability to have children (stemming, again, from the belief that these individuals are dangerous and irresponsible) or romantic relationships (Arboleda-Flórez, 2008).

Who are the Proponents of Stigma?

A large majority of the general population are known to perpetuate stigma against people with schizophrenia (as evidenced by figures such as the high percentage of people who would be unhappy socialising with patients) but there are factors that are linked to higher levels of stigmatising attitudes. For example, demographic factors such as gender, age, ethnicity, and level of education, as well as whether an individual has had previous contact with mental health consumers or services, have all been identified as indicators of increased stigma.

Gender. The literature that has examined the relationship between gender and stigma turns up inconclusive results. On the one hand, the majority of studies examining this potential relationship have found no evidence of an association between gender and mental illness-related stigma (e.g. Angermeyer & Dietrich, 2006; Angermeyer & Matschinger, 1997; Dietrich et al., 2004; Martin, Pescosolido, & Tuch, 2000). On the other hand, several other studies have found that there is a gender difference. However, even here, the results are not

consistent as some of these studies have found that men are more stigmatising than women (e.g. Angermeyer, Matschinger, & Holzinger, 1998; Ng, Martin, & Romans, 1995; Reavley, Mackinnon, Morgan, & Jorm, 2014) while others have found the opposite (e.g. Chou, Mak, Chung, & Ho, 1996; Chou & Mak, 1998; Gaebel, Baumann, Witte, & Zaeske, 2002; Lauber, Nordt, Falcato, & Rossler, 2004).

This apparent contradiction can be resolved, however, if we remember that stigma is composed of three components and so gender may affect each of these components differently. In other words, males may be more stigmatising when it comes to one particular element of stigma while females may be more stigmatising with regards to other elements. A closer look at the results for individual measures of stigma supports this idea. For example, Angermeyer and colleagues (1998) found that women reacted in a more prosocial way towards people with mental illness and desired less social distance than men (both examples of less discrimination) but men reacted with less anxiety than women (an example of lower prejudice). Similarly, another study by Angermeyer and Matschinger (2003) found that women reacted with more pity and less anger than men but men were less fearful of people with schizophrenia.

Age. There is also some evidence that an individual's age is related to the extent to which they stigmatise people with mental illness. However, as with gender, the results are mixed. Some previous research has found evidence that the relationship between age and stigma is a positive one with findings that the belief that people with schizophrenia are more likely to commit crime than a non-sufferer is more common amongst older survey respondents (Yildiz et al., 2010), and that as age increases, social distance and personal stigma scores also increased (Reavley et al., 2014). However, interestingly, in a mass survey of the Australian general public, Reavley and Jorm (2013, p. 611) found that respondents over 60 years old were less likely to believe that schizophrenia was caused by a "weak or

nervous personality” (an example of stigmatising stereotypical belief) than respondents under the age of 30 years old. Similarly, another study found a higher proportion of respondents aged 16-19 years old held negative opinions of people with schizophrenia than respondents aged 25 years and over (Crisp, Gelder, Goddard, & Meltzer, 2005).

Contact with mental health patients and services. A third factor known to be related to stigma is whether or not the individual has had contact with someone with a mental illness or has a history of mental illness themselves. Increased contact is strongly related to lower levels of stigma. Previous research has consistently found that people who have had more contact or experience with mental illness (either personally or by proxy) had less stigmatising attitudes and opinions (Philo et al., 1994). Specifically, people who had experienced more contact perceived people with mental illness to be less dangerous and less unpredictable, and were less fearful of and less angry toward people living with schizophrenia, depression, and alcohol dependence compared to respondents who had no history of contact with people living with mental illness (Braunholtz, Davidson, Myant, & O’Connor, 2007; Read & Law, 1999; Schomerus, Matschinger, & Angermeyer, 2013). In addition, when asked specifically about people living with schizophrenia, respondents who had a history of mental illness or knew someone with a mental illness had less desire for social distance and reacted more prosocially than respondents who had no history of contact (Schomerus et al., 2013).

Of course, while these findings do provide strong evidence for a relationship between contact with mental illness and low amounts of stigmatising attitudes, there is no evidence regarding the direction of causality within this relationship. It is impossible to conclude whether increased contact causes a reduction in stigma or whether people with low levels of stigma are simply more likely to seek out contact (either in the form of relationships with patients or help for themselves), or whether neither one directly causes the other and some

third variable is at work. Fortunately, other previous research has experimentally investigated the effects of increasing individuals' contact with mental health consumers, services, and workers, and shows clear evidence that increased contact does cause a reduction/improvement in stigmatising attitudes (e.g. Clement et al., 2012; Link & Cullen, 1986; Nguyen, Chen, & O'Reilly, 2012).

Level of education. Finally, an individual's level of education is also known to be related to stigma as people with higher levels of education are less stigmatising. This effect of education on stigma extends across all three major elements of stigma. For example, stereotypes such as people with schizophrenia are unable to live independently, are more likely to commit crime, and that schizophrenia is the result of a nervous personality were believed less often by those with higher levels of education (Reavley & Jorm, 2013; Yildiz et al., 2010). In a survey of the general public, respondents who had completed high school were also less likely to react with fear when presented with a vignette describing a person with schizophrenia or depression compared to people who had not finished school (Angermeyer & Matschinger, 2003a). Phillips (1966) found that those with university-level education desired less social distance from a person living with mental illness compared to those with high school-level education.

However, while higher education and contact are both factors closely related to lower levels of stigma members of the general community are still significantly more stigmatising of people with mental illness, even mental health professionals have still been shown to frequently endorse stereotypes regarding mental illness and mental health consumers (e.g. dangerousness and unpredictability) (Braunholtz et al., 2007; Loch et al., 2011; Reavley et al., 2014; Walt & Gillis, 1979).

Consequences of Stigma

Such pervasive and widespread stigma affects individuals with schizophrenia in many ways with far-reaching and long-lasting implications. In particular, stigmatised individuals are affected emotionally psychologically, and economically.

One particularly devastating psychological effect of stigma is the decrease in already low-levels (Kesting, Mehl, Rief, Lindenmeyer, & Lincoln, 2011) of self-esteem and self-confidence (Berge & Ranney, 2005; Pilgrim, 2005; Rahav, 1987; Wahl, 1999) that many patients experience (Link, 1987; Link & Phelan, 2001). This reduction in self-esteem is the most commonly cited effect of stigma (Wahl & Harman, 1989). In addition, low self-esteem often causes people to self-stigmatise which further lowers their self-esteem, reproducing the effects of the earlier depersonalisation and disempowerment that resulted from the primary stigma encounter (Wittorf, Wiedemann, Buchkremer, & Klingberg, 2010). Second, individuals with low self-esteem often fail to pursue and achieve work or life goals that healthy individuals easily attain meaning that a stigmatised individual's quality of life and potential for life satisfaction is likely to be severely reduced (Link, 1982, as cited in Corrigan, 2004; Link, 1987).

Many individuals who have encountered stigma frequently develop a strong sense of shame relating to their mental illness which often leads these individuals to discontinue use of psychiatric medications (Sirey et al., 2001) and to avoid engaging with psychiatric and other helping services (Berge & Ranney, 2005; Leaf, Bruce, & Tischler, 1986). Furthermore, stigmatised individuals will often avoid becoming affiliated with other mentally ill individuals by withdrawing socially or avoiding engaging with other mentally ill individuals and support groups in an effort to distance themselves from the group identity and the stigma that is attached to that identity (Corrigan, 2004; Markowitz, 1998). Unfortunately, the repercussions of such cessation or avoidance of treatment and support can be severe: the

individual could relapse which may lead to situations in which the individual could be exposed to additional prejudice and discrimination. An array of other psychological and emotional effects of stigma have also been cited, including an increase in emotional sensitivity (Wahl, 1999), an increase in general distress (Quinn et al., 2014), and difficulty in making and retaining friendships (Wahl & Harman, 1989).

Finally, because many people with schizophrenia are subjected to long-term unemployment, they are more likely to live in poverty and experience all the accompanying hardships: pollution, overcrowding, weak or non-existent social networks, exposure to increased crime rates, increased likelihood of substance misuse, and a lack of access to stress-reducing activities (e.g. holidays) (Pilgrim, 2005). These adversities, in turn, can lessen the potential for life satisfaction (Pilgrim, 2005).

People with schizophrenia are not the only ones negatively impacted by stigma; the families of people with schizophrenia often encounter stigma as well. As Angermeyer (1985, p. 473) noted, having a relative develop a mental illness is “one of the most devastating and catastrophic events that they can experience”. The first major way in which families are stigmatised is through social isolation - society as a whole often builds social barriers that isolate the families as much as the individuals with schizophrenia themselves (Lefley, 1989). Secondly, the families of patients, and parents in particular, are often blamed (by society, the media, and even by mental health professionals), at least in part, for their relative’s illness, and will experience guilt and distress as a result of this stigma (Lefley, 1989). For the family members of people with schizophrenia, the results of experiencing this stigma first hand, can lead to a range of negative results/effects including the disruption of their relationships with the ill relative, a reduction in their own self-esteem, and a reduced ability to make and retain friendships (Wahl & Harman, 1989). In addition, many families of patients are indirectly affected by stigma directed towards their relative; many families experience financial

hardship as a result of financially supporting their relative who may be chronically unemployed (Lefley, 1989), and many become stressed by the day-to-day burden of emotionally supporting, calming, and protecting their vulnerable relative (Lefley, 1987; Strazdins & Broom, 2007).

Destigmatisation Interventions

It is apparent that schizophrenia is a heavily stigmatised disorder and that, in order to better the lives of patients suffering from schizophrenia, it is important to develop interventions that will reduce stigmatising attitudes and beliefs, and in particular, discrimination amongst the general population (Berge & Ranney, 2005; Lauber et al., 2004). Since the deinstitutionalization movement has relocated the majority of mental health patients out of full-time institutionalised care and into the community, the role that the community plays in recovery and rehabilitation of the mentally ill has also shifted (Aviram & Segal, 1977). As well as mental health professionals and organisations, the general public is now directly responsible for supporting mental health patients in their reintegration into the community and should be willing to play a major assisting role in the care of the mentally ill (Walkey, Green, & Taylor, 1981). As Rahav (1987) notes, probably the most important factor in determining the success of mentally ill patients' return to the community is the attitudes of the community members.

Unfortunately, as reviewed above, the general public as a whole appears to remain heavily biased toward and exclusionary of people living with mental illness. Thus, it is imperative that methods of reducing the stigma attached to mental illness are explored – an aim that a great deal of recent research and campaigns has been devoted to. According to Corrigan and Penn (2015), there are three categories of strategy that have been used in an attempt to reduce stigma: protest, education, and promotion of contact between members of the general public and people living with mental illness. My study is primarily concerned

with exploring one type of education as a method of stigma reduction. Therefore, the following discussion shall briefly review previous efforts to use education for destigmatisation before discussing the present research.

Education as a destigmatisation tool. Previous efforts to reduce mental illness stigma have primarily focussed on education because it was theorised that by providing an individual with more information regarding people living with mental illness, these individuals would show increased sympathy and empathy, and that this would result in a decrease in stigmatising attitudes, beliefs, and actions (Dohrenwend, 1962; Lemkau & Crocetti, 1962).

Evidence in support of this theory has been accumulating for the past fifty years with research in the 1960s launching the investigation into the efficacy of education as a method of reducing stigmatising attitudes and beliefs regarding mentally ill individuals. One such study by Costin and Kerr (1962) compared the attitudes and beliefs of students who had undertaken an undergraduate abnormal psychology course with those of students in an undergraduate sociology course (a control group). Costin and Kerr (1962) utilised the Opinions about Mental Illness (OMI) scale (Cohen & Struening, 1962) which measures five types of stigmatising attitudes and beliefs: beliefs regarding the dangerousness and unpredictability of patients (authoritarian attitudes), paternalistic attitudes regarding patients, desire to control patients (social restrictiveness), beliefs that the mentally ill are only different from non-sufferers in degree rather than in kind, that effective treatment of mental illnesses is possible ('mental health ideology'), and belief that interpersonal factors (e.g. the experience of deprivation of parental love during childhood) can cause mental illness.

A comparison of the end-of-semester OMI scores between abnormal psychology students and sociology students revealed mixed results. Female students enrolled in the abnormal psychology course believed patients to be less dangerous and less unpredictable

than female sociology students and the abnormal psychology students of both genders showed lower levels of paternalistic attitudes compared to the sociology students at the end of semester. In addition, the abnormal psychology students (both male and female) showed a greater belief that interpersonal psychosocial factors are the cause of mental illness compared to the sociology/control students. However, there was no difference between the abnormal psychology students and sociology students in terms of mental hygiene ideology beliefs (the belief that the mentally ill are only different from non-sufferers in degree, not in kind, and that effective treatment if mental illnesses is possible) and social restrictiveness attitudes (desire to control patients). These results provide evidence that an educational intervention focussed on abnormal psychology can effectively alter at least some attitudes and beliefs – and for the better (Costin & Kerr, 1962).

A second study by Costin and Kerr (1966) employed an identical procedure to that of the 1962 study but compared graduate students enrolled in a ‘mental hygiene’ course (which taught students about a range of mental illnesses and their causes) with students taking a course unrelated to psychology (a control group). Once again, Costin and Kerr (1966) compared the end-of-semester attitudes and beliefs of the two student groups. The results of this comparison again show mixed results; there was no difference between groups in paternalistic attitudes, mental health ideology beliefs, or social restrictiveness attitudes, but female students in the ‘mental hygiene’ course believed patients to be less dangerous and less unpredictable than the control group, and ‘mental hygiene’ students (both male and female) showed a greater belief that interpersonal psychosocial factors are the cause of mental illness compared to the control group. Again, these results indicate that an educational intervention can effectively alter at least some attitudes and beliefs (Costin & Kerr, 1966).

Several more recent studies have found additional evidence for educational interventions as effective means of reducing stigma amongst a variety of target groups. For

example, Griffiths, Christensen, Jorm, Evans, and Groves (2004) surveyed individuals with elevated depression scores before and after they were exposed to one of two web-based education interventions (a depression literacy program that emphasized biogenetic causes for depression, or a cognitive behavioural therapy [CBT] program) or no education intervention (the Control Condition). A comparison between pre-intervention and post-intervention stigma ratings revealed a greater reduction for participants assigned to the intervention conditions compared to participants in the Control Condition but no significant difference was found between the depression literacy intervention and the CBT intervention. It is important to note that the effect sizes for any reduction in stigma reported by Griffiths et al. (2004) are small (ranging from -0.09 to 0.13). Nevertheless, this result provides evidence that web-based education programs can produce a significant reduction in stigmatising attitudes regarding people with depression (Griffiths et al., 2004).

Another study investigated how effective a demythologising education program was at reducing stigmatising attitudes in members of the general population (Corrigan, Watson, Warpinski, & Gracia, 2004) by measuring participants' stigmatising attitudes and beliefs before and after an education program that debunked several common myths regarding mental illness. A comparison between pre-intervention and post-intervention stigma ratings revealed a reduction in ratings of perceived dangerousness of mental health patients as well as an increase in willingness to help these patients. Such changes in attitudes between pretest and posttest were not found amongst participants assigned to a control group. These results lead Corrigan et al. (2004) to conclude that education focussing on demythologising mental illness is an effective means of reducing harmful attitudes and increasing positive behavioural intentions regarding patients.

Ritterfeld and Jin (2006) investigated how an entertainment-education strategy can affect the amount of knowledge and the level of stigma individuals have regarding people

with mental illness. A pretest-posttest design was utilised in which participants' belief in stereotypes (e.g. the belief that patients are unable to control themselves) and their attitudes (e.g. desire to control, desire for social distance, and emotional reactions) regarding patients, were measured before and after an educational intervention. For participants in six of the eight conditions, this educational intervention involved participants watching the movie *Angel Baby* (Rymer, 1995) and watching one of six trailers (created by the researchers) in which educational information about schizophrenia was given. An additional two control conditions were created in which participants either watched a trailer prior to watching the movie, or watched no trailer at all. Trailers for the movie depicted an advocate (either a patient or a psychiatrist) who educated the viewers on schizophrenia using one of three message styles: referring to schizophrenia in general, referring to the movie specifically (e.g. naming characters as examples) and with supporting movie footage, or referring to the movie specifically but without the support of movie footage.

The results of a comparison between the pretest and posttest knowledge scores for the control group revealed that accurate knowledge scores were significantly higher at posttest than at pretest (Ritterfeld & Jin, 2006). Furthermore, a comparison of knowledge scores between the movie-only group and groups that watched a trailer revealed the latter groups to have significantly more accurate knowledge (but there was no significant difference between groups that watched the trailer after the movie and the group that watched the trailer first). This indicates that watching a film which accurately and sympathetically portrays schizophrenia results in an increase in accurate knowledge regarding this disorder, and that the addition of an educational trailer increases accurate knowledge even more (Ritterfeld & Jin, 2006).

An examination of the amount of attitude change within conditions revealed a reduction in levels of stigmatising attitudes between pretest and posttest only for the groups

that watched trailers in addition to the movie (Ritterfeld & Jin, 2006). A further comparison between groups that watched the trailer after the movie and the group that watched the trailer pre-movie revealed a significantly greater reduction in levels of stigmatising attitudes in the post-movie-trailer groups compared to the pre-movie-trailer group. This indicates that the watching of an educational trailer significantly reduces stigmatising attitudes, particularly if the trailer is viewed after the movie. Further analyses revealed that stigma reduction was mediated by perceived educational value and perceived entertainment value of the movie. Considering these results, the authors concluded that a program that combines elements of entertainment with education and advocacy is an effective method of both increasing accurate knowledge and reducing the stigma attached to mental illness (Ritterfeld & Jin, 2006).

Lastly, another recent study investigated how people's attitudes and beliefs in relation to patients with either obsessive compulsive disorder (OCD) or schizophrenia changed due to an educational intervention (Warman, Phalen, & Martin, 2015). Participants' attitudes regarding perceived dangerousness and perceived unpredictability, and their desire for social distance from a person described in a vignette (this person was either labelled as having schizophrenia, OCD, or given no label) was measured before and after an educational intervention. This intervention involved all participants being informed of the DSM-5 (American Psychiatric Association, 2013) diagnostic criteria for both schizophrenia and OCD. A comparison of pretest and posttest scores revealed a significant reduction in perceived dangerousness, perceived unpredictability, and desire for social distance from patients with both schizophrenia and OCD. Thus, it appears that simply educating non-sufferers on the diagnostic features of mental illnesses such as schizophrenia and OCD can effectively and efficiently reduce the stigma related to that disorder (Warman et al., 2015).

Considering the results of these studies, it is apparent that educational interventions are an effective means of reducing stigmatising attitudes and beliefs in members of the

general public. Thus, it is no wonder that many large-scale public education programs have been instituted with the aim of reducing stigmatising attitudes and beliefs related to mental illness and, in turn, improve the lives of those suffering from mental illnesses. For example, in New Zealand, the government has supported two very successful campaigns: the 'Like Minds, Like Mine' campaign (Vaughan & Hansen, 2004; see also <http://www.likeminds.org.nz/>) and the National Depression Initiative (Ministry of Health, 2008; Wyllie, 2009) which includes two public destigmatisation campaigns: the John Kirwan campaign (Health Promotion Agency, n.d.-a) and a web-based campaign entitled 'The Lowdown' (Health Promotion Agency, n.d.-b).

However, despite the ever-growing popularity of implementing large-scale public-education programs such as those mentioned above, many researchers continue to debate the question of exactly what information should be taught. For the most part, public-education programs have focussed on providing information on the characteristics of certain mental illnesses (e.g. diagnostic criteria, prevalence), the causes of these disorders, and on the individuals who suffer from such disorders (i.e. disproving incorrect stereotypes regarding people with schizophrenia such as the belief that they dangerous, more criminal, unpredictable, etc. (Read, 2007)

Teaching a biogenetic model. In particular, the question of what to teach the public, specifically in regards to the causes of schizophrenia, has been a hot topic for discussion. Since the 1990s, stigma reduction efforts emphasised the biological and genetic causes of schizophrenia because it was assumed that if people with schizophrenia were viewed as ill (in other words, that mental illness is disease like any other), their behaviour would be viewed as out of their own control and thus, they could not be blamed for any deviance (Read, 2007). It was further assumed that, by reducing the blame placed on patients, sympathetic understanding and lenience would replace the stigma (Read, 2007; Spiro, Siassi, & Grocetti,

1973). In other words, just as the actions of an inebriated individual are understood in the context of their being influenced by alcohol or drugs, it was thought that the actions of a mentally ill individual could and should be understood as occurring within a context of being influenced by faulty biochemical processes (Farina, Fisher, Getter, & Fischer, 1978).

Criticism of teaching a biogenetic causal model. It appears that these assumptions regarding the appropriateness of teaching a biogenetic model were unfounded (Read, Haslam, Sayce, & Davies, 2006; Read, 2007). In fact, even a review of the research published prior to the implementation of the first biogenetic-focussed stigma reduction efforts shows there was no evidence for a connection between biogenetic causal views and a positive change in attitudes (Phillips, 1966, 1967; Sarbin & Mancuso, 1970). More recent research has confirmed this. Surveys in both 1990 and 2001 revealed that belief in a biological causal explanation for schizophrenia was related to a greater desire for social distance (Angermeyer & Matschinger, 2005). Moreover, the results of trend analyses show that in the ten years between surveys, there was a significant increase in both the number of respondents who endorsed a biological explanation and an increase in the average desire for social distance (Angermeyer & Matschinger, 2005). In other words, it appears that improving the public's mental health literacy through the teaching of biological causes did not have the expected effect of improving the stigma associated with schizophrenia, and may even be increasing stigma (Read et al., 2006).

Mehta and Farina (1997) suggest that reason the biogenetic model might not reduce stigma is perhaps because people tend to patronise those who are unable to control their own behaviours (as with children). Alternatively, they suggest that the biogenetic model draws discrete divisions between healthy and unhealthy people, promoting the idea that people living with mental illness are categorically different and do not share in our common humanity (Mehta & Farina, 1997).

Teaching a psychosocial model. Considering this evidence against the usefulness of a biogenetic causal model as a method of stigma reduction, Read (2007) suggested that the general public ought to be taught about the psychosocial causes instead. Indeed, there is evidence that belief in the psychosocial causes of mental illness is related to reduced prejudice and discrimination in comparison to belief in biogenetic causes. For example, a survey of the Egyptian public found that respondents who cited social factors as a cause desired less social distance than those citing other causes (Coker, 2005). Another survey in New Zealand also found that individuals who already subscribed to a psychosocial model of mental illness desired less social distance than individuals who subscribed to a biogenetic model (Read & Harré, 2001). Furthermore, several studies have investigated the effects of demythologising seminars or courses and found that when a psychosocial model is taught, stigma is successfully reduced (Morrison, Becker, & Bourgeois, 1979; Morrison & Teta, 1979, 1980; Read & Law, 1999).

In summary, there appears to be far greater support for teaching a psychosocial causal model than for teaching a biogenetic model in an effort to reduce stigma. However, none of these studies discussed so far have directly compared the stigma-reduction efficacy of teaching these opposing causal models using an experimental paradigm. Fortunately, investigations into the effects of teaching different causal models has begun.

Experimental Research: Comparing the Effects of Two Teachings

A study by Farina, Fisher, Getter, and Fischer (1978) explored how exposure to different etiological models of mental health influenced attitudes and beliefs regarding mental illnesses and those who suffer from such disorders. Participants were provided with a causal explanation of mental illness that focussed either on biogenetic or psychosocial causes, and then completed a questionnaire asking how degrading they thought it would be to be a patient and to receive treatment, and how much control they believed patients had over their

condition. The results of both of these experiments showed that participants exposed to the psychosocial causal explanation believed patients had more control over their condition (i.e. less stigma) compared to participants who had been taught about biogenetic causes. No other differences were found.

Another study by Fisher and Farina (1979) continued this line of research using a quasi-experimental design in which university students were taught either a purely psychosocial causal view or a psychosocial-biogenetic (mixed) view as part of an abnormal psychology course. A post-intervention questionnaire revealed that participants taught the psychosocial view were less likely to believe mental health problems are out of the patients' control and placed more value on planning solutions to or seeking professional help for mental health problems compared to participants taught the mixed causal view. These results suggest that teaching a psychosocial view produces less-stigmatising attitudes than teaching a mixed causal view. Thus, it appears that an individuals' attitudes regarding mental illness are influenced by that person's causal beliefs, with participants exposed to an intervention focussed on teaching only psychosocial causes holding less-stigmatising attitudes than participants taught about both biogenetic and psychosocial causes of mental illness.

A similar study was conducted by Boysen and Vogel (2008). Participants were either given a biogenetic or psychosocial causal explanation for either schizophrenia or addiction, before being asked to rate the extent to which they perceived their attitudes to have changed because of the explanation. In rating perceived attitude change, participants considered the extent to which they agreed with four stigmatising attitudes: that mental illness is sign of weakness, that mental illness is the fault of the sufferer that non-sufferers should not live in the same neighbourhood as a patient centre, and that non-sufferers should not date people who were formerly mentally ill. Boysen and Vogel (2008) found only one significant difference in perceived attitude change between causal explanations; participants who read a

biogenetic causal explanation (for either schizophrenia or addiction) rated a greater perceived reduction in the belief that mental illness is a sign of weakness than participants who read a psychosocial explanation. No other significant differences in perceived attitude change between etiological explanations were found.

Considering the results of these three studies alone, one might conclude that, while some differences were found, overall, there is no advantage to teaching one causal explanation over another. However, a crucial limitation of Boysen and Vogel's (2008) research ought to be considered as a potential explanation for the lack of differences in perceived attitude change between conditions. The use of a subjective measure of attitude change (i.e. a self-report of perceived attitude change) rather than an objective measure such as utilising attitude scales in a pretest-posttest design and then calculating distance allows for the possibility of personal bias affecting results; it is possible that participants were simply not aware of how their attitudes were affected by the intervention and may have based their ratings of perceived attitude change on a self-conception belief of how easily influenced they are by outside information. Thus, it is possible that a study that utilises a more robust, objective measure of attitude change could return different results than Boysen and Vogel's. This limitation is addressed in later studies such as those discussed below.

Furthermore, as with Farina, Fisher, Getter, and Fischer (1978) and Fisher and Farina (1979), there was no control group included and attitudes were tested at only one time point. Without the inclusion of either a control condition or a pretest-posttest design, the picture is incomplete. More recently, research into how education about the causes of schizophrenia can reduce stigma have utilised far more robust methodologies, including control conditions in which a group of participants is not provided with any information on causal factors. To date, three studies have utilised an experimental design. All three follow a similar paradigm in which participants are taught either a biogenetic, a psychosocial, or no causal explanation.

Each of these four studies will be discussed below before proceeding on to a discussion of the present study's methods.

More Robust Experimental Research

A study conducted by Lincoln, Arens, Berger, and Rief (2008), utilised a pretest-posttest experimental design in which the influence of one of two etiological explanations for schizophrenia upon participants' level of stigma was examined. Participants were exposed to either a biogenetic or psychosocial causal explanation of schizophrenia (via a leaflet and a short video), or if they had been assigned to the control group, they read some information on glaciers and watched a video about water molecules and polar ice. The extent to which participants held stigmatising attitudes was measured at pretest and posttest both explicitly (with a 33-item questionnaire measuring perceived dangerousness, unpredictability, prognosis, and patient blame, and the seven-item Social Distance Scale [Link, Cullen, Frank, & Wozniak, 1987]) and implicitly, using an Implicit Association Test.

Lincoln and colleagues (2008) found that participants, on average, showed high levels of pre-existing stigma and that there was a significant reduction in stigmatising attitudes between pretest and posttest for both the biogenetic and psychosocial conditions (but not the control condition). This initial result seems to indicate that etiological education does reduce stigma but that efficacy of stigma reduction is not influenced by the specific type of causal explanation taught. However, post-hoc analyses revealed that both the psychosocial and biogenetic conditions showed a decrease in desire for social distance between pretest and posttest, but only the psychosocial condition showed a significant decrease in perceived dangerousness and the biogenetic intervention caused a significant reduction in patient blame for the disorder and perceived unpredictability. No significant changes were found in implicit attitudes between pretest and posttest.

Lincoln and colleagues concluded that both the biogenetic and psychosocial interventions fight stigma but that they work in different ways, reducing different individual components of stigma. Thus, they made the recommendation that an effective antistigma intervention should combine the biogenetic and psychosocial etiological explanations and teach the vulnerability-stress model instead of a one-sided causal explanation.

Considering the results of Lincoln and colleagues in isolation, such a recommendation would seem the next logical step on the road to developing an antistigma education campaign. However, research conducted before and after Lincoln et al.'s study indicates that an educational intervention that provides information on both the biogenetic and psychosocial etiologies of schizophrenia might not be as effective as one might hypothesise.

The first of these studies, by Walker and Read (2002) six years prior to Lincoln and colleagues' research utilised a pretest-posttest experimental design similar to that of Lincoln and colleagues' but with the substitution of a combined etiological explanation for the control condition. Participants were assigned to either a biogenetic causal explanation, a psychosocial causal explanation, or a combined explanation. Participants' level of stigma was measured at pretest and posttest using the Total Attitude Scale (TAS), comprising seven items which tap into participants' attitudes towards dangerousness and unpredictability of people with mental illness, whether patients should be controlled, and how much social distance should be maintained.

At the time of pretest, the mean TAS score were found to be significantly lower than the midpoint indicating that participants' pre-existing attitudes regarding people with mental illnesses were mostly negative – a result similar to the findings of Lincoln et al. (2008). An analysis of the change in TAS scores between pretest and posttest found no significant changes for any of the three conditions, overall. However, when individual scale items were examined separately, two significant differences were found: at posttest, participants in the

biogenetic condition rated mental health patients as significantly more dangerous and unpredictable compared to their pretest ratings, and participants in the combined condition also rated mental health patients as significantly more dangerous at posttest compared to pretest (Walker & Read, 2002). Additionally, contrary to expectations, the psychosocial intervention did not cause any significant improvements in attitudes (Walker & Read, 2002).

The authors concluded that the presentation of a biogenetic causal explanation does not improve attitudes toward mental health patients but, instead, caused an increase in perceptions of dangerousness and unpredictability (Walker & Read, 2002). Walker and Read (2002) suggested that destigmatisation programs should avoid the use of medical terminology such as diagnostic labels and include information on psychosocial causal factors when discussing mental illness - despite the fact their study provided no evidence that the presentation of a psychosocial causal explanation decreases stigmatising attitudes.

More recently, the stigma-reducing efficacy of educating the public with a vulnerability-stress model was again assessed in direct comparison to a biogenetic and a psychosocial intervention, but with the addition of a fourth condition - no etiological explanation. Schlier, Schmick, and Lincoln (2014) employed a pretest-posttest design, measuring participants' desire for social distance and stereotypical beliefs (dangerousness, unpredictability/incompetence, prognosis, and attribution of responsibility for disorder) regarding people with schizophrenia. Similar to the findings of Walker and Read (2002) and Lincoln et al. (2008), not all elements of stigma were changed as a result of the interventions, and those that were, were affected differently by the biogenetic and psychosocial causal explanations.

For ratings of patient dangerousness and blame, participants in the biogenetic condition showed a greater reduction in perceived dangerousness than the vulnerability-stress condition, and a greater reduction in perceived blame compared to participants in the

psychosocial condition. Stereotypes regarding patients' unpredictability and prognosis, and desire for social distance were unaffected by the interventions.

Schlier, Schmick, and Lincoln (2014) conclude from these results that an etiological education focussing on the vulnerability-stress model is no more effective at reducing stigma than a one-sided model. However, although the biogenetic intervention had the most favourable effects in terms of reducing stigma, the authors note these effects were small (they only affected two types of stereotypical belief). Thus, Schlier and colleagues make the suggestion that future research should ignore etiological education altogether, and instead focus on exploring other potential methods of stigma reduction.

Due to the contradictory nature of previous results, it is difficult to draw conclusions regarding the exact effects of different etiological explanations on particular elements of stigma. For example, Walker and Read (2002) found perceived dangerousness increased in response to a biogenetic or vulnerability-stress (combined) causal explanation, while Lincoln et al (2008) found the psychosocial explanation reduced perceived dangerousness and the biogenetic explanation caused no change. Schlier et al.'s (2014) results create further confusion with the finding that perceived dangerousness was reduced as a result of the biogenetic causal explanation. However, it can be concluded that, while there appears to be evidence that etiological education reduces overall stigma (although Walker and Read's results contradict this finding of both Lincoln et al. and Schlier et al.), there is no evidence for the theory that different causal explanations can lead to different amounts of stigma reduction.

Current Research

Since the results of previous research appear to be somewhat contradictory, it is impossible to reach a conclusion regarding whether there is difference in how stigma is affected by different causal explanations. Therefore, the current study aimed to further

explore this question. This study conceptually replicated Lincoln et al.'s (2008) experiment in that the effects of teaching different causal explanations upon participants' stigma was examined. However, unlike, Lincoln et al.'s (2008) research and other similar studies which have tended to either measure only one or two elements of stigma or have measured stigma as a single construct, the current study utilised a broader range of stigma measures allowing for each of the three components (stereotypical belief, prejudice, discrimination) to be examined fully and separately. These additions might allow for a more comprehensive and delicate exploration of the differential effects that teaching causal explanations might have on stigma levels. Thus, this study sought to both replicate and extend previous research.

The present study comprised two experiments, each directly comparing the stigma levels of participants who are exposed to either a biogenetic causal explanation, a psychosocial causal explanation, or no causal explanation (a control condition). In both experiments, I aimed to answer the following two research questions. First, I ask if teaching people about the causes of schizophrenia reduces stigma compared to no education. In other words, when participants are given taught about either the psychosocial or a biogenetic causes of schizophrenia, will they show lower levels of stereotypical belief, prejudice, and discrimination compared to participants taught nothing about the causes? Second, I ask if there is a difference between a biogenetic causal education and a psychosocial causal education in the extent to which stigma is reduced. In other words, will participants taught about the psychosocial causes of schizophrenia have lower stigma than the participants taught about the biogenetic causes of schizophrenia, as Read (2007) suggests?

Experiment One

Experiment One involved introducing participants to the concept of schizophrenia via an eight-minute compilation of video interviews of patients with schizophrenia and then exposing participants to one of two causal explanations for schizophrenia (or no causal

explanation as was the case for participants assigned to the Control Condition) before measuring their stigma levels. The three elements of stigma (Angermeyer, Matschinger, & Schomerus, 2013; Angermeyer & Matschinger, 2003b; Corrigan & Watson, 2002) were measured separately with the use of four comprehensive questionnaires: the Schizophrenia Stereotype Questionnaire (and a subscale, the Schizophrenia Perceived Dangerousness Subscale) measured participants' stereotypical belief, the Emotional Reactions Questionnaire measured participants' prejudice, and the Desire to Control Questionnaire and the Social Distance Scale measured participants' discrimination. Thus, the independent variable in this experiment was presence and type of causal explanation and the dependent variables were questionnaire scores. The mean scores for each questionnaire were compared between Experimental Conditions in an effort to investigate whether our manipulation of causal explanation significantly affected stigma.

Hypotheses and Predictions

In keeping with the results of Lincoln and colleagues (2008), I hypothesised that teaching a causal explanation of schizophrenia to members of the general public would reduce stigma. On account of this hypothesis, I predicted that participants in this experiment who are provided either a biogenetic or a psychosocial causal explanation would have lower levels of stereotypical belief, prejudice, and discrimination compared to participants who are not provided with any information on the etiology of schizophrenia (i.e. the control group).

Secondly, in light of the findings discussed above regarding the differential effectiveness of causal explanations (Lincoln et al., 2008; Schlier et al., 2014; Walker & Read, 2002), I also hypothesised that one type of causal explanation may be more effective at reducing stigma than another type of causal explanation. However, since this previous research has not all components of stigma are affected equally (e.g. Angermeyer et al., 1998; Lincoln et al., 2008), I hypothesised that, the causal explanation that was the most effective

would differ depending on which component of stigma was being examined (Boysen & Vogel, 2008; Farina et al., 1978; Fisher & Farina, 1979; Lincoln et al., 2008; Schlier et al., 2014; Walker & Read, 2002). In other words, I expect to find, as Lincoln and colleagues did, that when the three components of stigma are analysed individually, they would be affected differently by the biogenetic and psychosocial explanations.

On account of this second hypothesis, I make three predictions regarding the differential effects of the biogenetic and psychosocial conditions on the three individual elements of stigma. First, in light of previous research showing that perceived unpredictability and blame (examples of stereotypes) became reduced between pretest and posttest for participants in the biogenetic condition but not the psychosocial condition (Lincoln et al., 2008; Schlier et al., 2014), I predicted that participants provided a biogenetic explanation would show reduced levels of stereotypical belief compared to participants provided a psychosocial explanation. Second, since previous research (Lincoln et al., 2008; Schlier et al., 2014; Walker & Read, 2002) has consistently demonstrated that a biogenetic and psychosocial causal explanation affect perceived dangerousness differently, I predicted that participants provided a biogenetic explanation would show significantly different levels of perceived dangerousness compared to participants provided a psychosocial explanation. However, since the results of previous research are inconclusive regarding which causal explanation produces a more positive result, I made no prediction regarding the directionality of this difference in perceived dangerousness.

Lincoln and colleagues (2008) found that, while participants' desire for social distance was reduced between pretest and posttest for both the biogenetic and the psychosocial conditions, there was no significant difference in the amount that social distance was reduced between these two conditions. In line with these findings, I predicted that there

would be no significant difference in desire for social distance between participants provided with a biogenetic causal explanation and those provided a psychosocial causal explanation.

Since no previous research into the differential effects of causal explanations has included measures of prejudice (emotional reactions) or desire to control, I make no predictions regarding these measures of stigma.

Method

Participants

A total of 195 undergraduate psychology students enrolled in their first year of study at Victoria University of Wellington participated in a 30-minute experiment in exchange for course credit. Two participants were excluded because they incorrectly answered the final content-check question asking them to recall which causal explanation they had read as this indicated that they may have not paid sufficient attention to the video content. Two more participants were excluded because, when asked if they knew anyone who suffered from schizophrenia, they answered that they suspected that they personally showed symptoms of schizophrenia (although neither mentioned that they had been officially diagnosed). I considered that, even if these two individuals were incorrect regarding their self-diagnosis, the fact that they believed they were sufferers of schizophrenia was a significant factor that would likely bias their answers to the stigma-measuring scales.

The final sample comprised 191 participants. Of these 191 participants, 155 were female, 34 were male, and two participants identified their gender as 'other'. The average age of this final samples was 19.03 years old ($SD = 3.72$) and the majority (152 of the 191) identified at least one ethnicity as 'New Zealand European'. A small minority of participants (36 of the 191) claimed to know someone with schizophrenia. When asked if they had any personal history of mental illness, 108 of the participants answered "no" while 58 answered "yes" and 24 answered "unsure".

Participants were semi-randomly assigned to one of three conditions (the Biogenetic Condition, the Psychosocial Condition, or the Control Condition) based on which experimental session they attended. That is, each experimental session as a whole (comprising four to nine participants) was assigned to a condition prior to the commencement of that session so that all participants taking part in that session would complete the same version of the experiment. In this experiment, 62 participants were assigned to the Biogenetic Condition, 65 were assigned to the Psychosocial Condition, and 64 to the Control Condition.

Materials

Demographic questions. At the beginning of the experimental session, all participants were asked to provide some basic demographic information: age, gender, and ethnicity (participants were allowed to select more than one ethnicity category). In addition, participants were asked if they had any personal experience of a mental health issue and if they knew anyone who suffered from schizophrenia.

Video clips. All participants watched a set of three video clips, played consecutively, that each showed a person with schizophrenia talking about their life experiences. Each video showed a different patient being interviewed and answering questions about their hallucinations, delusions, and general life experiences as a person with schizophrenia, but did not provide any information regarding the causes of schizophrenia. These videos were shown to all participants in order to introduce the concept of schizophrenia in a manner that facilitated a deeper understanding of the complex concept of schizophrenia than would be possible through simply reading a description of the symptoms of schizophrenia.

Participants first watched a two minute clip of a man called Bob who was taking part in an interview for a television documentary called 'Into Madness' (Raymond & Raymond, 1989). This clip of Bob provided participants the opportunity to witness a typical presentation of two key symptoms of schizophrenia – disorganised thought and delusions – as he talked

about the spider, the praying mantis, and the grasshopper, the edge of the world, the devil coordinating the Kmart walk, “straighthood”, kidney stones, and how “the bulk wins”.

The second video clip was a three-minute section of a clinical interview of a patient called Etta taken from the instructor materials that accompany Barlow and Durand's (2008) *Abnormal Psychology: An Integrated Approach*. Etta also demonstrated two key symptoms of schizophrenia when she described auditory hallucinations and delusions of control and persecution (Jesus, The Eagle and General Motors are telling her to do things and threatening to kill her).

The third and final video clip was taken from another television documentary, and shows a patient called Gerald being interviewed by a psychiatrist, Dr Llewellyn Bigelow. During this interview, Gerald exhibited delusions of persecution as he described his belief that someone was going to put him in jail or electrocute him. Gerald also exhibited disorganised thought in terms of loose association and thought derailment. At the end of this video clip, another psychiatrist, Dr Darryl Kirch, explains to the viewers that Gerald also experiences auditory hallucinations, inappropriate mood, and delusions of grandiosity.

In total, the video material played for eight minutes.¹

Content-check questions. At two separate stages during the experimental session, participants were asked content-check questions to assess whether or not they had been

¹ All three of these videos can be viewed in their original unedited entirety on YouTube. The video of Bob can be viewed at <https://www.youtube.com/watch?v=v1XO6o-9mqQ>. The video of Etta can be viewed at <https://www.youtube.com/watch?v=mKi0aP-e-X0>. The video of Gerald can be viewed at <https://www.youtube.com/watch?v=gGnl8dqEoPQ>. Readers should note that the videos as they appear on YouTube were clipped for use in the current study.

paying attention to the video and written material. The first set of content-check questions, placed directly after the third video clip (Gerald), were created to test participants on easily-memorable topics that had been discussed by the patients in the videos in order to assess whether participants had been paying attention to the content of the videos.

These three multi-choice questions were identical for all participants, regardless of Experimental Condition, and were always shown in the same order:

1. The interviewer asks Bob about a collection of items in his room. What is this collection that Bob talks to us about?
2. Who did Etta believe was sending her messages?
3. Gerald talks about some concerns of his. Which of the following does Gerald NOT talk about?

For each of these three questions, participants were able to select only one answer (See Appendix A for the answer options provided for these questions). If participants had answered two or more of these content-check questions incorrectly, their responses would have been excluded from further analysis as they would have been judged to have paid inadequate attention. In Experiment One, no participants were excluded from analyses for this reason.

At the end of the experimental session, all participants assigned to the Biogenetic or Psychosocial Conditions were asked one final question: “Can you remember which factors we listed as potential causes of schizophrenia?” Participants could select one of two options to answer this question: either “genetics” or “trauma, problematic childhood, and stress”. It was decided that participants who incorrectly answered this question had not been paying sufficient attention to the causal explanation and these participants’ data was excluded from further analyses. Two participants were excluded for this reason.

Information about schizophrenia. All participants read three short paragraphs describing the key symptoms of schizophrenia (hallucinations, delusions, disorganised thought, and disorganised behaviour). In explaining each of these symptoms of schizophrenia, participants were explicitly reminded of the patients with schizophrenia shown in the videos and how symptoms were manifested in each of these patients. For example, participants were reminded how the subject in the second video, Etta, thought that Jesus and The Eagle were watching her when she went to post the mail – a classic example of a paranoid delusion. See Appendix B for a complete copy of these three paragraphs.

Causal explanations. Immediately after reading the description of schizophrenia and its symptoms, participants assigned to the Biogenetic Condition and the Psychosocial Condition read two paragraphs that detailed an explanation of the causes of schizophrenia. The causal explanation given to participants differed between Experimental Conditions such that participants in the Biogenetic Condition were informed that schizophrenia was caused by genetic mutations and neurochemical imbalances while participants in the Psychosocial Condition were informed that schizophrenia was caused by negative life experiences such as severe stress, trauma, and/or a problematic childhood. Participants in the Control Condition were not given any information regarding the cause of schizophrenia but moved directly on to the next stage of the experiment. (For a full copy of the causal explanations given to participants in the Biogenetic and Psychosocial conditions in Experiment One, see Appendix C.)

Scales measuring stigma. In order to measure stigma level, participants responded to forty-seven statements encapsulated within four scales (one of which included a subscale) each using five-point Likert scales (1 = “strongly disagree”, 2 = “disagree”, 3 = “neutral/undecided”, 4 = “agree”, and 5 = “strongly agree”). These statements allowed participants to rate the extent to which they believed stereotypes regarding people with

schizophrenia (e.g. that people with schizophrenia are dangerous, unpredictable, unreliable, personally weak, attention-seeking, or had criminal tendencies), the extent to which they held feelings of anger, pity, and fear, and the extent to which they desired to control individuals with schizophrenia or to separate themselves and other from people with schizophrenia. All four scales used to measure participants' stigma are discussed in further detail below (see Appendix D for a full list of all items belonging to each of the questionnaires used in the current study).

Measuring stereotypical belief. Stereotypical belief was assessed using the Schizophrenia Stereotype Questionnaire (SSQ) and the Schizophrenia Perceived Dangerousness Subscale (SPDS). The first of these scales, the SSQ, broadly measures the extent to which participants subscribe to common stereotypical beliefs regarding individuals with schizophrenia. The SSQ was created by combining an adaptation of Griffiths, Christensen, Jorm, Evans, and Groves' (2004) Personal Depression Stigma Scale (a subscale of their complete Depression Stigma Scale), a modified version of Link, Cullen, Frank, and Wozniak's (1987) Perceived Dangerousness Scale, and a sample of seven items created by the present researchers.

Griffiths and colleagues' (2004) subscale was primarily modified in order to become more schizophrenia-specific by altering the wording of each item to mention schizophrenia instead of depression. For example, "Depression is a sign of personal weakness" (Griffiths et al., 2004) was modified to read as "Schizophrenia is a sign of personal weakness". Further modifications included deleting the last two items of the Personal Depression Stigma Scale ("I would not employ someone if I knew they had been depressed", "I would not vote for a politician if I knew they had been depressed") and expanding the seventh item ("If I had depression I would not tell anyone") into two items: "If I had suffered from schizophrenia at

some point in my past, I would not tell anyone” and “I would not tell anyone if I was experiencing symptoms of schizophrenia every day in my present life”.

Schizophrenia Perceived Dangerousness Subscale. The modified version of Link and colleague's (1987) Perceived Dangerousness Scale, while incorporated into the SSQ, can also be analysed as an independent subscale. As with Griffiths and colleagues' (2004) scale, the Perceived Dangerousness Scale (Link et al., 1987) was adapted to become more schizophrenia-specific by altering the wording of each item to specifically mention “people with schizophrenia” instead of the more general “former mental patients”.

The SPDS is composed of a total of eight items to which participants respond using a five-point Likert scale ranging from “strongly disagree” to “strongly agree”. Scores on the SPDS ranged from 8 to 40 with higher scores indicating a higher level of perceived dangerousness when the participant is considering people with schizophrenia.

New items. The following seven items created by the present researcher were also included in the SSQ in order to capture other stereotypical beliefs that were not measured by items taken from the Griffiths and colleagues (2004) or Link and colleagues (1987):

- People with schizophrenia are just acting.
- Sometimes people with schizophrenia don't know what is best for them.
- People with schizophrenia are out-of-control.
- Quite often, people who suffer from schizophrenia are criminals.
- People with schizophrenia can still be intelligent.
- People with schizophrenia can still lead meaningful lives.
- People with schizophrenia can easily control their behaviour.

Total SSQ. Thus, the total SSQ comprised a total of 23 items, each of which participants respond to using a five-point Likert scale ranging from “strongly disagree” to “strongly agree”. Scores on the total SSQ ranged from 23 to 115 with higher scores indicating

a higher level of stereotypical belief and, thus, greater stigma. According to Griffiths et al. (2004), the Personal Depression Stigma Scale had good internal consistency, with a Chronbach's alpha coefficient of .76. Link and colleagues (1987) reported excellent internal consistency for their Perceived Dangerousness Scale ($\alpha = .85$). In this experiment, the complete SSQ also had good internal consistency with a Chronbach's alpha of .819, and the SPDS had good internal consistency with a Chronbach's alpha coefficient of .778.

Measuring prejudice: The Emotional Reactions Questionnaire. Recent research into the emotional reactions that individuals experience regarding people with mental illnesses has found that prejudiced emotional reactions commonly fall into one of three categories: pity, fear, or anger (Angermeyer & Matschinger, 2003a, 2003b; Schomerus et al., 2013). A confirmatory factor analysis conducted by Angermeyer and Matschinger (2003b) found that these three categories can be accurately assessed with the use of nine items: fear, uneasiness, feelings of insecurity, pity, empathy, desire to help, anger, ridicule, and irritation. The statements included in the Emotional Reactions Questionnaire used in the current study are based on these nine items. For example, ridicule was measured with the following item: "I feel like laughing at people with schizophrenia and I sometimes would like to make fun of them."

Thus, in total, the ERQ utilises nine statement-items to measure the level of prejudicial emotional reactions. Participants respond to each of these nine items using a five-point Likert scale ranging from "strongly disagree" to "strongly agree". Scores on the ERQ ranged from 9 to 45 with higher scores indicating a more prejudicial emotional reaction towards people with schizophrenia (and, thus, a higher level of stigma). Angermeyer & Matschinger (2003a) reported good internal consistency for all three emotional reaction factors, with Chronbach's alpha coefficients of .79 for the 'fear' factor, .74 for the 'pity' factor, and .77 for the 'anger' factor. In this experiment, there was good internal consistency

for only one of the three emotional reaction factors: Chronbach's alpha coefficients were .75 for 'fear', .41 for 'pity', and .48 for 'anger'. The ERQ as a whole scale was found to have moderately good internal consistency in this study with a Chronbach's alpha of .60.

Measuring discrimination. Two questionnaires were used to measure the extent to which participants discriminated against people with schizophrenia: the Desire to Control Questionnaire (DCQ) and the Social Distance Scale (SDS). The DCQ measured the extent to which participants believe that control should be exerted over people with schizophrenia (a form of discrimination). The SDS assessed the extent to which participants desire social distance from individuals with schizophrenia.

Desire to Control Questionnaire. The DCQ was developed for the purpose of this study by taking five items from the Anti-Coercive Treatment section of Nevid and Morrison's (1980) Libertarian Mental Health Ideology Scale (LMHIS) and adapting these items in such a way as to become more schizophrenia-specific. For example, items 35 ("Involuntary hospitalization is a necessary procedure in order to provide appropriate treatment for the severely mentally ill") and item 24 ("The segregation of the potentially violent patient in locked, closed wards, is a necessary form of psychiatric treatment") (Nevid & Morrison, 1980) were combined and re-worded to read: "People with schizophrenia should be hospitalised (even if they don't want to be) if their behaviour becomes too strange".

In addition to the three items created from Nevid and Morisson's LMHIS, the following five items created by the present author were included in the DCQ:

- People with schizophrenia need to be watched closely by doctors, psychiatrists, psychologists, and the police.
- If a doctor knows it will help them, people with schizophrenia should be given medications even if they don't want it.
- People with schizophrenia should not be allowed to have children

- People with schizophrenia should not be allowed to go out in public
- People with schizophrenia should not be allowed to hold normal jobs

In total, the DCQ comprised a total of eight statement-items to which participants respond using a five-point Likert scale ranging from “strongly disagree” to “strongly agree”. Scores on the DCQ ranged from 8 to 40, with higher scores indicating a greater desire to control people with schizophrenia and, thus, a greater degree of discrimination (and stigma). In this experiment, the complete DCQ had good internal consistency, with a Chronbach’s alpha coefficient of .725.

Social Distance Scale. The SDS was originally developed by Bogardus in 1925 as a Guttman-style scale and later modified by Link and colleagues (1987) to utilise a Likert-scale for each item. This scale utilises seven questions that each ask how a participant would feel associating with a person with schizophrenia across a range of social situations. In the past, this scale has been used worldwide to measure desire for social distance with regards to a range of different populations including mentally ill individuals (e.g. Dietrich et al., 2004), people with obesity (Sikorski et al., 2015), religious groups (Hunt, 1956), ethnic groups (Parillo & Donoghue, 2005), disabled people (Benton, Siegel, Derrick, & Wallace, 1968), homosexuals (Staats, 1978), and more . For the purposes of the current study, all seven questions were modified to ask specifically about social interactions with a person with schizophrenia. For example, instead of asking how the participant would feel about working with a person of a different ethnic background, the question would read “How would you feel about working at the same job as a person with schizophrenia?”

In total, the SDS comprised seven questions asking how the participant would feel interacting with a person with schizophrenia at different levels of social proximity. Participants responded to each question by rating their degree of happiness-to-interact using a five-point Likert scale ranging from “definitely unhappy” to “definitely happy”. All seven

items of the SDS were reverse-scored so that higher scores indicated a greater preference for social distance from people with schizophrenia (and thus a higher degree of discrimination and stigma).

Scores on the SDS ranged from 7 to 35. Previous research has reported good internal consistency for the SDS with Chronbach's alpha coefficients of .88 (Penn, Kohlmaier, & Corrigan, 2000), .89 (Wiesjahn, Brabban, Jung, Gebauer, & Lincoln, 2012), and .90 (Angermeyer & Matschinger, 2004; Dietrich, Matschinger, & Angermeyer, 2006). In Experiment One, there was also good internal consistency for the SDS, with a Chronbach's alpha coefficient of .91.

Procedure

Having given informed consent, all participants answered the three demographic questions regarding gender, age, and ethnicity, as well as the two questions regarding their personal mental health history and whether they know someone with schizophrenia. Following these questions, all participants watched the three short videos each depicting a person showing common symptoms of schizophrenia. At the conclusion of the videos, all participants answered three multi-choice content-check questions which assessed whether they had paid attention to the videos.

All participants then read three short paragraphs outlining the symptoms of schizophrenia. Then, participants assigned to either the Biogenetic Explanation Condition or the Psychosocial Explanation Condition read one of two causal explanations for schizophrenia (either biogenetic or psychosocial in nature, depending on the condition they had been assigned to). Participants assigned to the Control Condition did not read any information regarding the causes of schizophrenia but, instead, proceeded directly to the next task.

The final task for all participants involved the completion of a series of four questionnaires – the SSQ (including the SPDS), the ERQ, the DCQ, and the SDS – that assessed participants' attitudes and beliefs regarding individuals with schizophrenia. These four questionnaires were presented in the same order to all participants. Following these questionnaires, participants in the Biogenetic or Psychosocial Conditions were asked to recall which causal explanation they had read – either biogenetic or psychosocial. Finally, participants were provided with an opportunity to give written feedback regarding any part of the experimental session. Upon exiting the lab, all participants were provided with a debriefing document outlining the purpose of the experiment, the contact details of the researchers, where to find further information on schizophrenia, and the contact details of mental health support organisations. All participants completed this experiment within 30 minutes.

Results

Preliminary Analyses

A series of preliminary analyses were conducted in order to assess if any demographic variables were related to the dependent measures, and whether they needed to be considered as covariates when performing later analyses. Preliminary analyses involving age and ethnicity showed that these two variables had no effect on stigma and so they were not considered as covariates.

Gender. Five independent samples t-tests were conducted to compare the stigma scores for males and females. For the purposes of these t-tests, the two participants who described their gender as “other” were excluded. These t-tests revealed a significant difference for two of the five measures. First, male participants ($M = 25.85$, $SD = 4.14$) were more likely than female participants ($M = 24.22$, $SD = 4.22$) to believe that people with schizophrenia are dangerous, $t(187) = -2.05$, $p = .042$, $d = .39$. Second, male participants (M

= 22.91, $SD = 4.37$) were also more likely than female participants ($M = 20.97$, $SD = 4.05$) to want to control people with schizophrenia (through incarceration or involuntary treatment), $t(187) = -2.49$, $p = .014$, $d = .46$. Thus, gender was found to affect some elements of stigma and was treated as a covariate in later analyses involving SPDS and DCQ scores.

Participants' relationship with a patient. Five independent samples t-tests were conducted to compare stigma scores between participants who knew someone with schizophrenia ($n = 36$) and participants who did not know someone with schizophrenia ($n = 155$). These t-tests revealed a significant difference on three of the five measures. First, participants who knew someone with schizophrenia ($M = 58.39$, $SD = 8.25$) were less likely to hold stereotypical beliefs regarding people with schizophrenia compared to participants who did not know a patient ($M = 61.81$, $SD = 8.01$), $t(189) = 2.30$, $p = .023$, $d = .42$. Second, participants who knew someone with schizophrenia ($M = 16.64$, $SD = 3.23$) held less prejudicial attitudes than participants who did not know a patient ($M = 18.15$, $SD = 3.49$), $t(189) = 2.38$, $p = .018$, $d = .45$. Third, participants who knew someone with schizophrenia ($M = 19.28$, $SD = 5.37$) had a lesser desire for social distance from patients compared to participants who did not know a patient ($M = 21.19$, $SD = 4.51$), $t(189) = 2.20$, $p = .029$, $d = .39$. In short, participants who knew someone with schizophrenia showed less stigma and, thus, whether participants knew someone with schizophrenia was treated as a covariate in later analyses involving SSQ, ERQ, and SDS scores.

Personal history of mental illness. Five one-way between-groups analyses of variance (ANOVAs) were conducted to explore the impact of personal history of mental illness (PHMI) on stigma scores. Participants were divided into three groups according to their response to the following question: "Have you had any personal experience with a mental health issue (e.g. personally suffered from depression, anxiety, etc.)?" Of the total sample of 191 participants, 108 answered 'no', 58 answered 'yes', and 24 participants were

“unsure”. One participant did not answer this question and therefore was excluded from the present analyses. For each of these five ANOVAs, Levene’s test for equality of variances was conducted and was found not to be violated. Accordingly, these ANOVA results were assessed using a standard level of significance ($p < .05$).

These ANOVAs revealed a significant difference between PHMI groups on three of the five stigma measures. First, there was a statistically significant difference in SSQ scores between the three PHMI groups, $F(2, 187) = 7.07, p = .001, \eta^2 = .07$. Post-hoc comparisons using the LSD test indicated the mean SSQ score for the ‘No’ Group ($M = 63.06, SD = 8.15$) was significantly higher than the mean SSQ scores for both the ‘Yes’ Group ($M = 58.74, SD = 7.59$) and the ‘Unsure’ Group ($M = 58.58, SD = 7.66$). There was no significant difference in SSQ scores between the ‘Yes’ Group and the ‘Unsure’ Group.

Second, a statistically significant difference was also found in SPDS scores between the three PHMI groups, $F(2, 187) = 7.11, p = .001, \eta^2 = .07$. Post-hoc comparisons using the LSD test indicated the mean SPDS score for the ‘No’ Group ($M = 25.46, SD = 4.13$) was significantly higher than the mean SPDS scores for both the ‘Yes’ Group ($M = 23.22, SD = 4.19$) and the ‘Unsure’ Group ($M = 23.08, SD = 3.98$). There was no significant difference in SPDS scores between the ‘Yes’ Group and the ‘Unsure’ Group.

Finally, there was a statistically significant difference in ERQ scores between the three PHMI groups, $F(2, 187) = 4.84, p = .009, \eta^2 = .05$. Post-hoc comparisons using the LSD test indicated the mean ERQ score for the ‘No’ Group ($M = 18.57, SD = 3.50$) was significantly higher than the mean ERQ scores for both the ‘Yes’ Group ($M = 17.07, SD = 3.18$) and the ‘Unsure’ Group ($M = 16.96, SD = 3.26$). There was no significant difference in SPDS scores between the ‘Yes’ Group and the ‘Unsure’ Group.

The remaining two ANOVAs found no significant differences between the three PHMI groups in terms of DCQ scores, $F(2, 187) = 0.59, p = .555, \eta^2 = .01$, or SDS scores,

$F(2, 187) = 1.24, p = .291, \eta^2 = .01$. Thus, I conclude that, if a person has personal experience with mental health issues, belief in stereotypes (in general and those relating specifically to dangerousness), and prejudicial attitudes are reduced compared to people who have no personal experience of mental health issues. However, PHMI does not affect an individual's desire to maintain social distance from or to control people who suffer from schizophrenia. Thus, PHMI was treated as a covariate in later analyses involving SSQ, SPDS, and ERQ scores.

Primary Analyses: Dependent Variables

I predicted that when comparing participants assigned to the Control Condition with participants assigned to the other two conditions, that participants in the latter two conditions (i.e. those taught a causal explanation) would have lower levels of stigma than those in the Control Condition. When directly comparing the Biogenetic to the Psychosocial Conditions, it was expected that participants assigned to the Biogenetic Condition would show reduced levels of stereotypical belief. I also predicted that there would be a significant difference in the level of perceived dangerousness between participants in the Biogenetic and Psychosocial Conditions but no significant differences in desire for social distance.

A series of one-way between-groups analyses of variance (ANOVAs) were conducted with Experimental Condition as the between subjects variable and with covariates included as appropriate. For each of these five ANOVAs, Levene's test for equality of variances was conducted and was found not to be violated in most cases. Levene's test for equality of variances was found to be violated for the DCQ analyses but a calculation of the variance ratio assured me the results were fairly homogeneous. Accordingly, all ANOVA results were assessed using a standard level of significance ($p < .05$).

Schizophrenia Stereotype Questionnaire. The first analysis of covariance (ANOVA) examined whether there were any differences between the Experimental

Conditions (Biogenetic Condition, Psychosocial Condition, Control Condition) in terms of participants' SSQ scores, while controlling for two covariates: whether participants knew someone with schizophrenia and whether they had a personal history of mental illness.

Whether participants knew someone with schizophrenia and PHMI were both confirmed as significant covariates by this ANOVA, although these variables only explained two percent and five percent, respectively, of the variance in SSQ scores. After adjusting for both PHMI and whether participants knew a person with schizophrenia, this ANOVA revealed differences in SSQ scores between Experimental Conditions that approached significance, $F(2, 186) = 2.92, p = .056$, with a small effect size ($\eta_p^2 = .03$).

Post-hoc comparisons using the LSD test showed one significant difference between Experimental Conditions: the mean SSQ score for the Psychosocial Condition ($M = 62.82, SD = 8.80$) was significantly higher than the mean SSQ score for the Control Condition, ($M = 59.38, SD = 8.20$). The mean SSQ score for the Biogenetic Condition ($M = 61.29, SD = 7.06$) did not differ significantly from either the Psychosocial Condition or the Control Condition. In other words, participants in the Psychosocial Condition perceived people with schizophrenia in a more stereotypical way than participants in the Control Condition, but the participants in the Biogenetic Condition did not differ from either of the other conditions. These results do not confirm my predictions that participants in both the Biogenetic and Psychosocial Conditions would perceive people with schizophrenia in a less stereotypical way than participants in the Control Condition.

Schizophrenia Perceived Dangerousness Scale. A second ANOVA examined whether there were any differences between the Experimental Conditions in terms of participants' SPDS scores, while controlling for two covariates: gender and PHMI. Gender was found to not be a significant covariate but PHMI was confirmed as a significant covariate. However, a partial eta squared value of .06 indicated that PHMI only explained six

percent of the variance in SPDS scores. After adjusting for PHMI, this ANOVA revealed a statistically significant difference in SPDS scores between the conditions, $F(2, 186) = 4.04$, $p = .019$, with a small effect size ($\eta_p^2 = .04$).

Post-hoc comparisons using the LSD test indicated two significant differences between Experimental conditions. First, the mean SPDS score for the Psychosocial Condition ($M = 25.65$, $SD = 4.42$) was significantly higher than the mean the mean SPDS score for the Biogenetic Condition ($M = 24.24$, $SD = 3.66$). Second, the mean SPDS score for the Psychosocial Condition was significantly higher than the mean SPDS score for the Control Condition, ($M = 23.47$, $SD = 4.41$). There was no significant difference in the mean SPDS scores between the Biogenetic Condition and the Control Condition. In other words, participants in the Psychosocial Condition perceived people with schizophrenia as more dangerous than participants in either the Biogenetic Condition or the Control Condition, but there was no difference between participants in these two latter conditions. This result confirms my prediction that participants provided a biogenetic explanation would perceive dangerousness significantly differently compared to participants provided a psychosocial explanation. However, my prediction that participants in both the Biogenetic and Psychosocial Conditions would show significant lower amounts of stereotypical belief (of which perceived dangerousness is a part) was not confirmed.

Emotional Reactions Questionnaire. A third ANOVA examined whether there were any differences between the Experimental Conditions in terms of participants' ERQ scores, while controlling for two covariates: whether participants knew someone with schizophrenia and whether they had a personal history of mental illness. Whether participants knew someone with schizophrenia and PHMI were both confirmed as significant covariates by this ANOVA, although these variables only explained two percent and four percent, respectively, of the variance in ERQ scores. After adjusting for both PHMI and whether participants knew

a person with schizophrenia, this ANOVA revealed no significant differences in ERQ scores between the conditions, $F(2, 186) = 1.194, p = .305, \eta_p^2 = .01$. In other words, participants' prejudicial attitudes (or their emotional reactions) regarding people with schizophrenia was not affected by Experimental Condition. These results do not support my prediction that participants in both the Biogenetic and Psychosocial Conditions would show significantly less prejudice than participants in the Control Condition.

Desire to Control Questionnaire. A fourth ANOVA examined whether there were any differences between the Experimental Conditions in terms of participants' DCQ scores, while controlling for gender as a covariate. Gender was found to not be a significant covariate. The remaining results of this ANOVA found a statistically significant difference in DCQ scores between the conditions, $F(2, 187) = 3.70, p = .027$, with a small effect size ($\eta_p^2 = .04$).

Post-hoc comparisons using the LSD test indicated one significant difference between Experimental Conditions: the mean DCQ score for the Psychosocial Condition ($M = 22.35, SD = 4.20$) was significantly higher than the mean DCQ score for the Control Condition, ($M = 20.30, SD = 4.72$). There was no significant difference between the mean DCQ score for the Biogenetic Condition ($M = 21.15, SD = 3.23$) and either the Psychosocial Condition or the Control Condition. In other words, participants in the Psychosocial Condition wished to control people with schizophrenia more than participants in the Control Condition, but the participants in the Biogenetic Condition do not differ from either of the other conditions. This result does not support my prediction that participants in both the Biogenetic and Psychosocial Conditions would show significantly less discrimination (of which desire for control is an element) than participants in the Control Condition.

Social Distance Scale. The fifth ANOVA examined whether there were any differences between the Experimental Conditions in terms of participants' SDS scores, while

controlling for one covariate; whether participants knew someone with schizophrenia. This variable was confirmed to be a significant covariate, although it only explained three percent of the variance in SDS scores. After adjusting for whether participants knew a person with schizophrenia, this ANOVA revealed no significant difference in SDS scores between the Experimental Conditions, $F(2, 187) = 1.24, p = .291, \eta_p^2 = .01$. In other words, participants' desire for social distance from people with schizophrenia was not affected by Experimental Condition. While this results supports my fourth prediction that participants in the Biogenetic and Psychosocial Conditions would not differ on desire for social distance, it does not support my first prediction that participants in both the Biogenetic and Psychosocial Conditions would show significantly less discrimination (of which desire for social distance is an element) than participants in the Control Condition.

Conclusion

In summary, the results of this first experiment show three key findings. First, contrary to my predictions, there were no significant differences in prejudicial attitudes and desire for social distance between Experimental Conditions, indicating that these elements of stigma were not affected by our experimental manipulation. Second, and also contrary to my predictions, participants in the Psychosocial Condition were found to have higher levels of stereotypical belief regarding schizophrenia and were more likely to desire control over individuals with schizophrenia compared to participants in the Control Condition. However, participants in the Psychosocial Condition showed no difference in levels of stereotypical belief and were no more likely to desire control over individuals with schizophrenia compared to participants in the Biogenetic Condition. This result indicates that, while providing a psychosocial causal explanation appears to increase stereotypical belief and desire for control over people with schizophrenia compared to providing no causal explanation, it is no better or worse than providing a biogenetic causal explanation. Our final

finding is that participants in the Psychosocial Condition perceived a higher level of danger from people with schizophrenia compared to participants in either the Biogenetic or Control Conditions, indicating that providing a psychosocial causal explanation increases perceived dangerousness compared to providing either a biogenetic causal explanation, or none at all.

Considering the above findings, it is apparent that our results are rather complex. I can conclude that an experimental manipulation of causal explanations returns mixed results and that all elements of stigma are not affected equally. Teaching members of the general public a biogenetic causal model does not increase stigma and teaching a psychosocial causal model does not reduce stigma, as previous authors have claimed (Read & Harré, 2001; Read, 2007). In fact, the results of this current experiment indicate that teaching a psychosocial causal explanation can result in an increase in stigma compared to teaching a biogenetic causal explanation or no causal explanation at all. Thus, it appears that, contrary to the claims of Read (2007), teaching a purely psychosocial causal explanation is not an effective method of reducing schizophrenia-related stigma in members of the general population. Similarly, providing information on the biogenetic causes of schizophrenia is not something to be avoided as it is apparent that such information does not lead to an increase in stigma; if anything, a biogenetic causal model will lead to reduced stigma.

Discussion

Experiment one explored two key research questions. First, I investigated whether providing an education about the causes of schizophrenia reduces stigma compared to no education, and second, whether there was any difference in the stigma reduction between a psychosocial education and a biogenetic education. The results of this experiment gave no evidence that providing an etiological education reduced stigma and only minimal evidence that there was a difference in stigma reduction effectiveness between a biogenetic and a psychosocial education. Thus, my preliminary conclusion is that an etiological education is

not an effective means of reducing the stigma attached to schizophrenia. A series of ANOVAs comparing participants' scores on five different measures of stigma supports this conclusion.

There were no significant differences in prejudice (as measured by the ERQ) or desire for social distance (a measure of discrimination) between the three experimental groups indicating that these components of stigma were unaffected by etiological education. In addition, all other analyses found that participants given a causal explanation (either biogenetic or psychosocial) were either just as likely or even more likely to hold stigmatising attitudes compared to participants given no causal explanation. Thus, contrary to my prediction, providing an education on the causes of schizophrenia did not lead to lesser amounts of stereotypical belief, prejudice, or discrimination.

There was only one instance of a significant difference appearing between participants provided with a biogenetic education and participants given the psychosocial education; a significant difference in SPDS scores showed that a psychosocial causal explanation leads to an increase in perceived dangerousness compared to a biogenetic explanation. This result does support my prediction that there would be a difference in the amount of stigma reduction between the Biogenetic and Psychosocial Conditions. On the other hand, previous research has found that teaching a psychosocial causal model results in either a decrease or no change in perceived dangerousness (Lincoln et al., 2008; Schlier et al., 2014; Walker & Read, 2002) – findings that completely contradict the results of the present research. Perceived dangerousness is just one component of stigma, however, and since all other analyses comparing the Biogenetic and Psychosocial Conditions revealed no significant differences, it is sensible to conclude that there is no major difference in the effectiveness of these two causal explanations in terms of reducing stigma.

In summary, experiment one provides no evidence that different etiological explanations affect stigma differently or that an etiological education is an effective method of reducing the stigma attached to schizophrenia – a result that contradicts previous research. As discussed previously, several studies have shown that etiological education is linked to stigma reduction. For example, two studies by Costin and Kerr (1962, 1966) found that people taught about the causes of mental illnesses in an abnormal psychology course held less paternalistic attitudes regarding patients and thought patients were less dangerous and less unpredictable than people not enrolled in the course. Several studies examining the effectiveness of seminars focussed on teaching participants about the psychosocial causes of mental illnesses have also found that evidence of successful stigma reduction (Morrison et al., 1979; Morrison & Teta, 1979, 1980; Read & Law, 1999). Other studies have focussed on comparing different types of etiological education, and while they disagree on which type of causal explanation is the most effective, many found evidence that teaching people something about the causes of schizophrenia is an effective method of stigma reduction. For example, Lincoln et al.'s (2008) results showed a significant reduction in stigmatising attitudes such as endorsement of stereotypes, blaming attitudes, and desire for social distance, when participants were taught about the causes of schizophrenia.

Therefore, considering that several studies have evidence that an etiological education reduces stigma, it is surprising that the results of the present experiment reveal no stigma reduction effect for either the Biogenetic or the Psychosocial Conditions. One possible explanation is that the majority of participants in experiment one held similar causal beliefs prior to engagement with the educational material and their beliefs were unchanged by this education because they were not convinced by the information they read. The methodology of this experiment was based on the assumption that participants who read an explanation of the causes of schizophrenia would replace their pre-existing beliefs with this new

information. If participants in experiment one were not convinced that the causal explanation was true, they may not have accepted that information into their conception of schizophrenia and its origins. Instead, they may have rejected the causal explanation provided and retained their original beliefs. If this was the case, participants' attitudes would only be based on the beliefs they entered the experiment with, rather than new beliefs matching the causal explanation they were exposed to.

Considering that the participants would have been exposed to information on the causes of schizophrenia prior to this experiment, many would be entering with pre-existing causal beliefs. It is possible that the majority of participants in experiment one held similar causal beliefs; especially considering that participants were sampled from a first year psychology course and therefore, had likely been exposed to similar information as a result of shared interests in the field of psychology. Since a person's attitudes are likely based on their beliefs, if participants' beliefs were not changed as a result of the experimental manipulation, participants' attitudes would also remain unchanged. Thus, I hypothesised that few between-groups differences were found because participants entered with similar beliefs and attitudes which remained unchanged by the educational interventions.

In connection with this hypothesis, I identify two key limitations with experiment one. First, the causal explanations provided to participants in the Psychosocial and Biogenetic Conditions were short (160 to 186 words), used cautious language, and only gave a brief overview of the causes of schizophrenia. As a result, participants may not have found these causal explanations convincing. Second, there was no check to ensure that participants accepted the causal explanation and believed it. To rectify these shortcomings, a second experiment was designed using the same basic paradigm but with several key alterations made to the causal explanations and with the addition of a manipulation check.

First, the causal explanations given to participants were altered significantly in terms of the language used, as well as the length and depth of the explanations. In Experiment One, the causal explanations were short and were written in a cautious manner. As discussed above, it is possible that by using short and carefully-worded causal explanations, there may be limited opportunity for participants to review and change their beliefs regarding the causes of schizophrenia, thus limiting the chances of seeing a between-groups difference in stigma scores. Considering this, substantial alterations were made to the causal explanations for Experiment Two in an effort to make them more convincing. The causal explanations were re-written in such a way that more in-depth and comprehensive information regarding the causes of schizophrenia was given to participants. For example, instead of only briefly mentioning that schizophrenia is caused by genes, the biogenetic causal explanation used in experiment two was expanded into a three-paragraph explanation of how genes contribute to the symptoms of schizophrenia and what evidence there is for the role of genes. Incidentally, as a result of adding more comprehensive information to the causal explanations, they were significantly lengthened (becoming at least a page long) which I hoped would allow participants more time to consider the validity of the causal explanations, and more opportunity for belief review.

In addition to including more in-depth explanations, stronger language was used to facilitate these explanations. For example, the psychosocial causal explanation used in Experiment One said “Researchers are still investigating the causes of schizophrenia but one popular theory says that schizophrenia is caused, at least in part, by the environment.” This statement and others like it in the education material for Experiment One was worded in a cautious manner which may have given participants the impression that this information was not fact. In contrast, the psychosocial causal explanation used in Experiment Two stated that “Schizophrenia is a form of mental instability that is caused by negative factors or events in a

person's social life." This statement leaves less room for doubt. It was my hope that these new causal explanations would be more convincing and, thus, believe all the information provided to be fact.

The second major alteration made to the causal explanations was the addition of a supporting image (see Appendix E). For the biogenetic causal explanation, a picture of two MRI scans showing the difference in ventricle size between a patient with schizophrenia and a healthy control was used to support the explanation that schizophrenia has been linked to neural abnormalities. For the psychosocial causal explanation, a graph demonstrating the link between population density and number of people with schizophrenia was used to support the explanation of how different social factors have been linked to the development of schizophrenia symptoms. It was hoped that these images would not only provide more information, but would allow participants to process this information in more than one way, thus allowing more opportunity to accept the causal explanation.

Finally, an introductory note was added to the causal explanation informing participants that they were reading an excerpt from a textbook on abnormal psychology written by a (fictional) doctor. This introduction read: "The following is an excerpt taken from a chapter of the popular psychology textbook: 'Introduction to Abnormal Psychology' written by Dr B. R. Ainsworth." All three of these alterations to the causal explanations were made in an effort to increase the convincingness of the explanations.

In addition to these alterations made to the causal explanations, a manipulation check was added to Experiment Two. This manipulation check consisted of a single question placed at the end of the experimental session which asked participants how convinced they were by the 'textbook excerpt' they had read. Since one concern with the methodology of Experiment One was that there was no way to check that participants actually accepted the information provided in the causal explanations, this measure of convincingness was included in order to

determine whether participants were accepting the information they read in the causal explanations. If participants rated the 'textbook excerpts' as convincing, this would mean they believed the information to be valid and likely accepted it. Then, if no between-groups differences were found in Experiment Two, the possibility that the information provided wasn't convincing enough could be ruled out. Additional content check questions which assessed participants' recall for key details discussed in the 'textbook excerpts' were also included in Experiment Two to assess participants' attention to material.

Experiment Two

Experiment Two was conducted in order to investigate if the findings of Experiment One are repeated when the causal explanations are more convincing. Experiment Two uses a similar experimental methodology but the causal explanations were significantly altered and a manipulation check was added. Participants in Experiment Two again watched an eight-minute compilation of video interviews of patients with schizophrenia, read one of two causal explanations for schizophrenia (or no causal explanation if they were assigned to the Control Condition), and then completed four questionnaires to measure their levels of stereotypical belief, prejudice, and discrimination. At the end of the experimental session, following completion of the scales, participants in the Biogenetic Condition or the Psychosocial Condition were also asked to rate how convinced they had been by the information they read in the causal explanation. This allowed for an assessment of whether the information presented in the causal explanations had been accepted by the participants because a limitation of Experiment One had been the lack of a manipulation check. Thus, as in Experiment One, the independent variable in this experiment was presence and type of causal explanation and the dependent variables were questionnaire scores.

Hypotheses

As for Experiment One, I hypothesised that teaching a causal explanation of schizophrenia to members of the general public would reduce stigma. I also hypothesised that a causal explanation would not affect all elements of stigma equally so that in some cases, one type of causal explanation may be more effective at reducing some components of stigma, while in other cases, both a psychosocial and a biogenetic explanation may be equally effective.

While the results of Experiment One did not confirm these hypotheses, previous research (Boysen & Vogel, 2008; Fisher & Farina, 1979; Lincoln et al., 2008; Schlier et al., 2014; Walker & Read, 2002) has found evidence that teaching a causal explanation can reduce stigma and that biogenetic and psychosocial explanations affect the individual components of stigma differently. Since it is possible that this could be explained by the causal explanations not being convincing enough, Experiment Two was conducted in order to investigate more convincing causal explanations would produce results in line with those of Experiment One or results concurrent with previous research (and, thus, my expectations). It was my expectation that the latter would be true. Thus, I retained my original hypotheses and predictions regarding the effect of the experimental manipulation on stigma scores.

Method

Participants

A total of 158 undergraduate psychology students enrolled in their first year of study at Victoria University of Wellington participated in a 45-minute experiment in exchange for course credit. Eight participants (five male, three female) were excluded from analyses for incorrectly answering two or more of the content-check questions as this indicated they may not have paid sufficient attention to the video and reading material in the experiment. The final sample comprised 150 participants, of which 124 were female and 26 were male. The

average age was 19.61 ($SD = 5.07$) and the majority (101 of the 150) identified at least one ethnicity as 'New Zealand European'. A small minority of participants (25 of the 150) claimed to know someone with schizophrenia. When asked if they had any personal history of mental illness, 87 of the participants answered "no" while 46 answered "yes", 16 answered "unsure" and one participant did not answer.

As in Experiment One, participants were semi-randomly assigned to one of three conditions (the Biogenetic Condition, the Psychosocial Condition, or the Control Condition) based on which experimental session they attended. That is, each experimental session as a whole (comprising four to nine participants) was assigned to a condition prior to the commencement of that session so that all participants taking part in that session would complete the same version of the experiment. In Experiment Two, 51 participants were assigned to the Biogenetic Condition, 46 were assigned to the Psychosocial Condition, and 53 to the Control Condition.

Materials

Demographic questions. As in Experiment One, at the beginning of the experimental session, all participants were asked to provide some basic demographic information: age, gender, and ethnicity (participants were allowed to select more than one ethnicity category). In addition, participants were asked if they had any personal experience of a mental health issue and if they knew anyone who suffered from schizophrenia.

Video clips. All participants watched a set of three video clips, played consecutively, that each showed a person with schizophrenia talking about their life experiences. These videos were identical to and were played in the same order as those used in Experiment One. In total, the video material played for eight minutes.

Content-check questions. At two separate stages during the experimental session, participants were asked content-check questions to assess whether or not they had been

paying attention to the video and written material. The first set of content-check questions, placed directly after the third video clip (Gerald), were created to test participants on easily-memorable topics that had been discussed by the patients in the videos in order to assess whether participants had been paying attention to the content of the videos. These three questions were identical to and were presented in the same order as the first set of content-check questions used in Experiment One. For each of these three questions, participants were able to select only one answer.

A second set of questions was placed immediately following the presentation of the causal explanation and assessed whether participants in the Biogenetic and Psychosocial Conditions had been paying attention to the content of these explanations. Participants in both conditions answered four questions regarding the causal explanation (see Appendix E for a full list of these questions). For participants in the Control Condition, no content-check questions were presented since no causal explanation had been given; these participants proceeded directly from reading a brief paragraph giving general information on schizophrenia to completing the stigma questionnaires.

If participants answered two or more of these content-check questions incorrectly, their responses were excluded from further analysis as they would have been judged to have paid inadequate attention. As previously mentioned, eight participants were excluded from analyses for this reason.

Information about schizophrenia and causal explanation. All participants in Experiment Two were provided with information about the symptoms of schizophrenia (this information was identical to that provided to participants in Experiment One). Participants assigned to either the Biogenetic or Psychosocial Conditions were also provided with a causal explanation. All participants, regardless of the condition they were assigned to, were told in an introductory note that the written material was an excerpt from an abnormal psychology

textbook written by a (fictional) author. This misleading detail was included in an effort to convince participants to accept the causal explanation they read.

Just as in Experiment One, the causal explanation given to participants differed between Experimental Conditions such that participants in the Biogenetic Condition were informed that schizophrenia was caused by genetic mutations and neurochemical imbalances while participants in the Psychosocial Condition were informed that schizophrenia was caused by negative life experiences such as severe stress, trauma, and/or a problematic childhood. As discussed earlier, the causal explanations provided in this second experiment were significantly altered from those used in the first experiment; they were longer, included more in-depth information, incorporated stronger language, and each was accompanied by an image. For a full copy of the causal explanations given to participants in the Biogenetic and Psychosocial Conditions in Experiment Two, see Appendix F.

Scales measuring stigma. In order to measure stigma level, participants completed the same four scales (including one subscale) that were used in Experiment One.

Measuring stereotypical belief. The extent to which participants believed stereotypes about people with schizophrenia was measured with the Schizophrenia Stereotype Questionnaire (SSQ). A subscale of the SSQ, the Schizophrenia Perceived Dangerousness Subscale (SPDS), measured the extent to which participants believed stereotypes specifically about the dangerousness of people with schizophrenia. Previous research has found these scales to have good internal consistency. In Experiment Two, these scales also had good internal consistency with Chronbach's alphas of .85 for the complete SSQ, and .795 for the SPDS.

Measuring prejudice. The extent to which participants reacted in a prejudicial manner to people with schizophrenia was measured with the ERQ. Previous research has found this scale to have good internal consistency. In Experiment Two, there was good internal

consistency for one of the three emotional reaction factors and moderately good internal consistency for another: Chronbach's alpha coefficients were .75 for 'fear', .62 for 'pity', and .45 for 'anger'. The ERQ as a whole scale was found to have moderately good internal consistency in this study with a Chronbach's alpha of .65.

Measuring discrimination. Finally, two types of discrimination were measured. Participants' desire to control people with schizophrenia was measured with the Desire to Control Questionnaire (DCQ). The DCQ is a new scale, developed by the author, and so there is no previous research into the reliability of this scale. In Experiment Two, however, the complete DCQ has good internal consistency, with a Chronbach's alpha coefficient of .75.

Participants' desire to maintain social distance from people with schizophrenia was measured with the Social Distance Scale (SDS). Previous research has reported good internal consistency for the SDS. In Experiment Two, there was also good internal consistency for the SDS, with a Chronbach's alpha coefficient of .872.

Manipulation check. At the end of the experiment, all participants were asked to rate how convinced they were by what they had read in the 'textbook excerpt'. Ratings were made on a sliding scale that ranged from one ("Not at all convincing") to ten ("Extremely convincing").

Procedure

Having given informed consent, all participants answered the three demographic questions regarding gender, age, and ethnicity, as well as the two questions regarding their personal mental health history and whether they know someone with schizophrenia. Following these questions, all participants watched the three videos each depicting a person showing common symptoms of schizophrenia. At the conclusion of the videos, all participants answered three multi-choice content-check questions which assessed whether they had paid attention to the videos.

All participants then read three short paragraphs outlining the symptoms of schizophrenia. Then, participants assigned to either the Biogenetic Condition or the Psychosocial Condition read one of two causal explanations for schizophrenia (either biogenetic or psychosocial in nature, depending on the condition they had been assigned to). Immediately following the presentation of the causal explanations, participants in the Biogenetic and Psychosocial Conditions answered another four multi-choice content-check questions which assessed whether they had paid attention to the content of the causal explanations. Participants assigned to the Control Condition did not read any information regarding the causes of schizophrenia, nor did they answer a second set of content-check questions. Instead participants in the Control Condition proceeded directly from reading about the symptoms of schizophrenia to completing the stigma questionnaires.

Next, all participants involved the completion of four questionnaires – the SSQ (including the SPDS), the ERQ, the DCQ, and the SDS – which assessed participants' attitudes and beliefs regarding individuals with schizophrenia. These four questionnaires were presented in the same order to all participants. Having completed all four questionnaires, all participants were asked to rate, on a scale from one to ten, how convinced they were by what they read in the 'textbook excerpt'.

Finally, participants were provided with an opportunity to give written feedback regarding any part of the experimental session. Upon exiting the lab, all participants were provided with a debriefing document outlining the purpose of the experiment and correcting the biased information the participants had read by explaining that both biogenetic and psychosocial factors are considered important in the etiologic of schizophrenia. In addition, this debriefing document provided participants with the contact details of the researchers, information on where to find further information on schizophrenia, and the contact details of

mental health support organisations. All participants completed this experiment within 45 minutes.

Results

Preliminary Analyses

A series of preliminary analyses were conducted in order to assess if any demographic variables were related to the dependent measures, and whether they needed to be considered as covariates when performing later analyses. Preliminary analyses involving age, participants' relationship with a patient, and personal history of mental illness showed that these three variables had no effect on stigma and so they were not considered as covariates.

Gender. A series of independent samples t-tests were conducted to compare the stigma scores for males ($n = 26$) and females ($n = 124$). Male participants ($M = 63.77$, $SD = 8.30$) were found to be more likely than female participants ($M = 59.81$, $SD = 8.85$) to hold stereotypical beliefs regarding people with schizophrenia, $t(148) = -2.10$, $p = .038$, $d = .46$. Male participants ($M = 26.08$, $SD = 4.58$) were also more likely than female participants ($M = 23.85$, $SD = 4.71$) to perceive people with schizophrenia as dangerous, $t(148) = -2.21$, $p = .029$, $d = .48$. Third, male participants ($M = 22.31$, $SD = 3.78$) were more likely to desire the power to control people with schizophrenia (through incarceration or involuntary treatment) than female participants ($M = 20.35$, $SD = 4.44$), $t(148) = -2.10$, $p = .038$, $d = .48$. Thus, gender was treated as a covariate in later analyses involving SSQ, SPDS, or DCQ scores.

Ethnicity. A series of independent samples t-tests were conducted to compare stigma scores between ethnicity groupings. For the purpose of these analyses, participants were placed into one of two ethnicity groups: the first group included participants who identified as 'NZ European' or 'Other European' ($n = 104$), and the second group included participants of

all other ethnicities (e.g. Maori, Pacific Islander, Asian; $n = 46$). These analyses revealed four key differences between ethnicity groups.

Non-European participants ($M = 64.04$, $SD = 8.51$) were found to hold more stereotypical beliefs regarding people with schizophrenia than Europeans ($M = 58.92$, $SD = 8.59$), $t(148) = -3.38$, $p = .001$, $d = .60$. Similarly, Non-European participants ($M = 26.15$, $SD = 4.94$) perceived people with schizophrenia as more dangerous than Europeans ($M = 23.38$, $SD = 4.43$), $t(148) = -3.41$, $p = .001$, $d = .59$. Non-European participants ($M = 22.13$, $SD = 4.65$) also desired more control over people with schizophrenia than Europeans ($M = 20.05$, $SD = 4.13$), $t(148) = -2.74$, $p = .007$, $d = .47$. Finally, Non-European participants ($M = 20.65$, $SD = 4.27$) desired more social distance from people with schizophrenia than European participants ($M = 19.17$, $SD = 3.85$), $t(148) = -2.10$, $p = .037$, $d = .36$. The only way in which European participants did not differ from Non-European participants was in terms of prejudicial feelings towards people with schizophrenia. Thus, ethnicity was treated as a covariate in later analyses involving SSQ, SPDS, DCQ, or SDS scores.

Primary Analyses: Dependent Variables

It was expected that participants assigned to the Biogenetic and Psychosocial Conditions would show less stereotypical belief, prejudice, and discrimination than participants assigned to the Control Condition. It had also expected that participants assigned to the Biogenetic Condition would show a lesser degree of stereotypical belief than participants in the Psychosocial Condition and the extent to which participants perceive people with schizophrenia as dangerous would differ between these two groups. To examine these hypotheses, a series of one-way between-groups ANOVAs were conducted with Experimental Condition as the between subjects variable and with covariates controlled for as appropriate. For each of these five ANOVAs, Levene's test for equality of variances was conducted and was found not to be violated in most cases; Levene's test was violated for the

ERQ analysis. Accordingly, ANOVA results for the ERQ analyses were assessed using the Welch test of equality of means. All other analyses were assessed using a standard level of significance ($p < .05$).

Schizophrenia Stereotype Questionnaire. The first ANOVA examined whether there were any differences between the Experimental Conditions (Biogenetic Condition, Psychosocial Condition, Control Condition) in terms of participants' SSQ scores, while controlling for two covariates; gender and ethnicity. Gender and ethnicity were both confirmed as significant covariates by this ANOVA, although these variables only explained five and eight percent of the variance in SSQ scores, respectively. After adjusting for both gender and ethnicity, this ANOVA revealed a significant difference in SSQ scores between Experimental Conditions, $F(2, 145) = 7.45, p = .001$, with a small effect size ($\eta_p^2 = .09$).

Post-hoc comparisons using the LSD test revealed two significant differences between Experimental Conditions: the mean SSQ score for the Psychosocial Condition ($M = 60.96, SD = 7.95$) was significantly higher than the mean SSQ score for the Biogenetic Condition ($M = 57.12, SD = 10.06$), and the mean SSQ score for the Control Condition ($M = 63.34, SD = 7.29$) was significantly higher than the mean SSQ score for the Biogenetic Condition. There was no significant difference in SSQ scores between the Psychosocial Condition and the Control Condition. In other words, participants in the Psychosocial Condition and the Control Condition perceived people with schizophrenia in a more stereotypical way than participants in the Biogenetic Condition, but the participants in the Psychosocial and Control Conditions did not differ from each other in terms of amount of stereotypical belief.

Schizophrenia Perceived Dangerousness Scale. A second ANOVA examined whether there were any differences between the Experimental Conditions in terms of participants' SPDS scores, while controlling for two covariates; gender and ethnicity. Gender and ethnicity were both confirmed as significant covariates by this ANOVA, although these

variables only explained five and nine percent of the variance in SPDS scores, respectively. After adjusting for both gender and ethnicity, this ANOVA revealed a significant difference in SPDS scores between Experimental Conditions, $F(2, 145) = 6.28, p = .002$, with a small effect size ($\eta_p^2 = .08$).

Post-hoc comparisons using the LSD test revealed one significant difference between Experimental Conditions: the mean SPDS score for the Control Condition ($M = 25.92, SD = 4.59$) was significantly higher than both the mean score for the Biogenetic Condition ($M = 22.75, SD = 4.91$). However, there was no significant difference in SPDS scores between the Psychosocial Condition ($M = 23.93, SD = 4.20$) and either the Biogenetic Condition or the Control Condition. In other words, participants in the Control condition perceived people with schizophrenia to be more dangerous than participants in the Biogenetic Condition, but participants in the Psychosocial Condition did not differ from participants in either of the other two conditions in terms of perceived dangerousness.

Emotional Reaction Questionnaire. A third ANOVA examined whether there were any differences between the Experimental Conditions in terms of participants' ERQ scores. Preliminary analyses had shown that none of the independent variables influenced ERQ scores. Thus, no covariates were controlled for in this ANOVA. As previously mentioned, Levene's test for equality of variances was violated for the present analysis, $F(2, 147) = 3.85, p = .024$. Accordingly, the Welch test of equality of means. This one-way ANOVA with Experimental Condition as the between-subjects factor revealed no significant differences in ERQ scores between the conditions, $F(2, 95.71) = 1.64, p = .200, \eta^2 = .02$. In other words, participants' prejudicial attitudes (or emotional reactions) regarding people with schizophrenia was not affected by Experimental Condition.

Desire to Control Questionnaire. The fourth ANOVA examined whether there were any differences between the Experimental Conditions in terms of participants' DCQ scores,

while controlling for two covariates; gender and ethnicity. Gender and ethnicity were both confirmed as significant covariates by this ANOVA, although these variables only explained four and three percent of the variance in DCQ scores, respectively. After adjusting for both gender and ethnicity, this ANOVA revealed a difference in DCQ scores between Experimental Conditions that approached significance, $F(2, 145) = 2.947, p = .056$, with a small effect size ($\eta_p^2 = .04$).

Post-hoc comparisons using the LSD test showed two significant differences between Experimental Conditions: the mean SPDS score for the Control Condition ($M = 21.94, SD = 4.21$) was significantly higher than both the mean score for the Biogenetic Condition ($M = 20.12, SD = 4.69$) and the Psychosocial Condition ($M = 19.87, SD = 3.99$). However, there was no significant difference in DCQ scores between the Biogenetic Condition and the Psychosocial Condition. In other words, participants in the Control Condition desired to control people with schizophrenia more than participants in either the Biogenetic or Psychosocial Conditions, but there was no real difference between the participants in these latter two conditions.

Social Distance Scale. The final ANOVA examined whether there were any differences between the Experimental Conditions in terms of participants' SDS scores, while controlling for a single covariate; ethnicity. Ethnicity was confirmed as a significant covariate in this ANOVA, although the amount of variance in SDS scores explained by this variable was found to be negligible ($\eta_p^2 = .01$). After adjusting for ethnicity, this ANOVA revealed a significant difference in SDS scores between Experimental Conditions, $F(2, 146) = 6.03, p = .003$, with a small effect size ($\eta^2 = .08$).

Post-hoc comparisons using the LSD test showed two significant differences between Experimental Conditions: the mean SDS score for the Control Condition ($M = 21.11, SD = 3.78$) was significantly higher than both the mean score for the Biogenetic Condition ($M =$

18.51, $SD = 3.88$) and the Psychosocial Condition ($M = 19.15$, $SD = 4.03$). However, there was no significant difference in SPDS scores between the Biogenetic Condition and the Psychosocial Condition. In other words, participants in the Control Condition desired more social distance from people with schizophrenia than participants in either the Biogenetic or Psychosocial Conditions, but there was no real difference between the participants in these latter two conditions.

Additional Analyses: Testing for Floor Effects

A series of one-sample t-tests were conducted in order to compare the mean scores of the Control Condition with the midpoint of each scale. As Table 1 shows, four of these t-tests revealed a significant difference between the mean score of participants in the Control Condition and the midpoint of the scale. Only the mean SDS score was not significantly different to the midpoint of this scale. This indicates that, participants who were not provided with any causal explanation for schizophrenia had very low levels of stereotypical belief, prejudice, and discriminatory attitudes. Since participants who were not educated on the causes of schizophrenia had significantly positively biased attitudes and opinions, this suggests that there is a possibility of a floor effect occurring.

Table 1

Mean Scores and Standard Deviations of Control Condition Compared to the Midpoints, and Significance Values of One-Sample t-Tests.

Scale	Mean	Std. Dev.	Midpoint	One-sample t-test sig.
SSQ	63.34	7.29	69	$p < .001$
SPDS	25.92	4.59	24	$p = .004$
ERQ	18.53	4.49	27	$p < .001$
DCQ	21.94	4.21	24	$p = .001$
SDS	21.11	3.78	21	$p = .113$

Note. $*p < .05$.

Additional Analyses: Convincingness Ratings

At the end of the experiment, all participants gave a rating of how convinced they were by the ‘textbook excerpt’ that they had read on a ten-point sliding scale. It was expected that participants would rate the new causal explanations on the positive end of this scale.

Across all Experimental Conditions, the mean rating of convincingness was 7.96 out of 10. A one-sample t-test was conducted in order to assess whether this mean was significantly different from the midpoint of the scale. As expected, the result of this t-test showed that the mean convincingness rating was significantly higher than the midpoint of the scale, $t(149) = 23.67, p < .001$. This indicates that, on average, participants in Experiment Two found the causal explanation to be highly convincing.

Convincingness and experimental condition. In order to ascertain if there was a statistically significant difference between the convincingness ratings between conditions (in particular, between the Biogenetic and Psychosocial Conditions) a one-way between-groups ANOVA was conducted with Experimental Condition as the independent variable and controlling for a single covariate: whether participants knew someone with schizophrenia. Levene's test for equality of variances was not violated for the present analysis, $F(2, 147) = .32, p = .730$. Accordingly, ANOVA results were assessed using the standard level of significance ($p < .05$). Whether participants knew someone with schizophrenia was confirmed as a significant covariate by this ANOVA, although this variable only explained three percent of the variance in participants' convincingness ratings. After adjusting for whether participants knew someone with schizophrenia, this ANOVA revealed no significant differences in convincingness ratings between Experimental Conditions, $F(2, 146) = 1.79, p = .170, \eta_p^2 = .02$.

In conclusion, our analyses of participants' ratings of convincingness reveal that participants rated the 'textbook excerpts' as highly convincing; a result that confirms our prediction and substantiates our claim that the causal explanation that participants read in experiment two was strong enough and credible enough to lead participants to believe that causal explanation to be true. In addition, our results show that the excerpts (which were differed between experimental conditions) were rated as equally convincing. In particular, I

was interested in assuring that the biogenetic and psychosocial ‘excerpts’ were rated as equally convincing to ensure that any differences in stigma scores could not be explained as the result of one causal explanation being more convincing than the other. There was no significant difference in the level of convincingness between Experimental Conditions. This indicates that any differences in stigma scores between conditions cannot be explained as the result of differences in convincingness between different causal explanations.

Conclusion

In summary, the results of this second experiment show four key findings. First, participants in both the Psychosocial and Control Conditions were found to have higher levels of stereotypical belief than participants in the Biogenetic Condition, but there was no difference in stereotypical belief between these former two Conditions. This indicates that providing a biogenetic causal explanation reduces the extent to which participants believe stereotypes regarding people with schizophrenia. Second, participants in the Control Condition were found to be more likely to desire control over and distance from individuals with schizophrenia compared to participants in both the Biogenetic and Psychosocial Conditions. However, participants in the Psychosocial Condition were no more likely to desire control over or distance from individuals with schizophrenia compared to participants in the Biogenetic Condition. This result indicates that, while providing a causal explanation appears to decrease desire for control over and desire for social distance from people with schizophrenia compared to providing no causal explanation, it also indicates that one causal explanation is not better than the other. Similarly, a third finding indicates that participants in the Control Condition perceived people with schizophrenia as more dangerous than participants in the Biogenetic Condition. However, there was no difference in perceived dangerousness between the Psychosocial Condition and either the Biogenetic or Control Condition. Again, these findings indicate that one causal explanation is not better than the

other in terms of reducing participants' beliefs that people with schizophrenia are dangerous. Our final finding is that there were no significant differences in prejudicial attitudes between Experimental Conditions, indicating that this element of stigma is unaffected by our experimental manipulation.

Considering the above findings, I can conclude three things. First, that providing an etiological education of any type successfully reduces discrimination (desire to control and desire for social distance). Second, that providing a biogenetic causal explanation, specifically, also successfully reduces perceived dangerousness and belief in other stereotypes. Third, that teaching members of the general public a biogenetic causal model does not increase stigma, as previous authors have claimed (Read & Harré, 2001; Read et al., 2006; Read, 2007). Instead, a biogenetic causal explanation was found to effectively reduce both stereotypical belief and discrimination. Thus, it appears that providing some form of etiological education is an effective method of stigma reduction, and that a biogenetic causal explanation is a more effective stigma reduction tool than a psychosocial explanation as it reduces multiple types of stigma.

Discussion

Experiment Two explored two key research questions. First, I investigated whether teaching participants about the causes of schizophrenia reduces stigma compared to no causal explanation. Second, whether there was any difference in the stigma reduction between a psychosocial explanation and a biogenetic explanation. The results of this experiment showed that providing an etiological explanation reduced both belief in stereotypes and discriminatory attitudes, but not prejudice towards people with schizophrenia. However, there was only minimal evidence of a difference in stigma reduction effectiveness between a biogenetic and psychosocial explanation. Thus, while I conclude that providing some type of explanation about the causes of schizophrenia is an effective means of reducing the stigma

attached to schizophrenia, I cannot conclude that one specific causal explanation reduces stigma more than the other.

When taught about either a biogenetic or psychosocial causal model, participants' desire to control and desire for social distance were both reduced compared to participants who were taught nothing about etiology. In addition, participants who were taught about the biogenetic causes of schizophrenia held significantly less stereotypical beliefs regarding people with schizophrenia and perceived patients as less dangerous than participants who were taught nothing about etiology. Thus, in support of my first prediction, providing an explanation on the causes of schizophrenia did lead to lesser amounts of stereotypical belief and discrimination. However, this prediction was not entirely supported; there were no significant differences in prejudice (as measured by the ERQ) between the three experimental groups indicating that this component of stigma was unaffected by etiological explanation. Nevertheless, these results are in line with previous research which has found evidence that teaching a causal explanation can reduce belief in stereotypes (Boysen & Vogel, 2008; Fisher & Farina, 1979; Lincoln et al., 2008), desire for social distance (Schlier et al., 2014; Walker & Read, 2002), and desire for control (Walker & Read, 2002).

The results of Experiment Two provide only limited support for my second hypothesis that, in a comparison between a biogenetic causal explanation and a psychosocial explanation, one type of causal explanation is more effective at reducing stigma. A significant difference between the Biogenetic and Psychosocial Conditions was only found in one instance - when a biogenetic explanation was found to produce lower levels of stereotypical belief than a psychosocial explanation. However, for all other components of stigma, both causal explanations were equally effective. Interestingly, this finding contradicts several previous studies. For example, Lincoln et al. (2008) found that a psychosocial explanation reduced perceived dangerousness when a biogenetic explanation did not, and a

biogenetic explanation reduced perceived unpredictability when a psychosocial explanation did not. Similarly, Schlier et al. (2014) found that a greater reduction in perceived blame when participants were given a biogenetic causal explanation compared to a psychosocial explanation.

In addition, contrary to my prediction, no significant differences in perceived dangerousness were found between participants taught the biogenetic model and participants taught the psychosocial model. This result also contradicts the results of three previous studies, all of which found evidence that participants provided with a biogenetic explanation would show significantly different levels of perceived dangerousness compared to participants provided a psychosocial explanation (Lincoln et al., 2008; Schlier et al., 2014; Walker & Read, 2002). However, these studies all showed different patterns of results in terms of the directionality of this difference. Walker and Read (2002) found that a biogenetic explanation caused a significant increase in perceived dangerousness while a psychosocial explanation caused no change. In contradiction, Schlier and colleagues (2014) found that a biogenetic explanation caused a significant decrease in perceived dangerousness while a psychosocial explanation caused no change. Furthermore, Lincoln and colleagues (2008) found that a biogenetic explanation caused no change while a psychosocial explanation caused a significant reduction in perceived dangerousness.

In summary, Experiment Two provided evidence in support of my hypothesis that teaching people about the causes of schizophrenia is an effective method of stigma reduction. More specifically, a purely biogenetic causal model will reduce two components of stigma – belief in stereotypes and discriminatory attitudes – while a purely psychosocial causal explanation appears to only lead to a reduction in discrimination. However, there is only limited support for my second hypothesis that, in a comparison between a biogenetic causal explanation and a psychosocial explanation, one type of causal explanation reduces stigma

more than the other. Thus, I must conclude that etiological explanation is an effective means of reducing the stigma associated with schizophrenia but that there is no clear evidence to support the idea that a biogenetic explanation is more effective at reducing stigma than a psychosocial explanation, or vice versa.

Strengths

Large sample sizes in both experiments can be considered as a major strength of the present study. In addition, the utilisation of four questionnaires is a major strength as such a comprehensive measure of stigma allowed for a careful examination of how causal explanations affect each of the three components of stigma. Previous research has so far only examined, at most, two components of stigma at a time. For example, Farina et al. (1978) and Fisher and Farina (1979) only measured stereotypical belief, while Lincoln et al. (2008), Schlier et al. (2014) and Walker and Read (2002) measured discrimination and stereotypical belief. However, since stigma is comprised of three components, it seems prudent that a robustly designed experiment would measure prejudice as well as stereotypical belief and discrimination. Thus, with the inclusion of a measure of prejudice (the ERQ), the methodology of the current study, has significantly improved upon previous research.

Limitations

The participant sample made up entirely of first-year psychology students, is the basis for two key limitations with the present study. First, with a sample comprising individuals with a similar level of education and all of a similar age (the majority of participants were aged 18 to 19), our results may not be generalizable to the general population. Instead our results only generalise to groups of young adults with secondary school and/or university education. In addition, the participant samples were also primarily comprised of people who identified as European meaning that while our sample was representative of a New Zealand population, our results are not globally generalizable.

A lack of generalizability is a limitation that is shared with the previous research in this area. Of the three experimental studies that utilised robust experimental methodology, similar that of the current experiment, only one, Schlier et al.'s (2014) study, recruited a general population sample. Lincoln et al. (2008) recruited medical and psychology students while Walker and Read (2002) recruited undergraduate mathematics students. Thus, it is important that future research in this area should include a wider range of participants in order to ascertain whether the results found in this and similar studies are also found for people of a wide variety of ages, ethnicities, and with different levels of education.

Second, since participants were all psychology students, it is likely that the majority came into the experiments with already favourable attitudes regarding people with schizophrenia which may have produced a floor effect. Alternatively, being psychology students, many participants may have believed that they ought to possess already favourable attitudes and, as a result, may have answered the questionnaires in a socially desirable manner rather than answering in a way that reflected their true attitudes and beliefs. Either way, if the majority of participants responded to the questionnaires in such a way that indicated largely favourable attitudes, there may have been no room for attitudes to improve – a floor effect. A comparison of the mean scores of participants in the Control Condition to the midpoints of each scale was conducted in order to investigate this possibility. These analyses revealed that the mean SSQ, ERQ, DCQ, and SDS scores for participants in the Control Condition were significantly lower than the midpoint of the scales indicating that the participants who were not exposed to any causal explanation held rather favourable attitudes regarding people with schizophrenia. Thus, the possibility that a floor effect is at work must be entertained.

Another limitation of the current study is that, while attitude change could be measured, there was no measure for actual behavioural change. Discrimination is

conceptualised as the behavioural component of stigma but, I assert that this component of stigma is actually made up of two components itself; an action component and an intention-to-act component. In other words, a person can hold discriminatory attitudes which are effectively separate from discriminatory behaviours. It is the difference between the desire to control a patient through involuntary incarceration and actually acting on this attitude and forcing a patient into care. In this study, only discriminatory attitude was measured. Unfortunately, attitudes and behaviours are not always congruent, and so evidence of a reduction in discriminatory attitudes does not necessarily equate to a reduction in discriminatory behaviours. Thus, there is no way of measuring whether the destigmatising effects of causal explanations extend to the practical side of stigma. Future research should rectify this by including a measure of actual behavioural change.

Implications

A key result of the present study is that a causal explanation does not affect all three components of stigma in the same way. For example, this was clearly demonstrated in Experiment Two when the biogenetic causal explanation led to a significant reduction in stereotypical belief and discrimination but not prejudice. This finding leads to an important theoretical implication – that while, the three components of stigma are interrelated (Corrigan & Watson, 2002; Corrigan, 2000), they also appear to be influenced independently of one another.

The practical implications of this relate directly to the question of how to design a successful, efficient destigmatisation program. Knowing that the three elements of stigma are individually influenced means that, in order to effectively reduce all three types of stigma, a program would need to target each component individually. A destigmatisation technique that effectively reduces one component of stigma, may not necessarily reduce the other components of stigma equally as well, or even at all. Thus, a multi-faceted approach

incorporating several different methods of destigmatisation is likely required.

Destigmatisation programs and campaigns must be based on careful consideration of which elements of stigma are to be tackled and then which techniques are the best for that particular case. For example, from the results of the present study, it is clear that a destigmatisation campaign primarily concerned with reducing belief in stereotypes regarding schizophrenia would only need to focus on teaching people about the biogenetic causes of schizophrenia. On the other hand, if the aim of a campaign was to reduce prejudicial attitudes, neither a biogenetic nor a psychosocial model would be effective and it would be appropriate to consider methods of destigmatisation other than etiological explanation. Considering this, it is important that future research further investigates the differential effects of causal explanations as well as other methods of destigmatisation. After all, if a destigmatisation program that successfully and efficiently reduces all components of stigma is to be developed, it is crucial that those implementing the program know exactly how to most successfully target each component.

Future Directions

As discussed above, a successful and efficient destigmatisation program would require a multi-faceted approach that utilises several different methods of destigmatisation targeting each individual component of stigma. In light of this, the primary goal of future research should be to continue utilising robust experimental methodology as we further investigate the differential effects of causal explanations as well as other methods of destigmatisation. Only through further comparisons of various different destigmatisation methods will it be possible to find the most successful and most efficient method for tackling each individual component of stigma. Future research should replicate this experiment and similar experiments to ensure that public campaigns are based on a robust effect that has been thoroughly investigated. Future research should also explore the differential effectiveness of

causal explanations compared to other stigma reduction methods (for example, increasing contact between stigmatiser and stigmatised). It is absolutely crucial that we establish, through careful experimental investigation what the most effective stigma reduction method is. After all, as discussed above, the small effect sizes found in this study indicates that causal explanations only reduce the stigma associated with schizophrenia by a minute amount. It is likely that another method of stigma reduction will be found to have a far greater impact.

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Appendix A

Content-Check Questions for Video Material

The following includes a complete list of all content-check questions that were given to all participants in both experiments in order to assess whether these participants had been paying attention to the content of the three videos. Participants were only able to select one answer for each question. The correct answers for each question are indicated by an asterisk.

1. The interviewer asks Bob about a collection of items in his room. What is this collection that Bob talks to us about?

- Trophies *
- Coins
- Stamps

2. Who did Etta believe was sending her messages?

- Her husband
- Buddha
- Jesus *
- Her mother

3. Gerald talks about some concerns of his. Which of the following does Gerald NOT talk about?

- He is worried people are going to electrocute him and put him in jail
- He is concerned that “that picture has a headache” *
- He is worried about his dog

Appendix B

Written Content: Information about Schizophrenia

The following includes a complete of the three paragraphs of information regarding the symptoms of schizophrenia which all participants read immediately following the content-check questions regarding the video material. All participants in both experiments and across all conditions read this material.

“People who suffer from schizophrenia can experience hallucinations meaning they may hear, smell, see, or feel things that are not there. One of the most common types of hallucinations people with schizophrenia experience is to hear voices like Etta and Gerald did. Quite often, these voices are mean and vicious. These voices might also tell the patient to do things.

Schizophrenia can also cause people to have delusions (false beliefs). For example, some people may become convinced that others are reading their minds, controlling how they think, or plotting against them. As you can imagine, such beliefs can be severely and persistently distressing, making the patient withdrawn and frantic. Remember, Etta thought that Jesus and The Eagle were watching her when she went to post the mail and Gerald thought people were going to kill him or electrocute him.

Another symptom of schizophrenia is disorganised thought and/or disorganised behaviour. You saw an example of this in all three videos but Gerald showed disorganised thought particularly well; recall how he didn’t make sense when he was talking about the sperm and the egg, fusion, and heat abstraction in the mind. Other people who are healthy may find it very

difficult to make sense of what a person with schizophrenia is talking about.”

Appendix C

Experiment One Causal Explanations

The following includes a complete copy of the causal explanations provide to participants in the Biogenetic and Psychosocial Conditions in Experiment One. These causal explanations were displayed to participants immediately following the presentation of the three paragraphs detailing the symptoms of schizophrenia (Appendix B).

Biogenetic Condition Causal Explanation

“Researchers are still investigating the causes of schizophrenia but one popular theory says that an imbalance of the natural chemicals in the brain may play a leading role in the development of schizophrenia. In other words, schizophrenia is simply a medical illness that affects the brain. Similar to how a malfunctioning insulin-production system leads to diabetes, schizophrenia arises when normal brain processes malfunction.

Experts believe that these malfunctions and imbalances in the brain are most likely caused by a person’s genes. In particular, 22 specific genes have been identified as being involved in the development of schizophrenia.

In other words, similar to how the colour of your hair is determined by your genes, a person’s mental health and likelihood of developing schizophrenia is determined by their genes. (Quite often, people with schizophrenia will also have relatives who suffer from the illness or another mental disorder.) This means that people with schizophrenia were, to a certain extent, born that way. These people haven’t done anything wrong that caused them to develop schizophrenia; they were simply born with a different set of genes to the rest of us.”

Psychosocial Condition Causal Explanation

“Researchers are still investigating the causes of schizophrenia but one popular theory says that schizophrenia is caused, at least in part, by the environment. That is, exposure to negative surroundings and life experiences has been linked to the development of schizophrenia.

Some key environmental factors that have been linked to schizophrenia are:

- severe stress or strain (e.g. due to job loss, stressful family life, financial worries, etc.)
- trauma (e.g. sexual abuse or the death of a loved one)
- problematic childhood

Whilst the presence of these factors does not automatically mean that a person will develop schizophrenia, when schizophrenic patients or their families look back in time, they are often able to identify at least one of these factors occurring in the patient’s past.

In other words, schizophrenia is likely caused when a person experiences a series of unfortunate and serious events. These people haven’t done anything wrong that caused them to develop schizophrenia; they have simply had a tough life.”

Appendix D

Stigma-Measuring Scales: Complete List of all Items

The following includes a comprehensive list of all four questionnaires (including one subscale) which were provided to participants in both experiments of this research in order to measure their stigma. Participants responded to each of the 47 items using a five-point Likert scale. In the case of the SSQ, SPDS, ERQ, and the DCQ, the Likert scale ranged from ‘1’ indicating ‘strongly disagree’ to ‘5’ indicating ‘strongly agree’. In the case of the SDS, the Likert scale ranged from ‘1’ indicating ‘‘definitely unhappy’ to ‘5’ indicating ‘definitely happy’.

Schizophrenia Stereotype Questionnaire (SSQ)

This questionnaire measures the extent to which participants subscribe to a range of common stereotypical beliefs regarding individuals with schizophrenia. The SSQ is composed of the following 15 items as well as the Schizophrenia Perceived Dangerousness Subscale (items 16 through 23). A higher score on the SSQ is indicative of a higher level of stereotypical belief. Items four, five, thirteen, fourteen, fifteen, seventeen, and twenty-one of the SSQ are reverse-scored.

1. People with schizophrenia could snap out of it if they wanted to.
2. People with schizophrenia are just acting.
3. Schizophrenia is a sign of personal weakness.
4. Schizophrenia is a real medical illness.
5. Most people with schizophrenia are as safe to be around as the average person.
6. It is best to avoid people with schizophrenia so you don’t become schizophrenic yourself.
7. People with schizophrenia are unpredictable.
8. If I had suffered from schizophrenia at some point in my past, I would not tell anyone.

9. Sometimes people with schizophrenia don't know what is best for them.
10. People with schizophrenia are out-of-control.
11. I would not tell anyone if I was experiencing symptoms of schizophrenia every day in my present life.
12. Quite often, people who suffer from schizophrenia are criminals.
13. People with schizophrenia can still be intelligent .
14. People with schizophrenia can still lead meaningful lives.
15. People with schizophrenia can easily control their behaviour.

Schizophrenic Perceived Dangerousness Subscale (SPDS). This questionnaire measures the extent to which participants subscribe to common stereotypical beliefs specifically regarding the dangerousness of individuals with schizophrenia. A higher score on the SPDS is indicative of a higher level of belief that people with schizophrenia are dangerous.

16. If a group of people with schizophrenia lived nearby, I would not be comfortable with children walking to school alone.
17. If a person with schizophrenia applied for a teaching job at a primary school and they were the best qualified for the job, I would recommend hiring them.
18. One important thing to know about people with schizophrenia is that you cannot tell what they will do from one minute to the next.
19. If I know a person has suffered from schizophrenia, I will be less likely to trust them.
20. The main purpose of mental hospitals should be to protect the public from people with schizophrenia and similar mental disorders.
21. If a person with schizophrenia lived nearby I would feel comfortable letting young children under my care to play on the footpath.
22. Although some people with schizophrenia may seem all right, it is dangerous to forget that they are mentally ill.

23. There should be a law forbidding a person with schizophrenia the right to obtain a firearms (gun) license.

Emotional Reactions Questionnaire (ERQ)

This questionnaire measures the extent to which participants experience prejudicial emotional reactions such as pity, fear, and anger when interacting with or thinking about people with schizophrenia. Items one, four, and eight measure a participant's pity. Items two, six, and nine measure a participant's fear. Items three, five, and seven measure a participant's anger. Items one, four, and eight of the ERQ are reverse-scored. A higher score on the ERQ is indicative of a higher level of prejudice.

1. I feel the need to help people with schizophrenia.
2. When I think about people with schizophrenia, I feel uncomfortable/uneasy.
3. When I think about people with schizophrenia, I feel angry.
4. I feel pity toward people with schizophrenia.
5. I feel annoyed/irritated by people with schizophrenia.
6. People with schizophrenia scare me.
7. I feel like laughing at people with schizophrenia and I sometimes would like to make fun of them.
8. I feel sympathy/empathy for people with schizophrenia.
9. People with schizophrenia make me feel insecure.

Desire to Control Questionnaire (DCQ)

This questionnaire measures both the extent to which participants believe that control should be exerted over people with schizophrenia and the extent to which they wish to exercise that control. A higher DCQ score is indicative of a higher level of belief that people with schizophrenia should be controlled and a higher level of desire to exert that control. Item three of the DCQ is reverse-scored.

1. People with schizophrenia need to be watched closely by doctors, psychiatrists, psychologists, and the police.
2. People with schizophrenia should be hospitalised (even if they don't want to be) if their behaviour becomes too strange.
3. Doctors, psychiatrists, and psychologists should not be able to take away a schizophrenic person's freedom or rights.
4. If a doctor knows it will help them, people with schizophrenia should be given medications even if they don't want it.
5. If a doctor knows it will help them, people with schizophrenia should be made to take treatments (such as counselling or electric shock therapy) even if they don't want it.
6. People with schizophrenia should not be allowed to have children.
7. People with schizophrenia should not be allowed to go out in public.
8. People with schizophrenia should not be allowed to hold normal jobs.

Social Distance Scale (SDS)

This scale assesses the extent to which participants desire social distance from individuals with schizophrenia. All items on the SDS are reverse-scored so that, after manipulation, a higher SDS score is indicative of a higher level of desire for social distance from people with schizophrenia.

1. How would you feel about someone with schizophrenia being your flatmate?
2. How would you feel about working at the same job as a person with schizophrenia?
3. How would you feel about having someone with schizophrenia as a neighbour?
4. How would you feel about having someone with schizophrenia taking care of some children for a couple of hours?
5. How would you feel about a person with schizophrenia marrying someone in your immediate family (brother, sister, parent)?

6. How would you feel about introducing a person with schizophrenia to a friend of yours?
7. How would you feel about recommending a person with schizophrenia for a job working for a friend of yours?

Appendix E

Experiment Two Causal Explanations

The following includes a complete copy of the causal explanations provide to participants in the Biogenetic and Psychosocial Conditions in Experiment Two. These causal explanations were displayed to participants immediately following the presentation of the three paragraphs detailing the symptoms of schizophrenia (Appendix B).

Biogenetic Condition Causal Explanation

Schizophrenia is a severe and disabling brain disease. More specifically, schizophrenia is a biological disorder in which certain genes cause brain abnormalities to develop as a person ages. These brain abnormalities are the cause of the symptoms that were discussed above. Scientists have discovered that there are two basic differences in the brains of patients with schizophrenia compared to non-patients. First, research has found that people with schizophrenia have an **imbalance of certain natural chemicals in the brain**. These naturally occurring chemicals, called neurotransmitters, allow the brain cells to communicate with each other. If there are too many or too few of these neurotransmitters, the brain will not function correctly. For example, schizophrenic patients are known to have abnormal amounts of the neurotransmitter dopamine. Abnormal levels of dopamine are known to be one of the key chemicals responsible for hallucinations in people with schizophrenia.

The second major biological difference is that **some brain structures are actually a different size or shape** in people with schizophrenia compared to people without this disorder. For example, scientists have found differences in the size of a particular set of brain structures called ventricles (the hollow spaces in the middle of the brain which hold cerebrospinal fluid). MRI scans like the ones below show that, compared to a healthy person, the ventricles of a person with schizophrenia are a great deal larger!

But what causes these malfunctions and abnormalities in the schizophrenic brain? Forty years of careful and thorough research has led scientists to conclude that it is a person's genetic makeup that is responsible for the development of schizophrenia. More specifically, there is a collection of **mutated genes** that, when present all at once, work together to cause the brain abnormalities discussed above and the resulting symptoms of schizophrenia. In other words, **schizophrenia is inherited** just like many other traits such as height, eye colour and personality.

The first important piece of evidence for a genetic explanation for schizophrenia comes from research that compares people with schizophrenia to their relatives who don't suffer from the disorder. For example, a famous study conducted in Sweden in 2006 looked at how often the parents or siblings of people with schizophrenia also had schizophrenia themselves. The results of this study (Lichtenstein et al., 2006) found that if a person has a first-degree relative with schizophrenia, they are up to thirteen times more likely to develop schizophrenia themselves than a person who has no relatives with schizophrenia. Similar studies using identical twins have found that if one twin has schizophrenia, there is a 50 to 80% chance that the other twin will also have schizophrenia. In short, these studies and many others like them have allowed the science community to conclude that schizophrenia is the result of genetics. The second piece of evidence for a genetic cause for schizophrenia comes from biogenetic research that has examined the human genome and located the genes that are responsible for schizophrenia. More specifically, it is mutations in these genes which cause schizophrenia. So far, scientists have identified at least **22 mutated genes** that implicated to cause the symptoms of schizophrenia. Researchers believe there are still more genes involved and the search for other mutated genes continues.

In summary, there exists a large collection of scientific research that provides evidence that schizophrenia is a biological disease that affects the brain and that this disease is passed on through genetic inheritance. While there is still a lot of research to be done on schizophrenia, one thing is known for sure: schizophrenia is a medical illness just like any other illness and it is caused by genetic and biological factors.

Psychosocial Condition Causal Explanation

Schizophrenia is a form of mental instability that is caused by negative factors or events in a person's social life. More specifically, the unusual behaviours and thoughts that are experienced by schizophrenic people are caused by exposure to negative events earlier in their lives. These are often extreme events that would cause psychological distress in most individuals but research has also identified that a lower level negative life experience (endured for a prolonged period of time) can also result in the onset of schizophrenia. Over the past seventy years, psychological researchers have carefully examined the differences between schizophrenic people and people with no mental instability. This vast collection of research has allowed us to identify which **negative life events are responsible for causing schizophrenia**.

Researchers have identified two sets of negative life events that can cause schizophrenia in individuals. The first set of negative life events include: emotional, physical, or sexual **abuse in childhood**; encountering intense **trauma**; exposure to **severe and prolonged stress**; or extreme **social isolation**. Extreme events like these cause such a high level of distress to most individuals that it is relatively easy to understand how a schizophrenia can develop as a result.

The second set of negative life experiences are not as obviously life-altering as the first set but when a person is exposed to them over a prolonged period of time they can do just as much harm. Included in this second set of negative life experiences is exposure to ongoing and personalised discrimination (including racism, sexism, and discrimination in relation to sexuality, religion, or class), a harmful relationship with parents or family, being an immigrant, and living in a densely populated urban environment (e.g. a city). When schizophrenic patients or their families look back into the past, they are often able to identify at least one of these factors occurring in the patient's past.

Whilst this second group of negative life experiences may be more difficult to imagine as causes of schizophrenia, there is a great deal of research spread over many decades proving that these factors are just as likely to cause a person to become schizophrenic as a factor from the first list. For example, a breakthrough study in 1999 in Holland confirmed that living in a densely populated urban area (urbanicity) was related to schizophrenia and also that the larger the town or city, the greater the likelihood that an individual would become schizophrenic. It is this last piece of information that proves that **urbanicity and population density are causes** of schizophrenia.

You may be wondering at this point why more people are not schizophrenic. After all, most of us are exposed to stress at one time or another – sometimes for a long period of time – but most of us do not become schizophrenic. Similarly, not everyone that has suffered abuse or witnessed horrific events become schizophrenic either.

So what is it that determines whether or not a negative life experience will cause a person to start thinking and behaving in a schizophrenic manner? Psychological researchers believe that the severity and duration of negative life experiences are the key to this puzzle. Recent

research has shown that the presence of one negative life event is often not enough to cause a person to become schizophrenic but rather it is the influence of several negative life events that cause a person to become mentally unstable. In other words, there is a cumulative effect at work. For example, an individual may have experienced many episodes of extreme stress over a period of many years or they may have witnessed a traumatising event and also have grown up in a densely populated city; in either case, more than one event will have eroded a person's psychological state and, thus, contributed to that person developing the thinking patterns and behaviours of a schizophrenic. On rare occasions, one single life event may be enough to cause a person to become schizophrenic if that event is extreme enough. However, cases like these are the exception rather than the rule.

In summary, there exists a large collection of scientific research that provides evidence that schizophrenia is directly caused by severely negative events in an individual's life. While there is still a lot of research to be done on schizophrenia, one thing is known for sure: it is the social environment in which a person grows up that determines whether or not that person will develop schizophrenic behaviours and thoughts.

Appendix F

Content-Check Questions for Causal Explanations (Experiment Two)

The following includes a complete list of all content-check questions that were given to and participants in the Biogenetic and Psychosocial Conditions in Experiment Two in order to assess whether these participants had been paying attention to the content of the causal explanations. Participants were able to select multiple answers for question one in both conditions and for question four in the Psychosocial Condition. For all other questions, participants could only select one answer. The correct answers for each question are indicated by an asterisk.

Biogenetic Condition

1. Schizophrenia is characterised by a few key symptoms. Tick all of the symptoms that were discussed in the textbook excerpt:

- Hallucinations (e.g. seeing things that aren't there) *
- Sadness
- Delusions (false beliefs) *
- Multiple personalities
- Disorganised thought and disorganised behaviour *
- Insomnia

2. Two differences between the brains of people with schizophrenia and non-sufferers were mentioned. Which of the following was NOT discussed?

- People with schizophrenia have an imbalance in the natural chemicals in the brain
- People with schizophrenia have no frontal lobe *
- People with schizophrenia often have larger ventricles than non-sufferers

3. What was discussed as the cause of the malfunctions and abnormalities in the brains of people with schizophrenia?

- Child abuse and/or stress
- Mutated and inherited genes *
- Illicit drugs
- None of the above

4. Two pieces of evidence for a genetic cause of schizophrenia were discussed in the textbook excerpt. Which of the following was NOT discussed?

- Studies using identical twins
- Genome research (genetics)
- Research using rats and mice *

Psychosocial Condition

1. Schizophrenia is characterised by a few key traits. Tick all of the characteristics that were discussed in the textbook excerpt:

- Hallucinations (e.g. seeing things that aren't there) *
- Sadness
- Delusions (false beliefs) *
- Multiple personalities
- Disorganised thought and disorganised behaviour *
- Insomnia

2. Negative life events are responsible for causing. Which of the following was NOT discussed as a negative life event that has been proven to cause schizophrenia?

- Trauma
- Severe stress
- Not enough friends *

- Discrimination (e.g. racism)
- Being an immigrant

3. The textbook excerpt discussed a Dutch study that researched the link between schizophrenia and living in a densely populated urban area. According to the result of this study, which of the following people is most likely to become schizophrenic?

- Someone who grew up in a small rural town
- Someone who grew up in a mid-size town
- Someone who grew up in a very large and very crowded city *

4. Which of the following factors are important to consider when thinking about how and when negative life events cause schizophrenia? (You can select more than one).

- Severity of the event (e.g. how intense, serious and emotional the event was) *
- Who else was involved in the negative life event
- At what age the event occurred
- Duration of the event (e.g. how long the event lasted for or how often it occurred) *