Reducing Stigma Toward Schizophrenia: An Investigation into what Information is

Most Effective at Decreasing Negative Attitudes to Schizophrenia

Author: Danielle Hayward

Supervisor: Dr John McDowall

School of Psychology, Victoria University of Wellington

A thesis

Submitted to the Victoria University of Wellington

in fulfilment of the requirements for the degree of

Master of Science in Psychology.

Victoria University of Wellington

2019

Table of Contents

Acknowledgements

This thesis would not have been possible without the valuable input and support of my supervisor Dr. John McDowall. Further, I would like to thank my mum and dad, Donna and Brian for their continued support and love during my time at university.

Abstract

Schizophrenia is a chronic mental illness that manifests psychotic symptoms and largely affects an individual's day to day functioning (Silva et al., 2017). In addition to the incapacitating symptoms of this disorder, patients with schizophrenia face another central concern: stigma (Stuart, 2016). In light of this, an abundance of previous research has been dedicated to discerning the most effective and feasible methods to reduce stigma towards mental illness (Corrigan, 2001). In particular, a large body of research has suggested that education - or more specifically, educating people about the causes of schizophrenia - may be an effective way to achieve this goal (e.g. Boysen & Vogel, 2008). So far, two causal explanations have dominated the literature; psychosocial causal explanations and biogenetic causal explanations. However, only a small number of experimental studies have directly compared the teaching of these opposing two models on levels of stigma (Lincoln, Arens, Berger, & Rief, 2008; Schlier, Schmick, & Lincoln, 2014; Walker & Read, 2002). The findings from these studies show that the effects of causal explanations on stigma are contradictory, thus highlighting the need for another experiment to discern the actual successfulness of these methods at reducing negative attitudes towards schizophrenia. Additionally, due to the mixed findings in the literature regarding the effectiveness of etiological information at lowering stigma, it seems warranted that further exploration into novel, educational teachings is conducted to establish whether causal information really is the most appropriate educational explanation to enlist if stigma reduction is the end goal. In the current research, two experiments were conducted where participants were provided different explanations for schizophrenia (both causal and non-causal in nature) or no explanation at all. Participants received either a biogenetic causal explanation of schizophrenia, a psychosocial causal explanation of schizophrenia, or a creative explanation for schizophrenia, (Experiment One). Comparatively, in Experiment Two participants were provided either a causal

explanation for schizophrenia (biogenetic, psychosocial, epigenetic) or, no information at all. Findings from both experiments suggested there were no significant differences between the levels of prejudice and discrimination of participants who saw information which was causal in nature, and those who did not. Further, no evidence was found to support the hypothesis that different causal explanations have varying effects on stigma. Moreover, the previously untested explanations for schizophrenia did not produce stigma reducing effects. Strengths, limitations, implications and future directions are discussed.

Reducing Stigma: An Investigation into what Information is Most Effective at

Decreasing Negative Attitudes toward Schizophrenia

Schizophrenia is a debilitating and taxing mental illness which encompasses a wide

range of cognitive, behavioural, and emotional symptoms. The World Health Organization

(WHO), labels schizophrenia as one of the most challenging diseases present in today's

society, affecting an estimated 1% of the population globally, or in other figures, 24 million

individuals (Maruta & Matsumoto, 2018). Positioned among the top 25 causes of disability

worldwide in 2013, it is the most prevalent psychotic disorder internationally (Huang, Amos,

Joshi, Wang & Nash, 2018).

According to the fifth edition of the Diagnostic and Statistical Manual of Mental

Disorders (DSM-5; American Psychiatric Association, 2013) the pathology observed in

patients with schizophrenia involves three core disabilities: positive symptoms, negative

symptoms, and cognitive dysfunctions. Among the primary symptoms of schizophrenia, the

positive manifestations tend to stand out (Roth, Lubin, Sodhi & Kleinman, 2009). Ordinarily

termed 'psychotic symptoms', these include; hallucinations, delusions, and thought disorders,

and often illustrate the patients loss of contact with reality. In addition, negative symptoms

(e.g. apathy, inappropriate mood and poverty of speech) and cognitive impairment (e.g.

impaired working memory and conceptual disorganization) are also key features of this

illness (Keefe, Silva, Perkins & Lieberman, 1999). Taken together, these symptoms can often

render patients difficult to hire and socially secluded. Although the symptoms of

schizophrenia can be severely incapacitating, there are several efficient treatment avenues -

often involving a combination of therapy sessions and antipsychotic medications - which

allow the majority of patients to effectively manage their symptoms, and lead a productive

life (Galletly et al., 2016).

Despite the development in the quality and effectiveness of mental health treatments and facilities over the past few decades, patients afflicted with schizophrenia face another central concern: stigma (Gaebel & Kerst, 2018). In terms of mental illness, stigma was originally defined as, "an attribute that extensively discredits an individual from a whole and usual person, to a discounted one" (Goffman, 1963, p.3), a statement that rings true for those with schizophrenia who are often negatively labelled and judged as a result of their mental diagnosis. Comparably, Crocker and colleagues (1998) suggested that, 'stigmatization occurs when an individual possesses (or is believed to possess) some kind of 'mark' or attribute which conveys a specific social identity that is devalued and disparaged in a certain social setting' (p.505). These 'social marks' can be manageable or unmanageable, detectable or undetectable and potentially related to appearance (e.g. physical), group membership (e.g. Maori) or behaviour (e.g. murderer)(Major & O'Brien, 2005).

It is important to note however, that exact definitions of stigma are still being debated - that is, there is no 'one size fits all' universal description available (Carter, Read, Pyle & Morrison, 2018). This is perhaps unsurprising as a plethora of research has recognised that stigma is both culture and context specific (as different societies select different human qualities to stigmatise based on social relevance), therefore it would make sense that it is conceptualised in various ways, depending on which discipline is defining it (e.g. politics, history, psychology) (Rüsch et al., 2005). In relation to the current investigation then, stigma can be defined as a 'socially generated mark', possessed by individuals showing symptoms of, or suffering from schizophrenia which ultimately leads to the mistreatment and degradation of these patients by healthy (non-diagnosed) individuals (Major & O'Brien, 2005).

Whilst the stigmatisation of individuals with mental illness is widespread (Dalky, 2012), it appears the amount of stigma experienced may differ according to diagnosis, with

patients suffering from schizophrenia, particularly susceptible to its effects (Shrivastava, Johnston & Bureau, 2012). As a difficult and persistent disorder, schizophrenia often causes noticeable disability and poor quality of life (Tandon, Nasrallah & Keshavan, 2009). These factors often contribute to the severity of stigma, indicating that the experience of this illness is not simply confined to the symptoms of the disease, but also, the numerous predominant negative societal attitudes which accompany it (Singh, Mattoo & Grover, 2016). Subjective accounts of individuals affected by schizophrenia show that the effects of stigma are often more upsetting and oppressive than the primary condition itself (Koschorke et al., 2014). Research has indicated that no other psychiatric diagnosis shares the same levels of persistent negative stigma as schizophrenia (Corrigan & Blink, 2016; Peluso & Blay, 2011; Schulze & Angermeyer, 2003).

Further, supporting literature suggests that schizophrenia is the most negatively viewed mental illness, resulting in a higher degree of stigma when compared to individuals with disorders such as depression (Mann & Himelein, 2004), anorexia nervosa (Corrigan et al., 2002) and bipolar disorder (Durand-Zaleski et al., 2012; Link, Phelan, Bresnahan, Stueve & Pescosolido, 1999). Additional research has reported that almost half of all patients with schizophrenia report experiencing considerable levels of stigma and two-thirds report perceived discrimination as a result of their mental illness (Brain et al., 2014).

For individuals with schizophrenia the experience of stigma pervades every aspect of life, including work and social interactions. Moreover, it negatively effects the motivation to cope with the disorder effectively, and subsequently adhere to treatment (Lincoln et al., 2007). Unfortunately, for a large number of individuals with schizophrenia, the stigma experienced in connection with their illness is a problem which appears more burdensome to overcome than the management of symptoms (Thornicroft et al., 2016).

Taking the above information into account, it is safe to presume that stigma has a considerable impact on the lives of individuals suffering from schizophrenia. However, what exactly are the mechanisms of stigma? What processes are involved in stigma to produce such detrimental effects on these people? The next section will explore this further;

Dimensions of Stigma

Stigma is a broad and multifaceted concept, and a large volume of work has attempted to examine the underlying elements that contribute to it (Ye et al., 2016). The stigma of mental illness has been largely differentiated into two groups; public (or social stigma) and self-stigma (or internalized stigma)(Corrigan, 2004; Rüsch, Angermeyer & Corrigan, 2005). Public stigma, which is the focus of this research, comprises negative stereotypes and prejudices endorsed by the general population which manifests into discrimination toward individuals with a mental disorder (Singh, Mattoo & Grover, 2017). On the other hand, self-stigma refers to individuals with a mental illness who internalize perceived prejudices, subsequently developing negative feelings about themselves and eventually reducing their self-esteem (Corrigan & Rao, 2012). Whilst self-stigma can be considered closely related to public stigma - the obvious difference is that public stigma operates on a group level, involving attitudes which specific groups hold about others, whereas self-stigma refers to an individual experience (Livingston & Boyd, 2010).

Consistent with a social psychology model, public stigma toward mental illness has been conceptualized as a problem containing three components: stereotypes (or stereotypical belief); prejudice (emotional reactions); and discrimination (Corrigan, 2000; Corrigan & Watson, 2002; Thornicroft, Rose, Kasaam & Sartorius, 2007). Essentially this means that stigma can be seen as an overarching issue containing three primary concepts: problems of knowledge (stereotypical belief), which is the cognitive component; problems of attitude (prejudice) which is the emotional element; and problems of behaviour (discrimination)

which is the behavioural component (Rössler, 2014). The three mechanisms of stigma should be seen as being arranged in a logical order, beginning with stereotypes and ending with discrimination (Angermeyer & Matschinger, 2004).

Stereotypes

The first element of this social psychology model is stereotyping (or stereotypical belief), the cognitive component of stigma. A stereotype in this instance can be defined as 'an over-generalized belief about a particular category of people, often based on incomplete or inaccurate information' (Cardwell, 1999; Corrigan, Larson & Rüsch, 2009). Stereotypes can include beliefs regarding the behaviour of someone with a mental illness, for instance, 'everyone with schizophrenia is violent and dangerous' and beliefs about the causes of the mental health problem (e.g. due to an individual's actions or inactions).

Researchers view stereotypes as a kind of 'knowledge structure' which is adopted by the majority of members in one social group about members of a different social group. These stereotypes are thought to be 'social' in nature as they signify collectively agreed upon notions regarding a specific group of individuals (Corrigan & Watson, 2002). Although we recognise the first contributing element of stigma as stereotypes (or stereotypical belief) stereotypes are not necessarily a bad thing, in fact they can be helpful by allowing us to make rapid judgements about groups who share specific traits. Stereotypes thereby enable us to react or adapt to situations quickly, without requiring additional information about the person/s involved (Corrigan & Watson, 2002).

Although a large number of individuals may possess the knowledge about a certain set of stereotypes, this does not necessarily mean they endorse them. That is, the activation of stereotypes is something that as humans, we cannot fully control, it is a form of subconscious action if you will – whereby, a person may apply a negative stereotype to someone unintentionally, despite that individual actually expressing positive feelings towards that

group (DeVine & Sharp, 2009). For instance, many individuals can elicit specific stereotypes

regarding gender (e.g. men are strong and should be the bread winner) however, the

knowledge of these stereotypes does not necessarily mean that individual believes they are

correct. This is where the second component of stigma, prejudice, is introduced (Corrigan,

2002).

Prejudice

Individuals who are prejudiced endorse these harsh stereotypes ("Yes! Everybody

with schizophrenia is aggressive and dangerous."), and as a consequence, produce negative

emotional reactions toward the specified group (e.g. "Everyone with schizophrenia scares

me," or, "I don't want to be around anyone with schizophrenia")(Corrigan & Blink, 2016).

Allport (1954), defines prejudice as, 'an antipathy based on faulty and inflexible

generalisation. It may be felt or expressed. It may be directed toward a group as a whole, or

toward an individual because he is a member of that group" (p.9). Most researchers have

continued to follow this same trend, frequently defining prejudice as a negative outgroup

attitude.

In comparison to stereotypes (which are beliefs) prejudice is principally a cognitive

and emotional response (Corrigan & Watson, 2002). That is, prejudice commonly involves an

evaluative (often harmful) component that may also yield an emotional response (e.g. anger,

fear) toward the stigmatized group (Corrigan & Watson, 2002). Prejudice can be

demonstrated in various ways, however, in terms of mental illness, the degree of willingness

a non-stigmatised individual expresses to engage socially with a stigmatised individual (e.g.

prepared to live next door to, work with, or befriend) is generally a good indicator of the level

of prejudice held (Pescosolido et al., 2010). Essentially, prejudice leads to discrimination -

the behavioural aspect of stigma (Collins, Wong, Cerully, Schultz & Eberhart, 2013).

Discrimination

The third component of this stigma model is discrimination. Whilst prejudice is an attitude or belief, discrimination is essentially a behavioural reaction to those beliefs (e.g. you feel anger towards people with schizophrenia and therefore you physically harm them) and is presumed to result from prejudice and/or stereotypes (Corrigan, 2004). This component often predicts some element of social exclusion, or negative social interaction with the stigmatised individual (Collins et al., 2013).

In relation to public stigma, three components of discrimination have been identified: coercion (authorities making a patient's decision as they believe the individual is incapable of doing so); loss of opportunities (e.g. employment and housing opportunities); and segregation (avoidance of and/or desire for social distance). This chain of stereotypes, prejudice, and discrimination make up public stigma, the way in which society in general perceives and reacts to individuals with a serious mental disorder, such as schizophrenia (Corrigan, 2007).

Ways People with Schizophrenia are Stigmatised

Whilst the aforementioned research breaks down the individual components of stigma, for the purposes of the current investigation (which is solely focused around schizophrenia) it is important to recognise the specific ways in which individuals with schizophrenia are impacted by these three facets of stigma. That is, while it is widely recognized that stigma constitutes a worldwide phenomenon, expressions of stigma and the actual experience of stigma are varied, manifesting in different ways according to who the stigmatiser, and the subject of the stigmatisation is (Koschorke et al., 2014). For example, there are a range of potential stereotypes to believe, numerous prejudices one can hold, and various discriminatory behaviours a 'healthy' individual may show towards someone with an illness, specifically schizophrenia (Corbiere, Samson, Villotti and Pelletier, 2012). For the purposes of the current experiment then, it is important to recognise the main factors that comprise each element of the stigma model, in relation to schizophrenia, in order to more

wholly understand how each component of stigma interacts to constitute an overall negative experience for the stigmatised individual.

Stereotypes Towards Individuals with Schizophrenia

Various research has shown that levels of stigma linked to mental illnesses such as depression and bulimia have been on a steady decline since the 1950's (Durand-Zaleski, Scott, Rouillon & Leboyer, 2012). Conversely, stigma levels associated with psychotic disorders such as schizophrenia, appear to be increasing (Link, Phelan & Collins, 2004). Whilst the reasons are unclear, it has been proposed that unfavourable (and often inaccurate) stereotypes of individuals with schizophrenia have become more common, rather than less, therefore suggesting this could be a contributing factor (Durand-Zaleski et al., 2012).

Individuals diagnosed with schizophrenia often encounter a range of stereotypes which are serious misrepresentations of their actually identities. For example, cross-cultural findings show, that incompetence, unpredictability and dangerousness are three of the most commonly endorsed traits to describe those with schizophrenia (Angermeyer & Matschinger, 2003; Durand-Zaleski et al., 2012; Morgan et al., 2018; Lyndon et al., 2016; Wiesjahn et al., 2014) despite the statistics suggesting these stereotypes are largely inaccurate. A plethora of research regarding the link between schizophrenia and dangerousness reinforces this statement: For example, in a study of 500 individuals, 74.2% reported that individuals with schizophrenia were potentially dangerous to those around them (Blay & Peluso, 2010). Consistent with this trend, in a survey of over 1,700 individuals, Crisp and Colleagues (2000) reported that the most commonly held belief amongst participants in their study was that individuals with schizophrenia are particularly dangerous when compared with other disorders. (Crisp, Gelder, Rix, Meltzer & Rowlands, 2000).

In terms of unpredictability, a study of medical students found that 71-89% of respondents believed people with schizophrenia were unpredictable, as individuals who will

most likely deal with clients who have a mental illness at some point in their career, this is obviously a worrying statistic (Magliano, Read & Marassi, 2011a). In direct comparison to the current research, a more recent study by Magliano and Colleagues (2016) conducted on psychology students reported that 59% of the sample considered individuals with psychotic symptoms to be both dangerous and unpredictable. This number was even higher when schizophrenia was mentioned to participants in the study.

Additional research on stereotypes reflects that the public believe individuals with schizophrenia are violent (Morgan et al., 2018; Reavley & Jorm, 2011) and dependant on others (Angermeyer & Matschinger, 2003; Van Dorn et al., 2005). For example, in a large nationally representative sample of individuals living in America, 61% believed that patients with schizophrenia were 'very likely' or 'somewhat likely' to act violently towards others, whereas only one third of those same respondents endorsed the same belief about individuals with depression (Pescosolido, Monahan, Link, Stueve & Kikuzawa, 1999).

Clearly, a substantial proportion of the general public believe that those with a mental illness are both violent and dangerous. However, it would appear these fears (and subsequent stereotypes) are largely unwarranted and exaggerated, as opposing evidence indicates that in comparison to those diagnosed with affective disorders, substance use disorders, and personality disorders, those with a diagnosis of schizophrenia represent a lower risk for violent behaviour overall (Monahan & Appelbaum, 2000). It has been proposed that the knowledge of these negative stereotypes - particularly those regarding dangerousness and violence - have been perpetuated by incorrect and misleading media sources (Bowman & West, 2018).

While mental illness is commonly portrayed in an adverse light through the media, whether it be in television shows (Parrot & Parrott, 2015) or movies (Byrd & Elliot, 1985) this stigmatizing depiction seems particularly true of schizophrenia, where both entertainment

(Owen, 2012) and news outlets (Gwarjanski & Parrott, 2017) persistently associate the illness with violent situations. Although schizophrenia is linked with violent behaviour in the literature, consistent with the aforementioned study, research suggests that individuals with schizophrenia are actually far more likely to be a victim of violence, as opposed to a perpetrator of it (Brekke, Prindle, Bae & Long, 2001). Persons with schizophrenia are also more likely to be a victim of violence than someone from the general population, without a mental illness (Walsh, Scott & McKenzie, 2003) suggesting this stereotype (much like the others) is rather deceiving.

A study by Penn, Kohlmaier and Corrigan (2000) revealed that strange was another common stereotype for individuals with schizophrenia, this factor best predicted high levels of social distance - a clear sign of discrimination - when compared with other stereotypes (Lee et al., 2005).

Emotional Reactions Towards Individuals with Schizophrenia

Whilst it is clear that many individuals possess knowledge of negative stereotypes towards individuals with mental disorders, as we discussed earlier, stereotypes or stereotypical beliefs, are not intrinsically a bad thing. They can in fact be an effective method of structuring knowledge as they create mental shortcuts, therefore helping us retain information with minimal effort (Corrigan, 2007). The real issues arise when the general public believe these imprecise, undesirable stereotypes, and further incorporate them into their belief systems, a concept known as prejudice. As discussed previously, prejudice is the endorsement of specific stereotypes AND the subsequent emotional reaction formed in response to these stereotypes (Corrigan, 2002). While there are a wide range of feelings people may experience when confronted by someone with a mental illness, according to research by Angermeyer and Matschinger (1997) and Corrigan (2000), there are three

primary emotional reactions: pity, anger and fear. Each of these reactions is linked to a specific stereotype regarding people with mental disorders (Corrigan, 2000).

Fear, the first emotional reaction, appears to be the most common prejudicial reaction towards individuals with schizophrenia. This statement is somewhat corroborated by Durand-Zaleski et al., (2012) who found that, in comparison to bipolar disorder and autism, schizophrenia generated the greatest amount of fear (65%) and distrust (31%) among survey respondents (Durand-Zaleski et al., 2012). Feelings of fear are thought to occur when someone endorses the stereotype that people with mental disorders are dangerous (Corrigan, 2000). This adverse emotional response is at least partially attributable to sensationalist media reporting, which as discussed above, often exaggerates the link between stereotypes such as dangerousness and schizophrenia (Angermeyer, Holzinger & Matschinger, 2010).

Fear in turn yields avoidant behaviours, or a strong preference for social distance. This too is supported by other research showing that perceptions of dangerousness increases the tendency to distance oneself from people with mental disorders (Angermeyer & Matschinger, 1997; Madianos, Madianou, Vlachonikolis & Stefanis 1987). Public fear (resulting from prejudice) reportedly remains a major obstacle to the social integration of patients and the adequate utilisation of effective treatment, two key components which can enhance the quality of life for a person living with mental illness.

Comparable to fear, anger - the second primary emotional reaction to people with mental illness - is said to originate from the belief that individuals with schizophrenia are dangerous. However, this belief is facilitated by another belief - that people with mental illness are in control of their symptoms (Corrigan, 2000). Therefore, in contrast to the fear reaction which is a primary result of the belief that people with mental illness are dangerous, anger results from the belief that these people are dangerous, however, they choose to be this way because they are in control of their behaviour (Corrigan, 2000). Unsurprisingly, much

like fear, public feelings of anger often lead to a range of discriminatory behaviours such as increased social distance (Angermeyer and Matschinger, 2003), coercion, and reluctance to help (Corrigan & Blink, 2016).

The third emotional reaction is pity. Pity is said to result from the belief that people with mental illness are unable to adequately care for themselves. Unlike the former two reactions, which often lead to discriminatory outcomes, pity can lead to positive actions being shown towards the stigmatised individual (Angermeyer & Matschinger, 2007).

Although the literature identifies three main emotional reactions towards individuals with mental illness that is not to say people's feelings are confined to these categories. Rather, there are a range of prejudices one may hold, which do not necessarily have to fit under the umbrella of these terms. For example, Thornicroft and colleagues (2007) note that emotional reactions such as anxiety, resentment, hostility, distaste and disgust are also relatively common when people are asked to describe their feelings towards schizophrenia.

While the above terms are essentially emotional reactions towards people with schizophrenia, one can imagine if an individual has these hostile feelings towards a certain group, their subsequent interactions with these people will presumably be rather negative. This is where potentially the most damaging aspect of this model – discrimination, comes into play.

Discrimination Towards People With Schizophrenia

People with schizophrenia report high rates of discrimination from members of the general community (Morgan, Reavley, Ross, San Too & Jorm, 2018). It is also increasingly common for these same individuals to describe hostile interactions with employers, landlords, the police, and even potential friends. It appears then, that perhaps the most recognisable form of stigma experienced by individuals with schizophrenia is discrimination - the behavioural component. Discrimination generally takes a punitive form and can be expressed

in many ways, although restricting access to certain opportunities and reacting adversely to the stigmatised group are two of the more common discriminatory behaviours (Corrigan & Blink, 2016). This component is perhaps the most visible in relation to the stigma model, and often the most detrimental to the stigmatised individual (Rössler, 2016).

As predicted for such a multifaceted phenomenon, the impact of experienced and anticipated (self-stigma) discrimination in combination is devastating, leading to adverse outcomes in many aspects of life including; the establishment of friendships, personal relationships and subsequent social interactions, access to adequate healthcare and sufficient housing (Gronholm, Henderson, Deb & Thornicroft, 2017), and reduced education and employment opportunities (Thornicroft et al., 2009), with many individuals experiencing social and economic hardship as a direct consequence of their disorder.

More specifically, schizophrenia can have a huge impact on an individual's social functioning, particularly the maintenance and formation of relationships. That is, discrimination (in this case) often involves some element of social exclusion - whereby, persons who are mentally ill, find it increasingly hard to form and maintain a relationship with others - whether this be family members, friends, prospective employers or colleagues. This is often due to an individual's misconceptions towards the stigmatised individuals illness and subsequent unwillingness to socialise or be around them (Singh et al., 2016).

This kind of behaviour is often referred to as a 'desire for social distance'. That is, the reluctance to engage in multiple forms of contact with an individual due to their mental health status, discriminatory attitudes towards individuals with a mental illness are commonly measured using this term (Angermeyer, Holzinger, Carter & Schomerus, 2011). This notion is prevalent in a large amount of research, for example, in a survey conducted in 1999, 61% of respondents indicated a preference for maintaining social distance from individuals with schizophrenia (Pescosolido et al., 1999).

As briefly discussed above, this connection between the desire for social distance and schizophrenia has predominantly been credited to a public misconception of danger - and the subsequent fear of potential violence - as opposed to the actual symptoms of the disorder (Van Dorn et al., 2005). However, regardless of the reasoning it continues to impact these individuals' everyday lives.

In addition to social exclusion, discrimination and prejudice also impact an individual's employment opportunities, where chronic unemployment is a problem many individuals with schizophrenia face. There is an abundance of evidence which suggests that persons with an illness, such as schizophrenia constantly miss out on prospective job opportunities purely because they have a mental disorder (Corrigan & Blink 2016; Morgan et al., 2018). Data taken from an Australian study emphasises this discrimination in the workforce, where it was reported that only 21.5% of people with a mental illness were employed compared to a rate of 74.2% in the general population (Morgan et al., 2012). Further, when interviewing prospective employers, Manning and White (1995) reported that a mental disorder was either 'usually' or 'always' considered a factor when making a decision to hire new staff. Avoidance by employers is particularly worrisome given the rehabilitation goals of many individuals with mental illness involves successfully securing a job (Corrigan & Blink, 2016). In direct relation to schizophrenia, Lee, Chiu, Tsang, Chui and Kleinman (2006) revealed that despite a similar level of education, patients with this illness were more likely to be unemployed and earn a low income compared to individuals with a physical illness (Lee, Chiu, Tsang, Chui & Kleinman, 2006). Despite the obvious financial repercussions of this, a patient's inability to secure a job can be detrimental to their mental health, often leaving them feeling inadequate and worthless.

Amongst the struggle of finding and securing employment, patients with schizophrenia often have immense difficulty acquiring long-term accommodation, where they

are consistently discriminated against by homeowners and property managers who are resistant to let their houses out to those with mental disorders (Kinoshita et al., 2013). This idea is highlighted through the higher rates of homelessness among the mentally ill population in Australia (5.2%) versus the general public (0.5%)(Morgan et al., 2012).

Further studies show that individuals with schizophrenia are often unfairly targeted by police, falsely accused, and subsequently charged for violent crimes due to the belief that these patients are all dangerous and violent criminals (Kinoshita et al., 2013). It is evident that discrimination pervades almost all aspects of life for persons with schizophrenia. Unfortunately, this discrimination is not without dire consequences for the well-being and social lives of the mentally disordered individual. Arguably, discrimination is the biggest obstacle to the provision of mental health care, a crucial factor for an enhanced quality of life in patients with schizophrenia (Beldie et al., 2012).

Research shows that healthcare workers can also be agents of stigma (Hansson, Jormfeldt, Svedberg & Svensson, 2013) despite increased contact and knowledge with this group. For example, data taken from 27 countries regarding discrimination toward schizophrenia reported that 39% of individuals with this illness felt they had been belittled by mental health staff (Thornicroft et al., 2009). The mere recognition (by the disordered individual) of these discriminating attitudes appears to be an issue in itself, whereby the experience of stigma then produces self-stigma in the disordered individual, which ultimately acts as a barrier against positive future actions and subsequently effects the overall course of the illness (Lannin, Vogel, Brenner, Abraham & Heath, 2015).

There are numerous negative consequences in regards to the mental health status of patients with schizophrenia which can occur as a result of discriminatory actions and prejudiced beliefs. For example, studies indicate that discrimination against individuals with a severe mental illness are linked to a multitude of negative outcomes including, decreased

self-esteem and hope (Corrigan et al., 2012) diminished quality of life and social withdrawal (Link et al., 1997; Thornicroft et al., 2009). Further research has found that both self-esteem and social adaptation are negatively impacted by social exclusion due to discrimination (Link, Struening, Neese-Todd, Asmussen, & Phelan, 2001).

Regardless of the actual prejudice and discrimination experienced by patients, the higher the perceived stigma (when stigmatized persons internalize perceived prejudices and develop negative feelings about themselves) the poorer the quality of life. Higher levels of alleged stigma can again result in dire consequences for the individual with schizophrenia, whereby they tend to avoid contact with the public and are unwilling to seek appropriate treatment for fear of marginalisation (Eisenberg, Downs, Golberstein & Zivin, 2009).

In reference to the aforementioned point, research contends that discrimination due to stigma is a critical contributing factor to a patients non-adherence to treatment (Brain et al., 2014). As one may predict, non-adherence to treatment can lead to various adverse outcomes such as; increased hospitalisation, higher costs for health care, progressive brain damage and higher rates of suicide (Brain et al., 2014). Further, medical adherence is crucial for persons suffering from schizophrenia, whereby the reduction of symptoms - which in turn prevents relapse - enables them to achieve a somewhat desirable standard of socialisation and living (Wang et al., 2018). International recommendations advise long term treatment with antipsychotics to counteract schizophrenia and improve overall well-being, thus highlighting the importance of finding methods to combat stigma (Brain et al., 2014).

In addition to non-adherence, postponement of treatment or help-seeking behaviour is a further undeniable consequence of stigma (or perceived stigma). The WHO (2001) cite stigma as a key barrier to successful treatment engagement, including seeking and maintaining participation in health services and treatment. In Hong Kong, stigma could explain an average postponement of two years from the onset of symptoms before the

majority of individuals experiencing psychosis entered psychiatric treatment or evaluation (Ho & Andreasen, 2001). This delay was commonly attributed to the patient wanting to avoid the stigma which all too often comes with a mental diagnosis (Ho & Andreasen, 2001). This has a major flow-on effect whereby, a longer duration of untreated illnesses - such as schizophrenia - is associated with a lengthier period to symptom remission once the treatment has been initiated, a reduced degree of recovery, greater probability of relapse and a poorer overall prognosis (Franz et al., 2010). It has been estimated that less than 40 percent of individuals with severe mental illnesses receive consistent mental health treatment throughout the year, potentially due to the stigma involved (Kessler et al., 2001). Research by Clement and colleagues (2015) reviewed 144 studies (90,189 participants) and found a consistent negative relationship between perceived mental illness stigma and help-seeking behaviour among people with mental disorders (Clement et al., 2015).

Concealment (attempting to hide a mental diagnosis from those around you) is another issue resulting largely from stigma (Lee et al., 2006). This action is often adopted by individuals with schizophrenia as a strategy of self-protection, in the hope that hiding their disorder may reduce the marginalisation towards them. Unfortunately, concealment has been found to be extremely painful, whereby the stigmatised individual is constantly internalising their negative emotions, thereby effecting overall quality of life.

Whilst stereotypes, prejudice and discrimination have an indisputably negative effect on the patient, family members of individuals with a mental illness are likely to testify that the experience of stigma also effects their self-esteem and ability to maintain friendships, whereby people constantly judge the mentally fit individual based on their interactions with somebody who is mentally unwell (Wahl & Harman, 1989). This is commonly referred to as 'courtesy stigma' and can lead to feelings of shame and embarrassment among those who associate with the stigmatised individual (Phelan, Link & Dovidio, 2008). Thus, the

experience of stigma is not simply confined to the ill individual - but also extends to members of their broader social network. This further highlights the need for an effective and efficient de-stigmatization intervention.

Sociodemographic Factors which Perpetuate or Diminish Stigma toward Mental Illness.

While it is clear that stigma is a pervasive and widespread issue, there are certain sociodemographic factors which appear to enhance or diminish an individual's level of stigma. For example, Addison and Thorpe (2004) discovered that in comparison to law students, counselling pupils were more accurate in their knowledge towards mental illness. This finding correlated to lower levels of stigma in the study population - indicating that knowledge towards mental illness may lead to a decrease in stigmatizing attitudes and behaviours (Addison & Thorpe, 2004). Moreover, past literature has suggested that individuals with prior contact to persons with a mental disorder tended to exhibit lower levels of stigma, compared to those with little to no prior contact (Desforges et al., 1991). Further research has reported that factors such as gender may play a role in relation to overall stigma levels. The following section will examine these factors in more detail - gender, knowledge about mental illness/ level of education, and prior contact with people who have psychological disorders - as possible factors related to mental illness stigma.

Personal Contact to an Individual with a Mental Illness

One factor that potentially affects attitudes towards individuals diagnosed with a mental disorder is previous history of contact to people with a mental illness (Angermeyer, Matschinger, & Corrigan, 2004). Allport (1954), first suggested the notion that a higher degree of social contact with members of a stigmatised group can help to alleviate negative attitudes toward them. The premise being that contact involves direct exposure to individuals with mental illness - this essentially challenges inaccurate stereotypes which often leads to an

increase in empathy and subsequent reduction in anxiety surrounding the illness (Stuart, 2016).

Research has frequently found that increased levels of contact to an individual with mental illness, is commonly related to more positive attitudes toward that person (Corrigan et al., 2001; Day, Edgren & Eshleman, 2007). Further research supporting this notion suggests that people who have greater knowledge about, or experience with, mental illness are less likely to force social restrictions upon them (Vezzoli et al., 2001) and further reject the popular stereotypes of dangerousness and unpredictability (Corrigan et al., 2001; Penn et al., 1994). Findings report that these same individuals with increased contact, also less frequently express a desire for social distance (Corrigan et al., 2001) one of the primary measures of stigma (or more specifically discrimination). Angermeyer, Matschinger and Corrigan (2004), further confirmed this relationship between familiarity and dangerousness, and familiarity and social distance, suggesting that increasing contact between persons who are mentally ill, and those who are not, appears to be an effective method for reducing discriminatory behaviour (and subsequent stigma).

Gender

There are contradicting results concerning the effects of gender on the public's attitudes towards individuals with a psychiatric diagnosis. On the one hand, multiple studies report that being female (as opposed to male) is associated with the endorsement of positive attitudes toward mental disorders (Gonzalez, Alegria & Prihoda, 2005). For example, one study found that younger males (i.e., 15–17, 18–24) were more likely to report negative attitudes toward mental health treatments when compared to younger females (Gonzalez et al., 2005). Consistent with this trend, findings from a more recent study exhibit that in a representative sample of adults living in Canada, men were more likely than women to endorse stigmatizing attitudes and beliefs (Cook & Wang, 2010). Even across countries this

trend appears relatively stable. That is, findings from various areas (Finland, Lithuania, Ireland, Italy and Portugal) indicate that females exhibit more positive attitudes towards mental illness than men (Chambers et al., 2010). This pattern was consistent in Germany too - where again, females were found to be more accepting of people with mental illness than males (Angermeyer et al., 1998). Alternatively however, several studies have reported the opposite; whereby experiments have found men have significantly less stigmatising attitudes towards mental illness than women (Akinbode & Tolulope, 2017; Lauber, Nordt, Falcato & Rossler, 2004). Opposingly, the discrepancies in this area continue, where in contrast to the studies above, there is a large body of research that has not found any gender differences in attitudes towards individuals who are diagnosed with a mental disorder (Addison & Thorpe, 2004; Angermeyer & Dietrich, 2006; Martin; Nordt, Rössler, & Lauber, 2006).

Although, the inconsistency in findings for this area should not come as a surprise when we reflect that stigma is a multifaceted phenomenon with several components, rather than a standalone construct. This means that while males may be more stigmatising on one facet of stigma (i.e. hold more prejudicial beliefs), females may be more stigmatising on the other (i.e. exhibit more discriminatory behaviour), or vice versa. Literature appears to support this idea, as when researchers have focused on a specific aspect of stigma, gender differences are often reported. For example, one systematic review found that women tended to project less blame and anger (i.e. prejudice) towards people with a mental illness whilst men expressed less fear than women (Holzinger, Floris, Schomerus, Carta & Angermeyer, 2012). Due to the mixed results reported above, the conclusion seems justified that gender does effect attitudes towards mental disorders, and therefore should be accounted for when studying stigmatising attitudes.

Level of Education (and Knowledge about Mental Illness)

The third factor that can influence a person's attitude towards patients with schizophrenia is knowledge. That is, research suggests that the acquisition of factual knowledge regarding psychiatric disorders often certifies a positive attitude towards individuals with a mental illness (Thornton & Wahl, 1996), where the opposite can be assumed regarding inaccurate information increasing negative attitudes. For example, Cangas and colleagues (2017) found that following an intervention to improve overall knowledge about schizophrenia, compared to the control group, those who received the educational intervention reported significantly lower levels of stigma than those who did not.

Moreover, irrespective of level of knowledge toward mental health specifically, the literature proposes that those with a higher education will already have less stigmatizing opinions compared to those with lower levels of schooling (Collins, Wong, Cerully, Schultz & Eberhart, 2013). For example, in a representative sample of Canadian adults, Cook and Wang (2010) reported less stigmatising attitudes (towards mental illness) among those with higher levels of education, in comparison to those with less. Comparable to this, in a more recent study by Yuan et al. (2016) lower education was consistently associated with more stigmatising attitudes towards schizophrenia. Jorm et al. (2012) also noted lack of education as a contributing factor resulting in higher levels of stigma.

However, much like gender, the results in this area are rather contradictory. There are numerous studies which suggest that even professionals who have substantial information regarding mental illness, may not be immune to the prevalent stigmatizing beliefs held by wider society. That is, studies show that healthcare professionals in general are likely to endorse social distance at rates similar to the general public - despite the fact they have a greater knowledge regarding mental disorders (Aghukwa, 2009; Reavley, Mackinnon, Morgan, & Jorm, 2014). Further, surveys find that healthcare professionals endorse the stereotype of blame (Ross & Goldner, 2009) and view individuals with an illness more

negatively, than positively (Lauber, Anthony, Ajdacic-Gross, & Rössler, 2004) despite sufficient knowledge in the area. On the contrary, in the 1990 General Social Survey of the United States, it was reported that those who had less formal education (and therefore less knowledge about mental illness) considered individuals with a mental illness as more dangerous than did the general community (GSS, 1990). Again, the contradictory nature of the aforementioned findings highlights the need for an effective de-stigmatisation programme which targets both the general public, and mental health professionals.

While there are clearly multiple mitigating factors contributing to the marginalisation of people with mental disorders, it begs the question as to what we can actually do in order to combat stigma. What methods are currently out there? And further, what efficacy do these actually have in achieving sufficient stigma reducing effects? The next section will discuss this:

Three Primary Stigma Change Strategies

Decreasing the stigmatizing views mentioned thus far is important for two pivotal reasons; 1) to alleviate the fears and misconceptions of the general public; and 2) to improve the lives of those living with a serious mental disorder (Couture & Penn, 2003). In response to the apparent pervasiveness and endurance of stigmatizing attitudes across time, a multitude of policies and anti-stigma initiatives have been designed and introduced in numerous countries in an attempt to combat the negative effects of stigma on individuals with severe mental illnesses. Whilst there are several suggested methods for how to effectively mitigate stigma, the literature identifies three primary approaches as having the most significant impact on alleviating negative attitudes and behaviours thus far: protest, contact and education (Corrigan & Penn, 1999).

Protest

The first strategy is protest. Protest is the attempt to quash stigmatizing behaviours through directly instructing the non-stigmatised individual not to focus on, or use, negative stereotypes. The two suggested mechanisms of a successful protest campaign are, 1) a clear message condemning stigma on moral grounds, and 2) a negative consequence if the stigmatizing attitude or behaviour occurs (Corrigan, Roe, & Tsang, 2011).

The protest approach has perhaps received the least research attention in comparison to the latter methods as it is thought to be relatively ineffective (Corrigan et al., 2012). In a review of the literature on stigma-reduction, Corrigan and Penn (1999) reported that studies which implemented the protest strategy, did not yield any significant stigma-reducing effects. In fact, some researchers have argued the implementation of this strategy has actually exacerbated stigma through attempting to suppress socially normalised language and practices (e.g., Corrigan & Shapiro, 2010).

Contact

The second stigma reduction strategy is contact. Unlike protest, the efficacy of contact interventions has been widely evaluated in the literature (Bhuller, Williams & Loi, 2018). These interventions involve promoting direct exposure to people with mental illnesses (Couture & Penn, 2003). The premise being that interacting with a stigmatised individual offers the opportunity to discredit stereotypical beliefs and prejudices regarding the marginalised group (Reinke, Corrigan, Leonhard, Lundin & Kubiak, 2004).

Recommendations have been proposed by several researchers regarding indicators that appear to maximise the effectiveness of contact interventions, these included points such as: contact should be face-to-face and one-on-one if possible; the target individual and contact person should be similar in terms of demographics; specific audiences should be targeted first (i.e. those who are likely to have more contact with mentally ill individuals such as hospital workers); the contact should include conversations regarding recovery and the

adverse effects that result from stigma associated with the illness. Additional literature substantiates this idea, suggesting an emphasis on recovery is key for producing stigma reducing effects (Gronholm et al., 2017).

As previously stated, contact is a well-supported strategy for encouraging attitude and behaviour change as it often combines providing accurate information (education) with the opportunity to interact with somebody experiencing a mental illness. In comparison to education and protest, contact yields the most consistent de-stigmatizing results (Corrigan et al., 2012). However, as one can imagine, an approach like this is costly and somewhat hard to achieve in large numbers and without a sufficient amount of planning and resources (Catthoor, De Hert & Peuskens, 2003). This brings us to the third stigma reduction strategy - education.

Education

Being a primary focus of this study, the education approach to destigmitization is of particular interest. Educational interventions aim to diminish stigma through providing people with accurate, fact based information which negates negative stereotypes (e.g. 'contrary to the belief that people with schizophrenia are violent, the actual rate that violent acts are perpetrated by people with schizophrenia compared to members of the general public is miniscule')(Morgan et al., 2012). The underlying assumption being that stigma is perpetuated by inaccurate information, therefore by challenging and correcting this misinformation, stigma is reduced (Casados, 2017). Education as a stigma reduction tactic encompasses several different approaches, some are longer than others; learning courses aimed at improving knowledge around mental illness are more lengthy, versus written or verbal methods - for example, books, information pamphlets, television advertisements and podcasts which are more brief (Corrigan et al., 2012).

A focus on discovering effective techniques to increase understanding around schizophrenia seems crucial, as despite numerous past efforts to increase awareness about this disorder, researchers commonly agree that knowledge regarding this illness remains relatively low among the public (Thorsteinsson, Bhullar, Williams & Loi, 2018). For example, literature reports that schizophrenia is one of the least recognisable disorders (Reavley & Jorm, 2011a). In 2014, a nationally representative sample of Australian individuals found that correct identification of schizophrenia was only 42%. Likewise, In Ireland, the rate of identification for schizophrenia was 34.1% in 2006 (O'Keeffe et al., 2016), relatively low compared to the correct identification rate of depression at 74% in 2011 (Reavley & Jorm, 2011a). While identifying the symptoms of mental illness in the community may not seem like a major problem, studies show a low knowledge base about schizophrenia often leads to a number of issues - these include: an inability to recognise the symptoms of schizophrenia; a lack of knowledge concerning appropriate treatment avenues; incorrect beliefs about the etiology of schizophrenia; and finally, an increase in stigmatising behaviours shown towards individuals with this illness (Thorsteeinsson et al., 2018).

Effectiveness of Educational Interventions at Reducing Stigma

In light of this information then, increasing education surrounding schizophrenia should be seen as a focus point for anti-stigma initiatives so that the wider public can recognise the symptoms of this illness more efficiently, and then, either engage in the appropriate help-seeking behaviours, or consciously make efforts to be inclusive and accepting of those with a mental illness. In a nutshell, most researchers are in agreement that the greater the knowledge about schizophrenia, the lower the stigma towards individuals with this disorder (Angermeyer & Matschinger, 2005; Wood & Wahl, 2006).

Fortunately, numerous researchers have begun to address this issue, testing the effectiveness of various educational campaigns, and further trying to distinguish the most

effective methods to combat stigma in order to best inform both practice and policy. Thus far, educational interventions aimed at increasing public knowledge about schizophrenia have yielded promising results, appearing to improve attitudes and raise awareness surrounding the disorder (Matschinger, & Angermeyer, 2003; Pinfold et al., 2003). For example, a recent study by Thonon et al. (2016) reported that individuals who watched an informative documentary about schizophrenia, reported fewer negative stereotypes and exhibited a reduction in their reported desire for distance from people with this illness, when compared with participants in the control group. Likewise, research by Schulze et al. (2003b) into the effectiveness of educational interventions revealed that students who undertook an anti-stigma education programme (involving group discussion and games) reported less negative stereotypes and an increase in willingness to socialise with individuals with schizophrenia, when compared to the control group in their experiment. Relatively recently, a meta-analysis evaluated 79 studies of public stigma change strategies specific to mental illness with data from more than 38,000 research participants (Corrigan et al., 2012a). Results reflected that both education and contact programmes led to a significant change in overall outcomes for respondents, including an improvement in attitudes and behavioural intentions.

Accounting for the successful results of past research regarding the efficacy of educational interventions at reducing stigma, and in particular Corrigan and Colleagues meta-analysis (2012a), it is unsurprising that the focus of many large scale anti-stigma initiatives often incorporate some kind of educational element. For example, in relation to New Zealand, the 'Like Minds, Like Mine' anti-stigma programme aims to improve knowledge and increase contact between the general public and people with mental disorders. Whilst not primarily concerned with improving education, the combination of increasing public knowledge and contact to persons with a mental illness which comprises the programme, has reportedly resulted in lower rates of discrimination towards individuals with mental disorders,

and a subsequent improvement in knowledge and attitudes, towards individuals with mental disorders amongst the New Zealand population (Gronholm et al., 2017). In addition, the 'Time to Change' programme in England has focused primarily on increasing public knowledge about mental disorders by discrediting popular myths (i.e. people with schizophrenia are violent) via large social media campaigns. Improvements in social distance, attitudes and stigma-related knowledge are among some of the reported benefits from the campaign so far (Henderson et al., 2016). The above information suggests there is considerable merit behind implementing educational interventions in regards to reducing stigma levels among the general public.

Which Educational Information is Best at Reducing Stigma?

However, despite the widespread popularity of educational interventions, research is yet to determine the most effective type of educational information to convey, particularly in regards to schizophrenia (McCrone, Knapp, Henri, & McDaid, 2010). That is, on the one hand, some educational programmes choose to emphasise the damaging effects stigma has upon the lives of those with mental disorders. While on the other hand, some highlight the potential reasons behind the development of mental illness, and further provide information on how to interact and support those with mental disorders. Therefore, what tailored information is most effective at reducing stigma remains an empirical question (Corrigan et al., 2012). Corrigan and Penn (1999) suggested that conveying the correct information is critical for improving attitudes and subsequently producing successful stigma reducing effects. Therefore, considering education is the most commonly employed approach to reduce mental illness stigma, ensuring these strategies are well informed should be of the utmost importance.

One focus of the educational literature thus far has been concentrated on explaining the causes of schizophrenia to the general public, and further assessing what effect this has on

reducing stigmatising attitudes. At present, two causal explanations are predominantly employed for anti-stigma initiatives and endorsed by the general public: psychosocial and biological/genetically (herein referred to as 'biogenetic') based explanations (Read & Harre, 2001), however both have produced mixed results in regards to stigma reduction. Therefore, the following section will explore the theory behind endorsing these causal models as a means to reduce stigma, and further investigate exactly what effect subscribing to these beliefs has had on changing negative attitudes toward mental illness, specifically schizophrenia.

Biogenetic Causal Model of Schizophrenia

Over the past few years, a biological perspective of schizophrenia has made a significant contribution to our understanding and subsequent treatment regimens for the mentally ill (Schomerus et al., 2012). A recent meta-analysis analysing public attitudes towards schizophrenia revealed that over 16 years (1990-2006) and across numerous countries, there has been a consistent trend towards a stronger endorsement of biogenetic causes to explain mental disorders such as schizophrenia (Schomerus et al., 2012). Further research supports this idea, suggesting that in recent years, a prominent anti-stigma strategy employed by many high-income countries has promoted biogenetic causes or 'a disease like any other' messages to the general public (Angermeyer et al., 2011).

However, despite the apparent popularity of biogenetic explanations, research in this area is rather contradictory. On one hand, there is an array of literature which suggests that biogenetic causal explanations decrease stigma by persuading the public that individuals with psychological disorders, are 'ill' in the same sense as individuals with medical conditions (e.g. cancer) (Read & Harre, 2001). However, on the other hand, a plethora of literature suggests that rather than reducing stigmatizing views, biogenetic explanations have had little

or no effect on social stigma and in some cases, may have even deepened it (Read & Harre, 2001; Pescosolido et al. 2010).

Theory Behind Biogenetic Causal Explanations of Schizophrenia

The expectation that biogenetically based causal attributions improve people's attitudes towards individuals with mental disorders comes down to one key concept; attribution theory (Corrigan, 2001). This theory suggests that if the origins of an illness are attributed to factors outside of an individual's control, people's reactions towards that individual will be less negative. That is, notions of guilt and accountability are often primary contributors to the rejection of people with mental disorders. Thus, because biological make-up is considered beyond the control of an individual, this association is thought to reduce the perceived responsibility of the affected individual for being 'ill', therefore reducing adverse reactions by the general public (Schomerus et al., 2014). Overall, anti-stigma initiatives have suggested that biogenetic explanations not only reduce public stigma, they also lower self-stigma for patients (i.e. the illness is not due to my own personality flaws) and those individuals whom the illness indirectly effects – such as families or friends involved (i.e. the disorder is not due to lack of parenting skills)(Angermeyer & Dietrich).

Challenging this view is the idea of 'genetic essentialism' (Haslam, 2011). This theory suggests that the applicability of biogenetic models to mental illness frequently leads non-stigmatised individuals to perceive people with mental disorders as fundamentally different to themselves - consequently resulting in stigmatising attitudes and behaviours. More specifically, genes are commonly perceived as a central part of our identity, that is, they define things such as who we are, and help explain our actions. Therefore, if genes are a 'defining' aspect of our 'being', a biogenetic explanation for mental illness would only exacerbate any stigma directed toward the disordered individual by creating the perception that there is a profound difference between 'us' and 'them' (Walker & Read, 2002). Research

suggests this explanation would make mental illness appear more persistent and portray the patient as less in-control (as their actions are determined by their genetic make-up), therefore fostering negative stereotypes. In agreement with genetic essentialism, Phelan and colleagues (2002) suggest that biogenetic explanations lead to a decline in hopefulness that the mental illness can be improved, and often increase the belief that family members are partially responsible for the illness and will probably be affected by that disorder as well (Angermeyer & Dietrich, 2006).

Based on the above theories there is a clear disparity in the literature as to whether biogenetic explanations will have positive or negative effects on the public's perceptions of mental illness, and, despite the widely accepted use of biogenetic explanations in anti-stigma programmes, it appears unclear whether promoting this model as a de-stigmatisation tool is entirely appropriate or fit for purpose. As mentioned previously, stigma is a multi-faceted concept, thus it consists of various opinions, emotions and behaviours (Corrigan, 2002). Therefore, due to the various dimensions that constitute stigma, it would be inaccurate to presume that biogenetic explanations will have the same effect on all stigmatizing opinions (e.g. that they will reduce or increase every aspect of stigma to the same degree). This notion appears to be reinforced by recent research which indicates that the effectiveness of biogenetic explanations at reducing stigmatising attitudes, varies according to which specific aspect is being measured (e.g. stereotypes or discrimination)(Lincoln et al., 2008).

Endorsing a Biogenetic Causal Model of Mental Illness

The following section will discuss this idea in more depth. That is - What are the findings in the literature which both support and negate the use of teaching a biological explanation of mental illness when attempting to reduce negative attitudes around mental illness? Does subscribing to biogenetic beliefs appear to diminish stigma? Or perpetuate it?

As discussed above, genetic essentialism predicts an increase in stigma through the belief that genes form the basis of our identity and therefore are indistinguishable from our behaviours, adding a perceived aura of uncontrollability to the mentally ill individual (Phelan, 2005). Much of the literature utilising the biogenetic framework reiterates this notion, proposing that they are predictive of an increase in stigmatizing attitudes and behaviours such as; a greater desire for social distance (Dietrich et al., 2004), higher ratings of perceived dangerousness (Read & Harré, 2001) and unpredictability (Boysen & Vogel, 2008; Read & Harré, 2001).

Research by Angermeyer and Matschinger (2005) somewhat confirms these sentiments. After conducting a trend analysis on two population surveys regarding causal beliefs and stigma, they found that participants who were more likely to endorse biological factors as a cause for schizophrenia, the more lacking in self-control, unpredictable and dangerous respondents rated these individuals to be. This then had a flow-on effect, whereby, the more dangerous respondents perceived the individual to be, the more likely they were to express an increased desire for social distance and a higher degree of fear. Analyses like these, appear to suggest that advocating biogenetic explanations actually decreases the prospect of social acceptance for individuals with schizophrenia, therefore potentially magnifying the stigma around this already polarising disorder. Consistent with these findings, Phelan (2005) reported that endorsing biogenetic causal explanations of mental illness were related to several stigmatising attitudes and behaviours among respondents in their sample. Specifically, participants in this group reported higher levels of perceived seriousness and persistence of mental disorders, as well as an increase in the belief that relatives and offspring would develop the same illness. Further, solidifying the above results, Pescosolido et al. (2010) investigated the effect of teaching different causal explanations on stigmatizing opinions surrounding mental health. They found that although the ascription of biogenetic

causes for schizophrenia had increased over a ten-year time period, from 76% (1996) to 86% (2006), public stigma and the subsequent perceived dangerousness of these individuals had also been amplified during this time. Additionally, a survey conducted by Read and Law (1999) ascertained that individuals who endorsed biogenetic causal beliefs for mental illness were also more likely to perceive patients with mental disorders as dangerous and unpredictable, and far more likely to avoid contact with them.

The above literature appears to portray the use of biogenetic explanations as a de-stigmatization tool in a rather negative manner, highlighting the various elements of stigma which often become exacerbated (e.g. increase in perceived dangerousness and unpredictability) when endorsing or teaching this kind of model. However, it would be unwise to completely discredit the use of biogenetic explanations as a stigma reduction technique, as it appears there are certain elements of stigma that biological explanations can improve. For example, whilst endorsing a biogenetic causal explanation does appear to intensify certain elements of stigma, the belief that mental illness is biologically driven has also been found to decrease certain stigmatising factors among the public, such as blame and punishment for actions resulting from the illness (Phelan, Cruz-Rojas & Reiff, 2002).

For example, Boysen and Vogel (2008) and Phelan et al. (2002), tested the effectiveness of biogenetic causal explanations at lowering stigma toward mental illness. Conflicting previous findings, both studies reported that biogenetic explanations appeared to effectively reduce one area of stigma, significantly lowering levels of blame among respondents. A relatively positive finding in contrast to those mentioned above.

Over the past few years, five main review articles have been published attempting to determine the efficacy of biogenetic explanations at reducing stigma amongst the public (Angermeyer et al. 2011; Jorm & Oh 2009; Kvaale et al. 2013a, b; Read et al. 2006). The first review, conducted by Read et al. (2006), reported that biogenetic causal explanations were

positively related to attitudes predicting unpredictability, fear, the desire for social distance and perceived dangerousness. Likewise, Jorm and Oh (2009), suggested that the endorsement of 'brain disease' as a cause for schizophrenia was associated with an increased desire for social distance. Consistent with the aforementioned findings, two meta-analyses reported that biogenetic explanations were positively associated with reduced blame, however, these same explanations were also associated with negative stereotype ratings such as unpredictability, dangerousness, fear (Kvaale et al., 2013a) and poor prognosis (Kvaale et al., 2013b). Finally, Angermeyer et al. (2011) reported that overall biogenetic explanations were associated with more negative attitudes toward mental illness, and consequently are an inappropriate method of reducing stigma toward this group.

Thus, both theory and empirical research suggest that the effectiveness of endorsing and teaching biogenetic attributions are variable, reducing some dimensions of stigma (i.e. blame, punishment) whilst appearing to magnify others (i.e. desire for social distance, perceived dangerousness).

Endorsing a Psychosocial Causal Model of Mental Illness

Bearing in mind the questionable results of utilising a biogenetic model of mental illness for reducing stigma, Read (2007) recommended that teaching people about the psychosocial causes of mental illness may yield more success in this field. In contrast to biogenetic explanations then, psychosocial reasoning reframes symptoms as developing as a result of certain life events which often involve some kind of trauma or environmental stressor (e.g. physical, sexual or psychological abuse, poverty). Whilst there are both positive and negative reviews regarding the effectiveness of these explanations at stigma-reduction, they appear relatively powerful at reducing certain aspects of stigma, particularly factors which consistently seem aggravated when using a biogenetic model. This psychosocial model is of particular interest when studied in a New Zealand context (as in the current study), as

research suggests New Zealanders tend to ignore a medical model of mental illness, opting to believe psychosocial reasons instead. For instance, a study conducted by Read and Harré (2001), reported that the New Zealand public tend to reject biological explanations of mental illness in favour of psychosocial explanations, particularly explanations focused on negative life events. Additionally, mental health professionals in New Zealand tend to agree with the promotion of psychosocial causes over biogenetic causal reasons, with one study reporting that 91% of clinical psychologists and 58% of psychiatrists endorsed more psychosocial, than biogenetic factors when queried about the causes of mental illness (Young, Read, Barker-Collo & Harrison, 2001).

There is certainly evidence which suggests that merely subscribing to psychosocial causal beliefs of mental illness (as opposed to biogenetic) is related to lower levels of prejudice and discrimination. For example, a survey conducted by Read and Harré (1999) reported that inidivudals who endorsed psychosocial causes for mental disorders also desired less social distance than respondents who did not. Consistent with this notion, in a study of the Egyptian public, individuals who cited psychosocial factors as a cause for mental illness reported less desire for social distance than people citing alternative causes (Coker, 2005). Providing educational interventions which highlight psychosocial causes of mental illness also appear to have positive effects on attitudes towards people with mental disorders. For example, Lam, Salkovsis and Warwick (2005) measured the effect of differing causal labels on the public's perception of various psychiatric problems, specifically schizophrenia. Respondents who were in the psychosocial condition rated individuals with mental disorders as more likely to be curable, less likely to pose a danger to both themselves and others, and reportedly considered the patients problem less disabling, when compared to the responses from other groups. Perhaps most recently, Longdon and Read (2017) investigated the effects of mental health anti stigma programmes according to their causal frameworks - they

indicated there is substantial evidence that anti-stigma campaigns which frame psychosis as a response to hardship and adversity are extremely effective at reducing levels of overall stigma. They also reported that psychosocial models more successfully 'humanize' people with mental disorders when compared with biologically dominated frameworks.

Taking the aforementioned information into account, it appears that teaching (or simply believing) a psychosocial model of mental illness has relatively de-stigmatizing effects on multiple components of stigma, including factors which are often magnified when using a biogenetic explanation. However, contrary to previous work, a study by Schlier et al. (2014) reported that subscribing to psychosocial causal beliefs of schizophrenia resulted in an increase in blame, a significant contributing factor to the stigmatisation of those with schizophrenia.

Ultimately, the literature is inconclusive in this area, both biogenetic and psychosocial explanations appear to constitute a decidedly mixed bag in reference to the general public's stigmatising attitudes (Rüsch, Todd, Bodenhausen & Corrigan, 2010). On a positive note, the endorsement of biogenetic causal models appear to be positively associated with weaker implicit blame, much like attribution theory would suggest. However, on the negative side, these explanations also appear to be associated with a stronger desire for social distance, and a greater belief that patients with schizophrenia are unpredictable and dangerous, as proposed by genetic essentialism (Rüsch et al., 2010). On the contrary, in the literature, psychosocial beliefs consistently appear to improve components of stigma which are often aggravated by biogenetic explanations (i.e. desire for social distance, unpredictability, lower levels of dangerousness) whilst simultaneously worsening (or not affecting) mechanisms of stigma which biogenetic explanations consistently seem to improve (i.e. blame). Taking the above information into account, it appears there is slightly more support toward delivering a stigma initiative which emphasises the psychosocial causes of schizophrenia, however we cannot say

this with complete confidence. That is, while the aforementioned literature assessed the theory behind teaching a biogenetic or psychosocial causal model of mental illness, and further, the effects of endorsing or teaching these two models on stigma separately, it did not account for how the results may differ when the two causal interventions are directly compared to one another. Moreover, as better informing and improving current educational anti-stigma campaigns is a primary goal of the current research, it is pivotal we investigate how delivering these causal models as part of a teaching intervention specifically, effects stigma. This will allow us to further ascertain whether one causal model should be favoured over the other. Luckily, analyses directly comparing the effects of teaching the opposing models has begun, however it is limited to a small number of studies;

Comparing the Effects of Two Causal Models

The first study to directly compare the effectiveness of teaching these opposing models on stigma was conducted by Farina, Fisher, Getter and Fischer (1978). For this experiment participants were provided either a biogenetic or psychosocial explanation for mental illness. Respondents then completed a survey that asked how humiliating they thought it would be to be a mental patient who required care for their illness, and further, the amount of control they believed the patient had over their mental disorder. In regards to the former question, no differences were found. However on the latter question, respondents in the psychosocial condition reportedly believed patients had significantly more control over their illness, compared to those in the biogenetic condition.

Similarly**,** Boysen and Vogel (2008) investigated the efficacy of teaching different educational interventions on 232 psychology undergraduates' attitudes towards individuals with mental illness - specifically schizophrenia. Participants were provided either a psychosocial or biogenetically based causal explanation of schizophrenia to read. They were then asked to rate the extent to which they perceived their attitude towards people with

schizophrenia had changed due to the nature of the explanation. Specifically, levels of discrimination (desire for social distance) and stereotypical belief (blame) were measured. Results revealed only one significant difference in attitude measures between the two causal groups; individuals who read the passage relating to the biogenetic causes of schizophrenia reported a significant reduction in 'blame.' No further differences in stigma between the two etiological explanations were reported.

Further research in this area was carried out by Lincoln et al. (2008) who assessed the impact of biogenetic and psychosocial causal explanations on 121 psychology students' attitudes (stigma) towards schizophrenia. Participants were provided either a biogenetic or psychosocial causal explanation for schizophrenia, via a short video and information booklet. Rather than reading causal information, participants in the control group read an excerpt about glaciers and viewed a short video about icecaps. Participants stigmatising attitudes were measured on both implicit and explicit measures, before and after the causal interventions. More specifically, explicit attitudes were assessed using a stereotype questionnaire designed by the authors of the experiment (which measured degree of patient blame, perceived dangerousness and unpredictability) and the Social Distance Scale (which assessed participants desire for distance from people with schizophrenia)(Link, Cullen, Frank & Wozniak, 1987), while implicit attitudes were measured using the Implicit Association Test.

Lincoln and Colleagues (2008) reported that pre-intervention levels of stigma were high across all three groups (i.e. biogenetic, psychosocial, control). However, this stigma was significantly reduced for both causal groups (but not the control) following the post-intervention stigma measurement. Further post-hoc analyses revealed a series of interesting findings regarding the two causal conditions. Interestingly, participants desire for distance was significantly reduced between pre and post-test measurements for both the causal

conditions. However, comparable to the findings of Boysen and Vogel (2008) only the biogenetic group experienced a reduction in patient blame. In addition, however, the stereotype of unpredictability was found to be reduced between pre and post-test measurements for the biogenetic condition (but not the psychosocial condition), whereas the stereotype of dangerousness was lowered among participants in the psychosocial condition (but not the biogenetic condition). Lincoln and colleagues (2008) concluded from their results that causal explanations certainly do reduce stigmatizing attitudes towards schizophrenia, however, they appear to work in distinct ways, reducing different aspects to one another. Accounting for these findings, they made the recommendation that perhaps a multidimensional approach, which incorporates both psychosocial and biogenetic information would be more appropriate. And, potentially more effective at reducing stigma overall.

Despite this recommendation, previous experiments conducted both before and after Lincoln and Colleagues (2008) work had assessed the destigmatizing effects of teaching a combined (diathesis-stress) model, compared to explanations which were predominantly biogenetic or psychosocial in nature, with little success. That is, these studies have generated relatively mixed results, and unfortunately, have not produced the positive outcomes that were originally anticipated.

Walker and Read (2002), were the first to consider a diathesis-stress model as a potential method of stigma reduction alongside other causal explanations. Comparable to the method of Lincoln and colleagues (2008), their study also utilised a pre-test, post-test experimental design. Participants pre-existing stigma was measured using the TAS (Total Attitude Scale) which assessed attitudes regarding perceived dangerousness and ratings of unpredictability towards people with mental disorders, whether people with mental illness should be able to make decisions for themselves, and finally, the amount of social distance someone wishes to have from a person with a mental illness. Respondents then viewed a

video of a patient describing the symptoms of their illness, followed by a doctor detailing one of three causal explanations for the illness (either psychosocial, biogenetic or combined in nature). Post-intervention stigma levels were then measured.

It was reported that participant's pre-existing attitudes towards mental illness were relatively negative overall. Additionally, no significant difference was found between the three causal conditions after analysing the change in TAS scores between pre and post intervention. However, when analysing scale items individually, a number of differences were reported. First, participants in the biogenetic condition reported a significant increase in perceptions of dangerousness and unpredictability. Second, participants in the combined explanation condition rated people with mental illness as significantly more dangerous compared to their pre intervention ratings. Further, the psychosocial intervention did not yield any improvements in attitudes toward mental illness between pre and post-test measurements. Their overall conclusion was that biogenetic explanations actually lead to an increase (rather than decrease) in negative attitudes (specifically dangerousness and unpredictability) towards mental illness and therefore, should not be used in anti-stigma interventions. Further, despite the fact that the psychosocial condition did not find any improvements in stigma, Walker and Read (2002) recommended incorporating psychosocial causal explanations into anti-stigma campaigns.

To the best of my knowledge, only one other study has investigated the effects of teaching a diathesis-stress model for schizophrenia compared to a biogenetic and psychosocial causal model on stigma. Utilising a pre-test / post-test experimental design, Schlier et al., (2014) compared the effectiveness of three different causal models at reducing stigma. However, unlike the study by Walker and Read (2002) a fourth condition for comparison was added- a control. First, participants pre-existing stigma was assessed on a series of self-report questionnaires which indicated the level of discriminatory attitudes

(desire for social distance) and stereotypical belief (unpredictability, perceived dangerousness, and patient blame) participants held towards patients with schizophrenia. Next, respondents received either a psychosocial, biogenetic, vulnerability-stress or control intervention. The causal conditions consisted of a short video and information text pertaining to the condition each participant was in, whilst the control group saw no information regarding etiology. Following this intervention, levels of stereotypical belief and social distance were reassessed.

Much like the aforementioned studies, the findings reported by Schlier et al., (2014) were relatively mixed. That is, not every component of stigma was reduced following the causal explanations, and the ones that were, were affected differently according to what causal intervention participants had been a part of. The endorsement of the stereotype, 'perceived dangerousness' was significantly lower for participants in the biogenetic condition compared to the other two causal explanation groups. Further, blame was decreased among respondents in the biogenetic condition when compared to those in the psychosocial group. However, no differences were found between causal conditions on measures of desire for social distance or perceived unpredictability. From these results, Schlier and colleagues (2014) concluded that vulnerability-stress models are no more effective than psychosocial or biogenetic causal explanations in terms of reducing stigma. Further, they noted that the effects of biogenetic and psychosocial are clearly limited and vary according to which aspect of stigma is being measured, and thus further research should focus on exploring alternative methods of stigma reduction, which are perhaps not etiological in nature.

Taken together, the findings directly comparing the effectiveness of causal education on reducing stigma are both complex and inconclusive. For example, Schlier and colleagues (2014) found that perceived dangerousness was reduced after participants were exposed to a biogenetic explanation for schizophrenia, whereas Walker and Read (2002) found that

biogenetic explanations increased these perceptions. Further, Lincoln et al., (2008) reported no change in levels of perceived dangerousness for participants in the biogenetic condition, but a reduction in these perceptions for participants in the psychosocial condition. Whilst it was hoped that directly comparing the two causal teachings would result in an obvious answer as to the best causal explanation to enlist for educational resources and anti-stigma interventions, the picture remains unclear. We can conclude that while etiological information appears to reduce stigma to some degree, the effect of each explanation varies, according to which aspect is being measured. This is perhaps unsurprising however, given that stigma is a multi-faceted phenomenon (Corrigan, 2000).

Therefore, it appears that the important task of developing effective educational anti-stigma programmes faces some crucial challenges, where more testing is warranted in order to discern whether causal information really is the most appropriate when trying to reduce stigma, and further, whether one causal explanation is considerably more effective at reducing stigma than the other.

The Link between Creativity and Schizophrenia

Due to the large disparity among the current literature, it would appear that perhaps the most appropriate move forward would be to develop an intervention which does not focus on the etiology of schizophrenia, but rather the potential reasons why this illness developed in the first place, and further, why it still exists today. The premise being, if normal individuals see the positive and potentially advantageous side to having an illness like schizophrenia, public stigma should be lessened, as it is arguably no longer a mental illness that has developed with no purpose. Schizophrenia has provided scholars with a perplexing question - why has a seemingly negative and highly debilitating disorder persisted for so long? Whilst there is still no definitive answer to this question, given the large proportion of individuals diagnosed with schizophrenia globally (around 1% of modern populations) research suggests

it is unlikely the disorder has developed solely from random mutations (Erlenmeyer-Kimling & Paradowski, 1966). Rather, it is believed that - despite its seemingly maladaptive nature - this disorder has been specifically selected throughout the years, due to its potential evolutionary advantages. While this may seem far-fetched and unfeasible, evolutionarily speaking it makes sense, as if a trait has no beneficial adaptation, it is generally selected out of the gene pool and subsequently ceases to exist.

One main theory predicts that there are certain evolutionary benefits to schizophrenia, such as enhanced creativity. The connection between madness and genius is one of the most persistent among the general public - having been established many years ago. This link is commonly referred to as the mad-genius hypothesis, and is often explored in terms of the potential relationship between creativity and psychopathology (Kar & Barreto, 2018). Creativity may be one of the only fields which depicts mental illness in a reasonably positive light, as opposed to being portrayed as something to avoid.

So what advantage does enhanced creativity have evolutionarily? It has been proposed by some that historically higher levels of creativity would increase chances of mating success (therefore producing a genetic advantage for the possession of mental illness). For example, studying a targeted sample of those in creative professions (poets and writers) and a sample of the general public, Nettle and Clegg (2005) found that the number of sexual partners an individual had, was positively correlated with certain dimensions of schizotypy. It can also be argued that creativity is a critical tool for human development and survival, increasing problem-solving behaviours and the ability to adapt to different experiences through providing an enhanced and diverse way of thinking. It is often hard for healthy individuals to move beyond their conventional method of thinking, however for individuals with schizophrenia, forming novel ideas may be slightly easier due to their versatile thought patterns resulting from the experience of hallucinations and delusions. Whilst research in this

area is still relatively scarce, in 2010, a study which conducted brain scans exhibited similarities between the thought pathways of schizophrenics and creative people (De Manzano et al., 2010). Consistent with this research, there appears to be a large proportion of creative geniuses who have suffered from mental health issues in the past, particularly schizophrenia (e.g. John Nash, the father of game theory; and Vaslav Nijinsky, the legendary dancer and choreographer).

Conversely however, there is an array of literature which suggests the 'link' between creativity and mental illness is inconsistent and inaccurate, and that the previous findings which reported connections between the two, are merely a coincidence (Kyaga et al., 2011). Therefore, in regards to the current writing piece - it must be noted that the concept of creativity as an argument for the continual presence (and apparent continued growth) of schizophrenia in society, is not being employed for this research based on its scientific accuracy or verification in the literature, but rather, as a novel avenue through which to explore potential alternative educational methods of stigma reduction. That is, unlike the psychosocial and biogenetic approaches which are largely theory driven, and tend to be in direct opposition of one another, the creativity explanation is relatively eclectic and not so much grounded in past work. Thus, while the previously reported methods of stigma reduction were based on etiology, and the potential factors which contribute to the development of schizophrenia, the creativity side of things is largely focused on why the disorder is still present today, and what advantages a disorder like this can offer society. As we are already aware, education is one of the primary methods of stigma reduction. Therefore, the possibility that presenting individuals with an alternative reason for the presence of mental illness in society should be explored. The addition of this novel component should be considered a direct response to the claims made by Schlier et al. (2014) who suggested testing the efficacy of an educational intervention which is not focused on the

causes of schizophrenia. Further, this component can act as somewhat of a 'control' condition for the current experiment, in order to assess levels of stigma between participants who have received causal information, and those who have not.

Current Research

Although a large body of research has looked at various ways to reduce stigma – often with a particular focus on etiological education, the findings in this area remain contradictory and unclear. Therefore, the current investigation aims to explore these issues further. More specifically, because of the conflicting results of previous studies, it has been rather difficult to ascertain which type of causal information is most effective at reducing stigma, when utilising an educational intervention. Whilst we know that providing certain biogenetic or psychosocial explanations for mental illness has proven to significantly diminish certain aspects of stigma, we must also be aware that both of these explanations can sometimes enhance specific components of stigma too (Schlier et al., 2014; Walker & Read, 2002). So far then, it has been hard to draw definitive conclusions as to what information should be included in educational anti-stigma programmes in order to garner the best results (e.g. psychosocial or biogenetic causal explanations). Additionally, biogenetic and psychosocial explanations appear to dominate the literature in these experiments with a large majority of previous educational studies focusing on these two causal models. Accounting for the inconsistent findings reported above, it remains of further interest whether providing etiological information is actually the most effective method when attempting to reduce stigma toward schizophrenia. In regard to this second point, the current study will test two novel explanations (related to schizophrenia) which have not yet been explored in the stigma reduction literature, in order to further discern what kind, and whether, etiologically based information really is the most appropriate method of reducing negative attitudes toward schizophrenia.

Further, as discussed, there are many elements that comprise stigma, thus studies enlisting only one or two questionnaires measuring single constructs of stigma could be interpreted as not having a wide enough scope to accurately interpret their results and the effects of different explanations on stigma. Therefore, unlike previous studies (Boysen & Vogel, 2008; Farina et al., 1979; Lincoln et al., 2008; Schlier et al., 2014; Walker & Read, 2002) which have compared the effectiveness of teaching a biogenetic or psychosocial causal model on one or two aspects of stigma (i.e. stereotypical belief and discrimination), the current research will enlist a more comprehensive method. That is, this research will utilise a series of questionnaires which allow all components of stigma (stereotypical belief, prejudice, discrimination) to be analysed independently of one another. The premise being that this will provide an indication of the most effective stigma-reducing explanation. These additions to the present study will allow us to achieve a more comprehensive review of the differential positive and negative outcomes that various educational interventions may have on stigma levels, and further allow us to establish exactly what information should be taught to reduce stigma. Overall, this study aims to both improve and extend on previous research in this area.

The current study contained two experiments, each comparing the stigma levels of participants who were presented either a causal explanation (i.e. biogenetic, psychosocial, epigenetic) for mental illness, or no causal explanation (i.e. novel creative explanation, or no explanation at all). Across both experiments, I investigated three key research questions. First, I ask whether teaching participants a causal explanation for schizophrenia reduces stigma compared to a novel, non-causal explanation, or no explanation at all. Second, I ask if there is a difference in the stigma reduction between the biogenetic and psychosocial causal conditions. That is, is one causal explanation clearly more effective at reducing stigma than the other? And finally, I ask whether there is an untested explanation for schizophrenia

(whether causal in nature or not) that is more effective than the current psychosocial or biogenetic causal explanations at reducing stigma.

Experiment One

Participants in Experiment One were first introduced to the idea of schizophrenia via a five minute video of a patient with this disorder talking to a psychiatrist. This patient exhibited most of the common symptoms associated with this illness. Each particpant then read a text exert detailing the main symptoms of schizophrenia before being exposed to one of three different experimental conditions- two containing causal information (psychosocial and biogenetic) regarding schizophrenia, and one focusing on novel, evolutionary (creative) information. The three components of stigma were assessed using four separate questionnaires; the Schizophrenia Stereotype Questionnaire (SSQ)(and Schizophrenia Perceived Dangerousness Subscale SPDS) measured participants tendency to believe stereotypes regarding schizophrenia, the Emotional Reactions Questionnaire (ERQ) measured respondents prejudice, and the Social Distance Scale (SDS) and Desire to Control Questionnaire (DCQ) assessed participants discrimination. Therefore, in the current study the independent variable was type of information presented to participants, while the dependant variable was participant's scores on the stigma questionnaires. To ascertain whether our manipulation of vignette had significantly altered participant's stigma, participants mean score on each questionnaire was compared between conditions.

Hypotheses and Predictions

Consistent with the findings from previous research comparing the effects of causal information on stigma, I hypothesised that regardless of which causal condition participants belonged to, there would be some amount of stigma reduction on at least one of our stigma measures, compared to the novel, creative condition (Boysen & Vogel, Schlier et al., 2014). Secondly, consistent with the knowledge that stigma is a multifaceted component and that

different causal explanations tend to exacerbate some components of stigma whilst diminishing others (Boysen & Vogel, 2008; Corrigan, 2000; Schiler et al., 2014), I predicted that the causal explanation which most effectively reduced stigma, would differ according to which aspect was being measured (i.e. stereotypical belief, prejudicial emotions, discrimination). That is, when analysed independently, the three components of stigma will be affected to different degrees according to whether the explanation is biogenetic or psychosocial in nature. In light of this second hypothesis, I make separate predictions based on the different components of stigma:

1) Consistent with the findings of Boysen and Vogel (2008) and Lincoln et al. (2008) who reported less blame among participants in biogenetic causal explanation conditions following an educational intervention, I predict that participants in the biogenetic condition will exhibit lower levels of stereotypical belief (and therefore less stigma) on the SSQ measure, when compared to the scores of participants in the psychosocial causal condition.

2) Secondly, as previous research has demonstrated that biogenetic causal explanations consistently appear to increase perceived dangerousness (Angermeyer & Matschinger, 2006; Haslam, Sayce & Davies, 2006; Walker & Read, 2002) when compared with psychosocial causal explanations- which has been found to decrease perceived dangerousness (Read & Law, 1999; Lam Salkorvsis & Warwick, 2005; Lincoln et al., 2008) I predict that participants in the biogenetic group will exhibit higher scores on the SPDS (and therefore view people with schizophrenia as more dangerous) than those in the psychosocial causal explanation condition.

3) In line with findings of Lincoln and Colleagues (2008) who reported no difference in the reported desire for distance among causal groups, I hypothesised that there would be no difference in the SDS scores of the biogenetic or psychosocial group.

4) To the best of my knowledge, as no previous studies have directly compared the effectiveness of teaching psychosocial and biogenetic causal explanations of mental illness and their effect on prejudice or desire to control, I make no prediction regarding participant's scores on the ERQ or DCQ.

Finally, as there are currently no other studies which utilise a vignette based on creativity and schizophrenia. I make no predictions regarding how this explanation will fare in terms of stigma scores, when compared with the two causal conditions.

Method

Participants

One hundred and fifteen undergraduate students enrolled in a first year psychology course at Victoria University of Wellington completed a 30 minute experiment in exchange for course credit. Seven participants were excluded for incorrectly answering at least one of the first two control check questions, regarding the contents of the video, suggesting that they had paid insufficient attention to the material in the clip.

A further 11 participants were excluded for answering the final control check question incorrectly. This question asked them to recall the content of the vignette condition they had been assigned too (i.e. a paragraph relating to schizophrenia and creativity, or schizophrenia being caused by biogenetics/ psychosocial factors). These participants were excluded as it was decided that if they answered the control check question incorrectly, they had paid insufficient attention to the written material in the vignette (our independent variable) and therefore their responses on the subsequent stigma measures may not have been equally as affected as other participants who had concentrated on the content.

The final sample comprised 97 participants. Of those individuals, 72 (74.2%) were female, 24 (24.7%) were male and 1 (1%) identified as 'other'. The mean age of the sample was 19.06 (*SD* = 2.94) and a large proportion of respondents (78.4%) reported 'New Zealand European' as an ethnic group they identify with. A large proportion of students (86%) reported that they had some previous experience with a mental health issue, whether that be themselves (*n* =37), experience with a close friend or relative (*n* = 38), or some experience in the community (*n* = 8). Only 14 individuals claimed to have no experience at all. Additionally, a small number (14.4%) of the sample population claimed to know somebody with schizophrenia.

Participants were randomly assigned to one of three conditions (biogenetic explanation condition, psychosocial explanation condition, creative explanation condition) through a randomizing function on Qualtrics software. That is, during each experimental session (comprising eight or nine individuals) participants were evenly assigned to one condition. Therefore, during a session with nine participants, three would be assigned to the creative condition, three to the psychosocial condition and three to the biogenetic condition. Following the exclusion of participants who answered the control check questions incorrectly, 36 participants were in the biogenetic causal condition, 37 were in the psychosocial causal condition and 24 were in the creative condition.

Materials

Demographic questions. At the start of the experiment, participants were required to answer a series of basic demographic questions relating to age, gender and the ethnicity they identify with (they were allowed to choose more than one option). Additionally, participants were asked if they had any personal experience with a mental health issue, and further if they knew anybody who suffers from schizophrenia.

Video Clip. Irrespective of condition, participants watched a five-minute video clip of a man named Gerald speaking to his mother and a psychiatrist. This clip was found on YouTube, however was originally sourced from a television documentary. During this interview, Gerald displays a range of symptoms commonly associated with schizophrenia, such as; disorganised speech, paranoid thinking, delusions, difficulty concentrating and dissociative thinking. More specifically, delusions of persecution are clear as he discusses his belief that someone is going to 'electrocute' him. Further, Gerald's disorganised thought is displayed through his frequent speech derailment (such as when he starts talking about sperm and eggs and ends his comment talking about how the brain is an atom). The video clip ends with Gerald displaying further derailment (in both his thoughts and speech) in his therapy session. The clip did not provide participants with any information regarding the potential causes of schizophrenia. Rather, it was utilised as an informative tool to show participants the symptoms of an individual with schizophrenia.

The video observed by respondents was clipped with the interview being cut short in order to save time and cull unnecessary information. In total, the clip played for five minutes and twenty one seconds and had a timing function attached so participants could not progress past the survey until the allotted time had passed.[1]

Content-Check Questions. Throughout the duration of the experiment, participants were required to answer three content check questions in order to assess whether they had paid adequate attention to the material they had seen and read during testing. The first two questions appeared mid-way through the experiment, directly following the video clip. The third content check question appeared after participants had been split into their respective

---

[1] The video of Gerald can be viewed exactly as it was seen by participants at this address: https://www.youtube.com/watch?v=gdIUAuAzxxU

causal (or non-causal) conditions and read a paragraph containing information either about the cause of schizophrenia, or why it may have developed in the first place.

The first two content check questions assessed whether participants had paid sufficient attention to the material in the video clip:

1. Gerald discusses some concerns of his. Which of the following does Gerald not talk about?

2. Gerald discusses some concerns of his. Which of the following does Gerald NOT talk about?

For both of these questions, participants could choose from three possible answers, however they were only able to select one of these options (Refer to Appendix A for the answer options). Participants who answered either of these questions incorrectly were excluded from the analysis.

The final content check question was designed to assess whether participants could correctly recall which condition they had been in; participants were asked, "Prior to this page, you read an exert which explained the most likely cause for schizophrenia, or the reason for its continual existence in society today. To ensure you have been concentrating, please recall the option which best reflects the contents of the information you just read." Participants could choose from 3 options – 'schizophrenia is caused by biology/ genetics', 'schizophrenia is caused by psychosocial factors' (trauma, problematic childhood, stress) or, 'schizophrenia is a creative adaptation'. If participants answered this question incorrectly, it was assumed they had paid insufficient attention to the manipulation condition in our experiment, and therefore were excluded from additional analyses.

Information about Schizophrenia. In order to solidify and verify the information from the video, participants read half a page of information explaining the key symptoms of schizophrenia (hallucinations, delusions, disordered thought/ behaviour). Throughout the text,

participants were prompted to think back to the clip of Gerald and how he displayed some of these symptoms when talking to his psychiatrist. For example, participants were reminded how Gerald talks about people who are searching for him to electrocute him and put him in jail- an example of a paranoid delusion. This component was introduced to the experiment to ensure participants had an accurate understanding of the symptomology of schizophrenia. See Appendix B for a complete copy of this information.

Vignette Information (Biogenetic, Psychosocial, Creative). Immediately following the text discussing symptoms of schizophrenia, participants were randomly split into one of three experimental conditions (biogenetic, psychosocial, and creative). Participants in the biogenetic and psychosocial condition both read an exert which detailed the most likely cause of schizophrenia. The content of each vignette was dependant on which condition each participant was in. For example, participants in the biological condition were instructed that schizophrenia develops due to brain abnormalities, whereas those in the psychosocial condition were informed that schizophrenia was the result of adverse life experiences (e.g. childhood trauma, stress, abuse). Participants in the creative control condition were not given any information regarding the causes of schizophrenia, rather, they read an exert explaining a possible reason schizophrenia still exists in society today (enhanced creativity has doubled as a survival tool). To view these vignettes in their entirety, see Appendix C.

Scales Enlisted to Measure Stigma. To comprehensively measure the various aspects of stigma identified earlier, participants answered forty-seven statements taken from four different scales (and one sub-scale). Each scale used a five point Likert to measure responses (1 = 'strongly disagree', 2 = 'somewhat disagree', 3 = 'neither agree nor disagree', 4 = 'somewhat agree', and 5 = 'strongly agree'). Each scale focused on measuring some component of stigma (i.e. level of stereotypical belief, prejudice and discrimination) in relation to schizophrenia. For example, the first scale (and subscale) allowed us to assess the

amount of stereotypical beliefs (i.e. people with schizophrenia lack control, are unpredictable and unsafe to be around) participants displayed towards individuals with schizophrenia. The second scale measured prejudice reactions (i.e. I feel pity, I feel annoyed, I feel irritated), and the fourth and fifth measured discrimination (in the form of distancing oneself from certain interactions with people with schizophrenia and the amount of control one wishes to exert over an individual with schizophrenias life) towards people with schizophrenia. These scales (and SPDS subscale) were first combined by Cunningham (2016) to form a questionnaire which would examine stigma levels in accordance with Corrigan's (2002) conception that stigma comprises three separate components. These scales are examined separately and more comprehensively below (please see Appendix D to view the full list of these scales used in the current experiment).

Assessing stereotypical belief. As discussed earlier, the first identifiable component of stigma is stereotyping (Corrigan & Watson, 2002). Thus, the degree to which participants endorsed certain stereotypical beliefs regarding schizophrenia was measured using a combination of the Schizophrenia Stereotype Questionnaire (SSQ) and the Schizophrenia Perceived Dangerousness Subscale (SPDS)**.** The SSQ was generated by merging an alteration of Griffiths, Christensen, Jorm, Evans, and Groves' (2004) Personal Depression Stigma Scale (a subscale of their Depression Stigma Scale), a modified version of the Perceived Dangerousness Scale (Link, Cullen, Frank, and Wozniak et al., 1987) and seven novel stereotypical items, specific to schizophrenia which were previously added by Cunningham (2016).

Schizophrenia Stereotype Questionnaire. Several alterations were made to the wording of items from the Personal Depression Stigma subscale by Cunningham (2016) in order to make the questionnaire more schizophrenia-specific. For instance, the statement 'people with depression could snap out of it if they wanted too' was changed to 'people with

schizophrenia could snap out of it if they wanted to' in order to match the current research interests. 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") were also removed, whilst the seventh item in the scale was extended to become two statements for the current research ("If I had suffered from schizophrenia at some point in my past, I would not tell anyone", "I would not tell anyone if I was experiencing symptoms of schizophrenia every day in my present life) rather than one ("If I had depression I would not tell anyone). The SSQ alone comprised a total of eight items.

Schizophrenia Perceived Dangerousness Subscale. Perceived dangerousness (and ultimately stereotypical belief) towards individuals with schizophrenia was measured using the Perceived Dangerousness Subscale (Link et al., 1987). Comparable to the aforementioned measure, the wording of this subscale was modified to make each article more closely related to schizophrenia, for example; "former mental patient", was transformed to, "people with schizophrenia". This scale consists of eight items in total. Responses were made on a 5-point Likert scale ranging from 1, "strongly disagree" to 5, "strongly agree" with higher scores (ranging from 8-40) indicating a higher level of perceived dangerousness towards people with schizophrenia.

Other Items. In addition to the former items, the following seven statements were added to the SSQ by Cunningham (2016). These items were included in order to gather a more comprehensive understanding of respondent's stereotypical beliefs that could not be captured through the items from the measures by Griffith et al., (2004) and Link et al., (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.

Full SSQ. Therefore, the complete SSQ consisted of 23 items in total with responses recorded on a 5-point Likert scale ranging from 1- indicating 'strongly disagree' to 5, indicating 'strongly agree.' Scores on the SSQ ranged from 23 to 115, with higher scores indicating a higher level of stereotyping, and therefore a higher level of stigma. The Personal Depression stigma scale has previously proven to have good internal reliability, with a Cronbach's alpha of .76 (Griffiths et al., 2004) while Link and colleagues reported great internal consistency for their Perceived Dangerousness Scale (α= .85). The reliability of the full SSQ has been demonstrated in past studies, with a Cronbach's alpha of .78 (Cunningham, 2016).

Assessing Prejudice*: the Emotional Reactions Questionnaire***.** The Emotional Reactions Questionnaire (ERQ) was initially created by Angermeyer and Matschinger (2003a) who identified (as mentioned above) that prejudicial reactions often come in three forms; pity, fear and anger. In the current study, the level of prejudice participants held towards individuals with schizophrenia was measured using this scale and therefore an alteration to the wording of several items was completed to make it more specific to research regarding schizophrenia. To illustrate, level of irritation (a measure of anger) was assessed through the following sentence: "I feel annoyed/irritated by people with schizophrenia."

In total the ERQ comprised nine statements, participants responded to each item on a five-point Likert scale ranging from 1- strongly agree to 5- strongly disagree. Scores ranged from 9 to 45 with higher scores relating to higher prejudicial feelings (more stigma). Good internal consistency for all three emotional reaction factors in this scale have been

demonstrated in past work. For example, Angermeyer & Matschinger (2003a) reported a Cronbach's alpha of .79 for the 'fear' component, .74 for the 'pity' component, and .77 for the 'anger' component.

Assessing Discrimination. The current study employed two scales in order to measure respondents reported discrimination towards people with schizophrenia; the Social Distance Scale (SDS)(Link et al., 1987) and the Desire to Control Questionnaire (DCQ)(Cunningham, 2016).

Desire to Control Questionnaire. The Desire to Control Questionnaire was redesigned by Cunningham (2016), to more closely fit a study based around schizophrenia. This was done by taking five statements from the Anti-Coercive Treatment section of the Libertarian Mental Health Ideology Scale (LMHIS)(Nevid & Morrison, 1980) and altering the wording to directly relate to schizophrenia. For example, "Involuntary hospitalization is a necessary procedure in order to provide appropriate treatment for the severely mentally ill" was altered to read: "People with schizophrenia should be hospitalised if their behaviour becomes too strange".

In addition to the items from the LMHIS (Nevid & Morrison, 1980), five novel items were added by to complete 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.

Taken together, the DCQ comprised eight items. Responses were made on a Likert scale ranging from 1- indicating "strongly disagree" to 5- indicating- "strongly agree." Participants score could range from 8- 40, with a higher score suggesting a greater interest in controlling people with schizophrenia, and therefore a higher degree of stigma (in the form of discrimination). The DCQ has demonstrated good internal reliability in past research with a Cronbach's alpha of .73 (Cunningham, 2016).

Social Distance Scale. While the SDS was originally designed by Bogardus in 1925, it was slightly altered by Link et al. (1987) who changed the formatting from Guttman-style to Likert scale responding. It is frequently used in stigma research as a representative measure for behavioural indicators of discrimination against people with mental illness (Penn et al., 1994; Corrigan et al., 2001). In this study it was used to measure the desire for social distance from one specific group: individuals with schizophrenia. Again, some of the wording of this scale was altered by Cunningham (2016) in order to make it more schizophrenia specific. For example, how would you feel about someone with a mental illness being your flatmate?" was changed to "how would you feel about someone with schizophrenia being your flatmate?"

In summary, the SDS totalled seven questions, with participants rating their degree of happiness to socialise on a Likert scale ranging from 1- 'definitely unhappy' to 5- 'definitely happy'. All seven items in the SDS were reverse coded so that a higher score indicated a greater desire for social distance from individuals with schizophrenia. Therefore, a higher score on the SDS reflects a greater level of discrimination and therefore a greater degree of stigma. Scores on the SDS range from 7 to 35. Previous research has suggested good to internal reliability for the SDS with a Cronbach's alpha of .76 (Svensson & Hansson, 2016).

Procedure

Upon entering the testing room, participants sat individually at a desk with a computer to complete the experiment. Each desk had a divider on either side to ensure that participants could not see each other's responses during testing. Testing groups comprised eight or nine participants, whilst the experiment took a maximum of 30 minutes per session. Upon sitting at their desks, participants read through an information sheet stating the purpose of the research- their attention was then directed toward the computer where they were asked whether they agreed to participate in the research, and subsequently began the experiment. The questionnaire in the current study was completed entirely on Qualtrics. Having given informed consent, each participant answered a series of demographic questions regarding age, gender and ethnicity. Further they were required to answer two mental health related questions; 1) have you had any prior experience with somebody with a mental illness? And 2) do you know anyone who suffers from schizophrenia? Following these questions, participants viewed a five minute video clip of a man exhibiting the common symptoms of schizophrenia (delusions, disorganised thought) whilst talking to his therapist. Two content-check questions were asked immediately following the video to ensure participants had paid attention to the contents of the clip.

Participants then read half a page of text concerning the symptoms of schizophrenia and how these symptoms related back to some of the actions from the media clip they previously watched. A randomizer function on Qualtrics then split participants evenly, into one of three experimental conditions; Psychosocial Causal Condition, Biogenetic Causal Condition or Creativity Condition. Participants then read a page worth of text (regardless of condition the vignettes were all a similar length), with the contents varying according to which condition each respondent was in. Participants were then asked to recall which vignette they had read prior to the stigma measures (biogenetic, psychosocial or creative). Irrespective of condition, participants then moved to the final stage of the experiment where they were

required to answer a series of four scales (and one subscale) – the SSQ, SPDS, the ERQ, the SDS and the DCQ – each which measured respondents attitudes towards people with schizophrenia. Questionnaires and response options were presented in the same order regardless of condition. Lastly, respondents were thanked for their participation and handed a debrief form highlighting the purpose of the research, the names and contact information of the researchers, and where to gather more information on mental health and schizophrenia.

Results

Preliminary Analyses

As previous research has demonstrated, a variety of factors can impact the amount of stigma individuals hold towards those with mental health issues (i.e. gender, prior experience with mental illness). Because of this, preliminary analyses were performed in order to assess whether one of our demographic variables - gender - was related to the dependant measures and would therefore need to be included as a covariate in later analyses. The factors of age, personal experience with a mental health issue ($n = 83$, experience with a mental health issues, versus, $n = 14$, no experience) and personal experience with schizophrenia ($n = 83$, no personal experience, $n = 14$, personal experience) were also considered as potential covariates, however, due to a large disparity between sample sizes in the two groups, these preliminary tests were excluded from this experiment.

Gender. Five independent samples t-tests were performed in order to compare the stigma scores of male ($n = 24$) versus female ($n = 72$) participants. For the purpose of these analyses, the one participant who disclosed their gender as 'other' was excluded. The results of these t-tests showed a significant difference on two (the SSQ and ERQ) of the five measures. In regards to the SSQ, in this study, males ($M = 38.42$, $SD = 6.81$) were significantly more likely than their female counterparts ($M = 34.75$, $SD = 4.77$) to hold stereotypical beliefs towards individuals with schizophrenia ($t(94) = 2.91$, p < .001, $d = .60$).

Continuing this trend, analyses on the ERQ revealed that males ($M = 19.79$, $SD = 4.45$) were significantly more likely than females ($M = 16.42$, $SD = 3.71$) to exhibit prejudice attitudes towards people with schizophrenia ($t(94) = 3.66$, $p < .001$, $d = .42$). The t-tests performed on the SPDS, DCQ and SDS all yielded non-significant results. Therefore, gender was found to have a significant effect on two of our measures of stigma and subsequently was included as a covariate in later analyses involving SSQ and ERQ scores.

Primary Analyses: Dependant Variables

Again, I hypothesised that when comparing the results of participants allocated to the non-causal (creative) condition with participants assigned to the causal conditions (biogenetic or psychosocial), that participants in the causal conditions would exhibit lower levels of stigma than individuals not provided any causal information (i.e. those in the creative condition). When directly comparing the two causal conditions, I predicted that those in the biogenetic group would exhibit less stereotypical beliefs (in general) than participants in the psychosocial condition. In contrast to the latter point, I hypothesised that participants in the biogenetic group would display higher levels of perceived dangerousness than the psychosocial group, however no difference on the desire for social distance measure. No predictions were made in regards to the ERQ or DCQ measure.

A total of five one-way between-groups analysis of variance (ANOVAs) were performed with Experimental Condition as the between subjects variable and covariates included as necessary. The aim of these analyses was to determine the effect of experimental condition (Biogenetic Causal Explanation, Psychosocial Causal Explanation and Creative Explanation) on stigma scores across the five dependent measures. Levene's test for equality of variances was performed for each of the five ANOVAs and found not to be violated in all cases. The results were assessed using a standard level of significance ($p < .05$).

Schizophrenia Stereotype Questionnaire. The first analysis of covariance (ANCOVA) investigated whether there were any differences between the Experimental Conditions (Biogenetic Causal Condition, Psychosocial Causal Condition, and Creative Condition) in relation to participant's scores on the SSQ, while controlling for one covariate: gender. Gender was confirmed as a significant covariate by this ANOVA, although this variable only explained six percent of the variance in SSQ scores. After adjusting for gender, this ANOVA revealed a statistically significant difference between experimental conditions, $F(2, 92) = 3.34$, $p = .040$, with a small effect size, ($\eta_p^2 = .06$).

Post-hoc comparisons using Tukey HSD revealed a significant difference between two of our experimental conditions. That is, the mean SSQ score for participants in the psychosocial causal condition ($M = 33.42$, $SD = 4.18$) was significantly lower than the SSQ score for respondents in the creative explanation condition ($M = 37.54$, $SD = 5.83$). Whereas, the mean SSQ score of participants in the biogenetic causal condition ($M = 35.17$, $SD = 5.47$) did not differ significantly from the psychosocial nor the creative condition. In other words, individuals in the creative condition appeared to perceive individuals with schizophrenia in a more stereotypical manner (and therefore a more stigmatising way) when compared to the psychosocial condition, however the level of stereotypical belief exhibited by participants in the biogenetic condition did not differ significantly from the former two groups. This result partially confirms my prediction that individuals not provided a causal explanation (i.e. the creative condition) would likely view patients with schizophrenia in a more stigmatising way than those in the causal explanation conditions (biogenetic, psychosocial). However, contrary to my initial predictions, participants in the biogenetic condition did not display significantly lower levels of stereotypical belief than participants in the psychosocial condition.

Schizophrenia Perceived Dangerousness Subscale. A second ANOVA was performed to analyse whether there were any differences between Experimental Condition

and participants' SPDS scores. No significant differences were found between conditions in this case ($F(2, 94) = 1.47$, $p = .236$, $\eta^2 = .03$). That is, the reported perceived dangerousness of individuals with schizophrenia did not vary significantly based on experimental condition. This did not support my initial hypothesis that there would be a significant difference between the level of perceived dangerousness in the biogenetic condition and psychosocial condition, with those in the biogenetic condition exhibiting higher levels of perceived dangerousness.

Emotional Reactions Questionnaire. A second ANCOVA was performed, this time to examine the potential difference between participant's scores on the ERQ and their experimental condition, while controlling for the covariate of gender. Gender was confirmed as a significant covariate by this ANCOVA, although this variable only explained eight percent of the variance in scores on the ERQ. After adjusting for gender, this analysis revealed no significant differences in ERQ scores between experimental conditions ($F(2, 92) = 1.82$, $p = .096$, $\eta_p^2 = .05$). This suggests that participants emotional reactions (measure of prejudice) concerning individuals with schizophrenia were not affected by the experimental group they belonged too (i.e. Biogenetic, Psychosocial, Creative).

Desire to Control Questionnaire. A further ANOVA was conducted to assess whether there were any differences between experimental condition and participants scores on the DCQ. This analysis revealed no significant differences between the three conditions, $F(2, 94) = 1.48$, $p = .232$, $\eta^2 = .03$. That is, participant's discriminatory attitudes (as assessed by reported desire to control) regarding individuals with schizophrenia were not affected by experimental condition.

Social Distance Scale. A fifth ANOVA was calculated to examine potential differences between the experimental conditions in relation to participant's scores on the SDS. Again, this ANOVA revealed no significant differences in scores on the social distance

scale between experimental conditions ($F(2, 94) = 1.21$, $p = .302$, $\eta^2 = .03$). That is, regardless of the explanation participants were provided (i.e. biogenetic, psychosocial, creative) their reported desire for social distance from individuals with schizophrenia did not differ. [2]

Discussion

Experiment One explored the following three research questions: First, does teaching participants a causal explanation of schizophrenia reduce stigma compared to a novel, non-causal explanation? Second, is there a significant difference in overall stigma levels when comparing the results of a biogenetically based educational intervention with a psychosocial educational intervention? And lastly, can we find an untested explanation for schizophrenia in the literature that is more effective at reducing stigma than the current causal explanations?

In regards to our first research question, the results from this experiment suggest that the nature of information provided to participants can effect stigma levels to a small degree. More specifically, it can be presumed that when individuals are provided an etiologically based explanation for schizophrenia which emphasises psychosocial factors, they are less likely to hold stereotypical beliefs, and therefore, less stigmatising views towards people with schizophrenia, than individuals who are provided no causal reason. This finding is partially consistent with my initial predictions that participants in the causal conditions would report lower levels of stigma than the creative group, who were not provided any causal information. However, it seems this effect may be rather small, whereby, the effectiveness of causal explanations at reducing stigma appears limited to the 'stereotype' component, making no difference to participant's levels of prejudice or discrimination.

In light of this point, no differences between stigma scores were reported on the measures of prejudice (as measured by the ERQ) or discrimination (as measured by the DCQ

---

[2] A series of factorial ANOVAs were conducted and confirmed the above results.

and SDS) among our three experimental conditions. This suggests that these factors of stigma are relatively unaffected by the nature of information (whether causal or not) provided to participants. Again, this contrasts my initial predictions that stigma levels would be higher across measures for individuals in the non-causal, creative condition. Ultimately, my preliminary conclusion is that providing etiological information (specifically psychosocial information) is an effective method of stigma reduction in regards to stereotypical belief, however not prejudice or discrimination.

Furthermore, these results suggest that biogenetic, psychosocial or creatively based explanations for schizophrenia are unable to produce significant stigma reducing effects on multiple components of stigma. This leads me to believe that the contents of the information participants are provided in regards to schizophrenia (whether it be causal or non-causal) makes very little difference to overall stigmatizing beliefs and attitudes toward that group.

The results from Experiment One did not support my prediction that providing a biogenetically based causal explanation for schizophrenia is any more (or less) effective than a psychosocial explanation in terms of stigma reduction. That is, there was no difference among the stigma scores of participants in the two causal conditions on any of our dependant measures, suggesting the actual nature of causal information does not have a significantly different impact on stigma levels towards schizophrenia. These results raise the question as to whether causal explanations actually do have varying effects on the different components of stigma as previous research suggests (e.g. Lincoln et al., 2008; Schlier et al., 2014).

Further and in response to the suggestions made by Schlier and colleagues (2014) regarding the potential effectiveness of non-etiologically based explanations of mental illness on stigma reduction, it appears that teaching a non-causal explanation of schizophrenia (centred on why schizophrenia exists today), is no more effective at lowering stigma than providing information that is causal in nature, in fact, it may exacerbate it. In regards to this

point, whilst the creative vignette discussed a popular, yet hypothetical idea, that schizophrenia exists in society today due to the various advantages it holds, it should be considered that perhaps causal explanations fare better in terms of stigma reduction due to the legitimacy of information provided by them. That is, while respondents in the creative condition read a relatively positive exert regarding the presence of schizophrenia globally, it may not have been convincing enough for them to believe the information in it - therefore leading to higher scores on the SSQ. This would be unsurprising given the study population comprised psychology students, and therefore it could be presumed that the majority of these individuals had some prior knowledge about schizophrenia. Given that the creativity-schizophrenia link is relatively unexplored and under researched, the likelihood that participants had never heard schizophrenia being discussed as an evolutionary adaptation is high. As a result, participants in this condition may have found the vignette information incomprehensible or far-fetched (compared to those in the causal conditions), therefore disregarding it and subsequently retaining the original stigmatising attitudes they entered the experiment with (i.e. their attitudes remained unchanged by the manipulation). As has been noted several times in the literature, the contents of the educational intervention is critical for predicting attitude change, and subsequently how much stigma is reduced (Rüsch et al., 2005). In this case, it appears utilising information which is causal in nature is slightly more appropriate if stigma reduction is the end goal.

Whilst we can acknowledge that to a small degree, psychosocial educational interventions exhibit positive stigma reducing powers (compared to being provided with no causal information), the zero significant differences found among the stigma scores of individuals in the biogenetic and psychosocial conditions contradict a plethora of previous research in this field which suggest the effects of causal explanations on stigma vary, according to which aspect is being assessed (Boysen & Vogen, 2008; Lincoln et al., 2008;

Walker & Read, 2002). This finding could possibly be attributed, at least partially to the fact that participants from the current sample were all enrolled in a first year psychology class and therefore may have already held less stigmatizing beliefs towards individuals with mental illness; as several studies have demonstrated, an increased mental health literacy (and therefore knowledge about mental illness) often means less stigmatizing views (Thorsteinsson, Bhullar, Williams & Loi, 2018). Thus, it is possible that stigma scores between causal conditions were less pronounced than they would be in a study conducted on the general population (non-psychology students) due to floor effects. A further possible explanation for this null finding is that, as mentioned above, the majority of our sample (being psychology students) likely already had a good understanding about the nature of mental illness, and presumably, held some pre-existing beliefs about the causes of schizophrenia specifically. As a result of this knowledge, it is possible that participants were already aware of the actual causes of schizophrenia, and therefore a) rejected the information provided in their condition, or b) went in with a pre-existing belief as to the cause of schizophrenia, and therefore, the opinions and attitudes they entered the experiment with remained unchanged by the explanation provided in the vignette. In connection with the above points, I acknowledge an obvious limitation of Experiment One was the assumption that participants would believe and further accept the information provided in their respective vignette.

A further limitation of Experiment One was the absence of a true control group. Whilst the novel creativity condition did allow us to compare the questionnaire scores of participants who were provided no causal information with participants who were provided causal explanations, we did not have any individuals who were part of a 'no intervention' condition. Therefore, there was no true baseline stigma score of participants who were not provided any information about schizophrenia (whether causal or not) to evaluate our results

against. Thus, we cannot say with complete confidence that the information in these vignettes had little to no effect on stigma, as we could not compare the stigma scores of people in the experimental conditions, with a group who read no information on schizophrenia at all (other than symptomology).

To address the aforementioned issues, a second experiment was designed which followed a similar paradigm to experiment one, however, with a number of crucial alterations. First, to address the issue that participants may not have believed the information provided in their vignettes, Experiment Two included an additional manipulation check, in the form of a 'convincingness rating'. That is, participants were asked, "how convinced were you that the explanation you read was factual and true?". As stated, Experiment One provided no indication as to whether participants actually accepted, the causal (or non-causal) information they had read. If participants were not entirely convinced by this explanation, the experimental manipulation may not have worked, subsequently having no effect on stigma, and thus the results gathered from this first study would be thrown into question. Through this addition, it was assumed that if respondents in Experiment Two did rate their vignette explanation as 'convincing' this could reassure us that participants likely did believe the explanation they read, and therefore assure us that the results garnered regarding the effects of different explanations on stigma, were accurate. Further, to account for the absence of a control group, Experiment Two employed a fourth, 'no information' condition. The premise being this would allow for a comprehensive comparison of stigma scores between groups who received an explanation of schizophrenia, and those who did not in order to give us a true assessment regarding the effects of our manipulation.

Finally, accounting for the significant finding in this experiment which suggested the creativity vignette actually resulted in an increase in stigma (or stereotypical beliefs to be exact) when compared with the causal explanations, this condition was dropped from

Experiment Two, and a novel, causal condition (based on epigenetics) was introduced in its place. As previously discussed, the literature constantly suggests that teaching people something about the causes of a mental illness has a positive effect on stigma levels. Thus, considering one of the aims of the current study is to find a novel explanation of schizophrenia which successfully reduces stigma (rather than increasing it), the addition of a previously untested causal condition seems to be the most logical step forward in the research. This idea will be discussed in more detail below:

Experiment Two

Experiment Two was conducted to investigate the same three research questions as Experiment One, however with a more robust methodology (e.g. the inclusion of a control group and convincingness rating) and the addition of a new causal explanation for schizophrenia. More specifically, I asked; does teaching participants a causal explanation for schizophrenia reduces stigma compared to no explanation at all? Is there a difference in the stigma reduction of biogenetic and psychosocial causal explanations for schizophrenia? And finally, is there an untested explanation for schizophrenia that is more effective than the current psychosocial or biogenetic causal explanations at reducing stigma?

In relation to this last research question, Experiment Two proposed that rather than using a biogenetic or psychosocial causal account for schizophrenia, an epigenetic explanation- which stresses the role of the environment on the expression of an individual's genetic code - may be more effective at reducing negative attitudes and subsequent stigma surrounding mental illness (Lebowitz, Ahn & Nolen-Hoeksema, 2013). Although schizophrenia is a heavily researched disorder, epigenetics offer a fresh direction for further study and understanding surrounding this illness. Therefore, it also offers a new avenue through which to study potential educational resources for stigma reduction. So what exactly is 'epigenetics'?

Epigenetics refers to changes which affect gene activity and expression. In terms of schizophrenia, this implies that the illness results from a complex interaction between external environmental influences and genetic predispositions (Roth et al., 2009). In simpler terms, according to epigenetics, although genes are associated with the development of schizophrenia, the presence of these genes does not necessarily mean they will be 'expressed' by the individual. This individual's external influences and interaction with aggravating environmental factors (e.g. diet, disease exposure, amount of exercise) ultimately determine whether these genes are expressed or not, and thus, whether schizophrenia develops in a certain individual.

Despite the growing relevance of epigenetics and schizophrenia in recent research, epigenetic causal explanations for schizophrenia have not yet been tested as an educational method of stigma reduction. Taking this into account, no other studies so far have directly compared the effects of a biogenetic, psychosocial and epigenetic causal explanation, on multiple components of stigma. Therefore, this will be a primary aim of Experiment Two. This second experiment uses a similar experimental methodology to Experiment One-however, a fourth condition (a control group) was introduced, one condition was changed (from a creative vignette- to a causal, epigenetic vignette) and a manipulation check was added to ensure participants actually accepted the information they had read in their respective conditions.

In Experiment Two, participants viewed a five minute video of a patient with schizophrenia being interviewed by a psychiatrist, read one of three informative explanations for schizophrenia (in comparison to experiment one however, all explanations were causal in nature) or no explanation if they were in the control condition, and then completed the same questionnaires assessing levels of stereotypical belief, prejudice and discrimination. In contrast to Experiment One, a 'Convincingness Rating' was added at the end of the second

experiment. This rating allowed for an accurate evaluation as to whether participant's stigma had changed as a result of their experimental manipulation (as opposed to other factors) by confirming they believed the information in the vignette was credible.

Hypotheses

For Experiment Two, consistent with past research (e.g. Lincoln et al., 2008) I hypothesised that teaching a causal explanation for schizophrenia (regardless of which one) would result in a reduction in stigma scores when compared to the control condition. Secondly, comparable to Experiment One, I predicted that biogenetic and psychosocial explanations would affect stigma differently (i.e. increasing it on some measures, while decreasing it on others). Although the results of Experiment One did not confirm these initial predictions, in line with a plethora of previous research which suggests causal explanations have varying effects on different components of stigma, I retain my initial predications regarding the outcome of stigma scores on each measurement. That is, I predict scores on the SSQ will be higher in the psychosocial condition when compared to those in the biogenetic condition. Further, I hypothesise that scores on the SPDS will be higher in the biogenetic condition than those in the psychosocial condition. Again, considering the null findings from experiment one on the ERQ and DCQ I make no predictions regarding these measures. Finally, as the epigenetic causal explanation is previously untested in the stigma literature, I make no prediction as to how this variable will effect stigma when compared to the alternative two causal conditions (psychosocial and biogenetic).

Method

Participants

One hundred and fourteen psychology students enrolled in a first-year, undergraduate psychology paper completed a 30-minute experiment in exchange for course credit. Three participants were excluded for answering one of the first two control check questions

incorrectly, suggesting they had paid inadequate attention to the video material. A further six participants were removed for inaccurately identifying which causal explanation they had read. The final sample comprised 105 participants, of these, 14 (13.3%) were male, and 91 (86.7%) were female. Ninety-nine percent of the sample were within the age bracket of 17-22. The average age of this final sample was 18.87 ($SD = 3.65$). Comparable to the first study, a large percentage of respondents (78.1%). identified their ethnicity as New Zealand European, while a minority of participants claimed to know someone with schizophrenia 16.2%) no participants disclosed that they suffered from the illness themselves. A large majority of this sample reported having some kind of experience (personal, family or friend, in the community) with a mental health issue (92.4%). No further demographic information was recorded.

Participants were randomly assigned to one of four conditions via a randomizing function in the Qualtrics software (Biogenetic Causal Explanation Condition, Psychosocial Causal Explanation Condition, Epigenetic Causal Explanation Condition and Control Condition). Following the exclusion of those who answered the control check questions incorrectly, 27 participants were left in the biogenetic condition, 27 in the epigenetic condition, 27 in the control condition, and 24 in the psychosocial condition.

Materials

Demographic Information. Demographic information was collected as in Experiment One.

Video Clip. Participants in Study Two watched the same five minute video clip as those in the first experiment.

Content-Check Questions. As with the first experiment, participants were asked a total of three content check questions over the duration of the experiment to evaluate whether they had paid sufficient attention to both the written and video materials. The first set of

questions were identical to those used in Experiment One and were created to test participants memory on simple details discussed in the video clip (See appendix A for answer options).

The third and final content check question assessed whether participants could recall which causal explanation they had read (i.e. epigenetic, biogenetic or psychosocial) and therefore whether they had paid sufficient attention to our experimental manipulation. This question directly followed the presentation of causal explanations. The wording of this question was altered in comparison to the first experiment, in an attempt to make clearer what was being asked. That is, other than simply asking participants to recall the causal or evolutionary (creative) information they had read. I added a note stating, "Please make sure you answer according to which explanation you just read NOT your personal beliefs on the matter." As it was speculated that perhaps so many participants failed this content check question in the first experiment due to a potential misinterpretation of what was being asked. In other words, rather than stating which condition they actually belonged to, a number of them may have answered according to their actual beliefs on the matter - this would account for the large number of participants who were removed from the analyses (due to answering incorrectly) in the creative condition, but not the psychosocial or biogenetic condition. In response to this question, participants could choose from three possible answer options, 'Schizophrenia is caused by Biology (genetics)', 'Schizophrenia is caused by Psychosocial Factors (trauma, problematic childhood, and stress)' or 'Schizophrenia is caused by Epigenetics (a combination of environmental AND biological factors)'. Participants in the control condition were unable to see the final content-check question as they did not read a causal explanation; rather, these participants proceeded directly on from reading the general information regarding symptoms of schizophrenia, to completing the stigma scales. As in the first experiment, if respondents answered any of these questions incorrectly, their response

was excluded from the analyses. As mentioned above, nine participants were excluded from further analyses for this reason.

Information regarding schizophrenia. Regardless of condition, every participant in Experiment Two read an exert explaining the main symptoms of schizophrenia, this was identical to the information provided in Experiment One. Please refer to Appendix B to view this material.

Causal Explanations. Participants assigned to a causal condition read information detailing either an epigenetic, biogenetic or psychosocial explanation for the disorder. The causal information provided differed according to condition. The contents of the psychosocial and biogenetic vignette were identical to Experiment One. A full copy of these explanations is provided in Appendix C.

The epigenetic causal explanation was created in the same way as the previous two vignettes, and detailed the way epigenetics contributes to the development of schizophrenia, stressing how specific life choices and experiences (e.g. diet, social interactions, drugs, and exercise) can influence the expression of certain genes, and ultimately make an individual more or less vulnerable to developing schizophrenia. All three explanations were similar in length (approximately one page worth of text). See appendix C for a full copy of this vignette.

Scales Used. Participants completed the exact same four scales (and one sub scale) as used in Experiment One. Stereotypical belief was measured using the SSQ (and the SPDS subscale), prejudice was measured using the ERQ, and discrimination was measured with the DCQ and SDS (see Appendix D for a full list of all scale items).

Convincingness Rating. Experiment Two finished with a question asking participants to rate how convinced they were by the causal explanation they had read (i.e. that biogenetic factors, psychosocial factors, or epigenetic factors contribute to the development

of schizophrenia). Responses were recorded on a Likert scale ranging from 1 – indicating, 'very convinced', to 4 – suggesting, 'not at all convinced'. For participants in the control condition, this question was not presented; therefore, these participants proceeded directly from the stigma measures, to the end of the survey.

Procedure

A similar procedure was followed for Experiment Two, as in Experiment One, the crucial difference being the removal of the creativity vignette and the subsequent addition of the epigenetic vignette, a control condition and convincingness rating. Again, Qualtrics was the programme used to record participants responses. As in Experiment One participants sat computers separated by partitions. They first answered a series of demographic questions, followed by viewing a video clip of a man with schizophrenia talking to a psychiatrist. After the video material, participants were asked content-check questions regarding the material in the clip, and then read a page worth of text detailing the symptoms of schizophrenia. Participants were then randomly assigned to one of four experimental conditions, where they read a text exert detailing a biogenetic causal explanation for schizophrenia, a psychosocial causal explanation for schizophrenia, or a causal explanation for schizophrenia based on epigenetics (based on the condition they had been assigned too). Following the presentation of these explanations, participants from the three causal conditions were asked a final multi-choice content check question, in order to assess whether they had paid adequate attention to the contents of the vignettes. Alternatively, participants assigned to the control condition did not read any information concerning the causes of schizophrenia, and therefore were not required to answer the third and final content-check question. Rather, these participants continued directly from the text exert about the symptoms of schizophrenia, to completing the stigma measures. Following this split into different conditions, the method remained the same as in Experiment One, with the exception of a 'convincingness rating' where, following the

completion of the stigma measures, participants from the causal conditions were required to choose how convinced they were by the causal information they had read.

Results

Preliminary Analyses

Preliminary analyses were conducted in order to assess whether one of our variables – whether individuals knew someone with schizophrenia – was related to any of the dependant measures and would therefore need to be considered as a covariate in later analyses. Five independent samples t-tests were performed to compare the stigma scores of participants who disclosed they knew somebody (i.e. family or friend) with schizophrenia ($n = 17$), and those who said they did not ($n = 88$). The results revealed that this variable had no effect on stigma scores and therefore was not included as a covariate in later analyses. Three further variables – age, gender, and previous experience with a mental health issue - were also considered for the same analyses, however due to a large disparity between sample sizes amongst these groups ($n = 97$, versus $n = 8$ for both variables) these tests were not conducted.

Primary Analyses: Dependent Variables

The dependant variable in this study were scores on the SSQ (and the SPDS subscale), the ERQ, the DCQ and finally, the SDS. A series of one-way between groups analyses of variance (ANOVAs) were conducted with Experimental Vignette as the between subjects variable. Levene's test for equality of variances was conducted and found not to be violated in all cases. All ANOVA results were assessed using a standard level of significance ($p < .05$).

Schizophrenia Stereotype Questionnaire. The first ANOVA examined whether there were any differences between the four experimental conditions (biological explanation condition, psychosocial explanation condition, epigenetic explanation condition and control condition) based on participants scores on the SSQ. The results of this ANOVA revealed a

difference in SSQ scores between conditions which approached significance ($F(3,101)$ = 2.51, p = .063), with a small effect size (η^2 = .07).

Post-hoc comparisons using Tukey HSD revealed an almost significant difference between two of our conditions. That is, the mean SSQ score for those in the control condition (M = 36.56, SD = 4.67) was higher than those in the biogenetic condition (M = 33.26, SD = 3.36). However, the mean score on the SSQ for participants in the psychosocial condition (M = 35.33, SD = 5.20) and the epigenetic condition (M = 35.83, SD = 5.12) did not differ significantly from any of the other conditions. In other words, participants in the control condition had more stereotypical beliefs regarding people with schizophrenia than participants in the biogenetic condition, however, the SSQ scores of individuals in the psychosocial and epigenetic conditions did not differ significantly from each other, nor the biogenetic or control condition. These results do somewhat support my hypothesis, that those in any of the causal explanation conditions would exhibit a lesser amount of stereotypical beliefs towards people with schizophrenia when compared with those in the control condition. However, these results do not confirm my predictions that when compared with the psychosocial condition, those in the biogenetic causal explanation condition would show a reduced amount of stereotypical belief.

Schizophrenia Perceived Dangerousness Subscale. A second ANOVA examined whether there were any differences in participants scores on the SPDS, based on their experimental condition. The results of this ANOVA revealed no significant differences between any of the four conditions ($F(3,101)$ = 2.03, p = .114, η^2 = .06). In other words, the experimental condition participants belonged to did not affect their reported perceived dangerousness of individuals with schizophrenia. This goes against my initial predictions - that those in the biogenetic condition would rate people with schizophrenia as significantly more dangerous than those in the psychosocial condition.

Emotional Reactions Questionnaire. A third ANOVA analysed whether there was any difference between experimental conditions in regard to participant's scores on the ERQ. The results from this ANOVA revealed no significant differences in ERQ scores between the four experimental conditions ($F(3,101) = 1.21$, $p = .311$, $\eta^2 = .04$). Again, this suggests that participants emotional reactions (prejudice attitudes) regarding individuals with schizophrenia was not affected by the experimental condition they belonged too. This refutes my initial predictions that participants in the causal conditions would display lower scores on our measures of stigma than the control condition.

Desire to Control Questionnaire. A fourth ANOVA analysed whether there was any difference between experimental conditions in regards to participant's scores on the DCQ. Again, the results from this ANOVA revealed no significant difference between conditions ($F(3,101) = 2.40$, $p = .072$, $\eta^2 = .06$). This suggests that respondents desire to control individuals with schizophrenia did not differ based on the experimental group they belonged too. This did not support my initial hypothesis, that participants in the control condition would score more highly on the DCQ (therefore showing higher levels of discrimination) than all of our causal conditions.

Social Distance Scale. The fifth and final ANOVA examined whether there were any differences between the experimental conditions in terms of participants' SDS scores. This ANOVA revealed a difference which approached significance, $F(3,101) = 2.55$, $p = .060$, with a small effect size, ($\eta^2 = .07$).

Post-Hoc comparisons using Tukey HSD tests revealed no significant differences between the four experimental conditions. In other words, participants reported desire for social distance from individuals with schizophrenia, in the control group ($M = 19.78$, $SD = 4.13$) biogenetic group ($M = 23.15$, $SD = 5.63$), psychosocial group ($M = 22.41$, $SD = 5.32$) and epigenetic group ($M = 22.75$, $SD = 5.32$) did not differ significantly from one another.

This result does not support my hypothesis that individuals in the control group would reportedly score higher on our measure of desire for social distance (and therefore show more discrimination) towards individuals with schizophrenia than participants in any of our causal conditions.

Additional Analyses: Convincingness Ratings

At the end of the experiment, all participants from causal conditions provided a rating of how convinced they were by the causal information they read. Ratings were made on a four-point Likert scale with 1, indicating 'Very Convinced', 2 indicating 'Somewhat Convinced', 3 indicating 'Not Very Convinced,' and 4 indicating 'Not at All Convinced.' It was predicted that participants from the causal conditions would rate the passage they read as 'very convincing. Across all experimental conditions, the mean rating of convincingness was 1.88. 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, $t(77) = -6.26$, $p < .001$. This suggests that overall, participants in Experiment Two found the causal explanations to be highly credible.

Conclusion

The results of this second experiment show a number of interesting findings. Firstly, participants in the control condition reported higher levels of stereotypical belief than respondents in the biogenetic condition (this finding approached significance). However, there was no difference in reported stereotypical belief between any of the causal conditions, nor the control condition and the epigenetic and psychosocial condition. Essentially, this indicates that providing people with a biogenetic causal explanation for schizophrenia has some degree of effectiveness in terms of reducing stereotypical beliefs (and subsequently

stigma) when compared with providing no information in regard to the causes of a mental disorder at all.

Secondly, and contrary to my initial expectations, providing individuals with etiological information regarding schizophrenia (compared with no information), did not make people any less stigmatising on measures of prejudice or discrimination, when compared with participants who were provided no information as to the root causes of this illness. This indicates that - other than biogenetic causal explanations exhibiting some ability to reduce stereotypical attitudes towards people with schizophrenia - the three etiological explanations provided, failed to successfully reduce all three components of stigma together (stereotypical belief, prejudice and discrimination), and therefore assumedly are not very effective methods of overall stigma reduction.

Additionally, inconsistent with my initial predictions, there was no difference between the stigma scores of participants in any of our causal conditions. This finding contradicts a wealth of previous research in this area which posits that the effectiveness of causal information on stigma reduction varies, according to which component is being measured (e.g. Lincoln et al., 2008; Schlier et al., 2014). This result further contrasts the claims made by researchers such as Walker and Read (2002), who suggested that biogenetic conceptions of schizophrenia lead to an increase in stigma compared to psychosocial explanations. Rather, the biogenetic explanation was found to reduce stereotypical belief compared to the control condition (a result which the epigenetic and psychosocial condition failed to achieve). In addition, the analyses of participants convincingness ratings validates my expectations that the causal explanations provided in this study were credible and believable enough to be accepted by participants, and therefore the lack of support for the aforementioned hypothesis, and subsequent lack of differences between stigma scores of the causal conditions cannot be attributed to an unpersuasive experimental manipulation. Thus, it would appear that one

causal explanation is not significantly more effective than the other, however a biogenetic causal explanation is more proficient at reducing stimga (specifically stereotypical belief) compared to reciving no causal information at all.

Taking the above findings into consideration, one can presume our results are rather complex. The second experiment supplied no evidence that providing any kind of causal information (compared to no information) regarding schizophrenia could successfully reduce prejudice, or discrimination, two of the three key elements that constitute stigma. More specifically, and contrary to previous research (Boysen & Vogel, Lincoln et al., 2008; Schlier et al., 2014), our results suggest that providing psychosocial causal explanations of schizophrenia fail to reduce any element of stigma compared to no information at all (stereotypical beliefs, prejudice, and discrimination). In this respect, biogenetic explanations may be perceived as slightly more effective at reducing stigma, as the biogenetic explanation in this experiment reduced stereotypical belief toward schizophrenia (compared to no information) whereas the psychosocial condition did not. In addition to previous research - the novel component in the current experiment, the epigenetic vignette, produced no better stigma reducing effects than being provided no information as to the causes of the disorder at all.

Unfortunately, this result goes against one of the primary aims of this study- to find an educational explanation for schizophrenia which has not yet been tested in the literature that can reduce stigma more effectively than the current causal models. Accounting for the aforementioned results, my preliminary conclusion then is that providing causal information is a relatively ineffective means of reducing overall stigma.

General Discussion

 Across two experiments, the current project investigated three key research questions. First, I asked whether teaching participants a causal explanation for schizophrenia

reduces stigma compared to a novel, non-causal explanation, or no explanation at all. Second, I asked if there was a difference in the stigma reduction of our psychosocial and biogenetic causal conditions. That is, was one of these causal explanations clearly more effective than the other? Third, I asked whether we could find an untested explanation for schizophrenia in the literature (whether causal or not) that is more effective than the current psychosocial or biogenetic causal explanations at reducing stigma?

Findings from Experiment One suggested that presenting etiological information, specifically psychosocial information, exhibits a slim degree of efficacy in regard to reducing stigma toward schizophrenia when compared with novel, non-causally based information. More specifically, in the first experiment, a psychosocial causal explanation resulted in significantly less stigma (in the form of stereotypical belief) towards individuals with schizophrenia, compared to individuals who read information that was creative (and therefore non-causal) in nature. However, no difference was found between the stigma scores on our biogenetic or psychosocial causal measures, which in contrast to my initial predictions and a wealth of previous research (e.g. Boysen & Vogel, Lincoln et al., 2008; Schlier et al., 2008), suggests that different etiological explanations, do not have different effects on stigma.

Findings from Experiment Two suggested that etiological interventions, specifically biogenetic causal interventions, are slightly more effective at reducing stigma (stereotypical belief) when compared to receiving no information regarding the causes of a mental illness at all. However, comparable to Experiment One, it appears that no one causal explanation is significantly more proficient than another causal explanation at reducing stigma, as there were no significant differences found between the stigma scores of the epigenetic, biogenetic and psychosocial causal conditions.

Taken together, our findings suggest some interesting things. First, it appears as though providing individuals with causal information about schizophrenia, is slightly more

effective at reducing stigma, than providing people with no information at all, or alternatively, no-causally based information. This statement is particularly relevant for biogenetic and psychosocial explanations, as across both experiments, at least one of these two conditions reduced stigma amongst participants compared to being provided no causal information at all. Further, this finding partially confirms my initial hypotheses for both studies- that individuals provided with causal information would show reduced levels of stigma compared to individuals not provided any causal explanations (i.e. the creative and control groups). This result also coincides with that of previous research by both Boysen & Vogel (2008) and Lincoln et al. (2008) specifically, who reported evidence that teaching a causal explanation of mental illness can lead to a reduction in stereotypical beliefs toward schizophrenia.

However, it appears that teaching causal information (compared to no causal information) has limited benefits in terms of stigma reduction. That is, contrary to my expectations, there was no significant differences in reported discrimination or prejudice between our experimental groups and control/ creative group for both experiments. This leads me to believe that these aspects of stigma are relatively unaffected by causal information, and thus casts doubt on the overall efficacy of promoting etiological information as a means to reduce stigma. As we know from previous research, despite years of promoting biogenetic and psychosocial causal explanations in an effort to reduce the stigma associated with schizophrenia, levels of public stigma remain relatively unchanged (Link et al., 2004). Bearing in mind the results of this study, which only reported a reduction in one component of stigma as a result of causal explanations (stereotypical belief), it is possible that perhaps causal explanations have no, or very little effects on prejudice or discriminatory attitudes. Therefore, possibly different methods to combat stigma, unrelated to the etiology of

schizophrenia (and apparently, creativity), are required to produce successful results which alleviate every component of stigma, not just one.

This point raises another interesting concern- what kind of information is the most effective in terms of stigma reduction? It is clear more research is required in order to definitively determine what information should be included in order to produce successful anti-stigma interventions. Whilst the results of this study suggest causal information is not the most comprehensive method of stigma reduction (successfully reducing one component of stigma, however failing to reduce the remaining two) the first experiment also tested a novel, non-causal explanation of schizophrenia on stigma reduction. This creative condition failed to produce any reductions in stigma compared to our causal conditions, in fact, it produced a significantly higher degree of stereotypical belief among participants in comparison to the psychosocial condition.

Although this information did not perform as we hoped, it was anticipated that perhaps by providing a novel causal model (epigenetics) in the next study, given the positive findings in the literature regarding teaching information that is causal in nature, we could find a positive stigma reducing result. However, when comparing the results from our novel epigenetic causal condition with all other conditions, including the control, no difference was found on any of our measures of stigma. Therefore, raising the question as to what type of educational information is the most appropriate and effective for creating successful anti-stigma interventions, as both causal and non-causal explanations in the current study have shown limited, if any, effects on stigma reduction.

It is possible that perhaps a multi component approach is most appropriate, which incorporates several different types of information, rather than focusing on only one educational theme. This makes sense when we remember that stigma comprises several

different components and therefore multiple forms of educational information are probably required in order to reduce all facets of stigma.

Further, and in contrast to my initial predictions, the current study provided no evidence that one causal explanation is more effective than another based on what component of stigma is being measured. Rather, across both experiments, all causal explanations were found to be equally as effective (or not effective) as one another. Again, this contradicts an abundance of previous research which differentiates between the effectiveness of different causal explanations on different measures of stigma (Lincoln et al., 2008; Schlier et al., 2014). For example, as discussed, Schlier and colleagues (2014) found that blame was reduced amongst participants after they were taught a biogenetic explanation for schizophrenia, however this same element of stigma increased when participants were taught a psychosocial explanation. Contrastingly, Walker and Read (2002) found that levels of perceived dangerousness increased following a biogenetic causal explanation of schizophrenia, however, these levels remained unchanged in the psychosocial condition from their sample. This finding is also inconsistent with a plethora of previous research which has analysed the efficacy of causal models individually, and repeatedly indicated that different elements of stigma are enhanced and diminished by different causal explanations (Angermeyer et al. 2011; Jorm & Oh 2009; Kvaale et al. 2013a, b). This result casts further doubt on the efficacy of certain etiological explanations at reducing stigma, and further reiterates the question - what educational information should be included in successful anti-stigma programmes in order to see a significant reduction in stigma?

In summary, the current research provided minimal support for my hypothesis that teaching participants about the causes of schizophrenia is an effective method of stigma reduction. More specifically, while both the biogenetic causal model and psychosocial causal explanation of schizophrenia reduced one component of stigma -stereotypical beliefs. They

failed to have any destigmatising effect on the latter two components of stigma- prejudice and discrimination. There was no evidence to support my hypothesis that when comparing the stigma scores of participants in our three causal conditions, one type of explanation would reduce stigma more effectively than the other. Thus, it is my preliminary conclusion that while providing etiological information about schizophrenia may be successful at reducing one component of stigma, when accounting for the remaining two unaffected elements (prejudice, discrimination) of stigma, it is an ineffective method of overall stigma reduction. While there may be benefits of causal teaching on one element of stigma, the present finding suggests that etiological information is unable to successfully reduce all components of stigma, and therefore when causal information is presented alone, it is not an effective means of overall stigma reduction. In addition, I found no evidence that presenting a novel explanation for schizophrenia (epigenetics and creativity) was an effective way to lower stigma, thus it appears future work is still plagued with the difficult task of finding a method of stigma reduction which can reduce multiple aspects of stigma, not just one.

Strengths

The large sample sizes across both experiments can be viewed as a major strength of this study. A further strong point of the current research was the introduction of two novel explanations for schizophrenia. One being causal in nature (epigenetic condition) and one focusing on why schizophrenia is around as opposed to how it comes about. The majority of previous research has been concentrated on psychosocial and biogenetic explanations for schizophrenia, often producing mixed results in terms of the effectiveness of both of these explanations at reducing stigma. The use of a novel creative condition in experiment one should be considered a strength of the current study, where, to the best of my knowledge, no previous research has examined the potential stigma-reducing effects of a teaching like this.

That is, while acting as somewhat of a control condition to compare the effects of causal explanations of schizophrenia with non-causal explanations of schizophrenia, this also offered a fresh avenue through which to explore alternative information types to reduce stigma. And further, to substantiate the claims in the literature that educational information which stresses the etiology of a mental illness, is one of the more effective methods of stigma reduction toward mental illness. The creation of this first vignette can essentially be seen as a response to the claims of Lincoln et al., (2008) who reported that information that makes schizophrenia seem more understandable and less intimidating appears to be the most helpful at reducing stigma.

Moreover, the use of another novel, causal explanation for schizophrenia (epigenetic) is a clear strength of the current investigation. Whilst previous research has compared the effects of a diathesis-stress model, a psychosocial causal model and a biogenetic causal model (Schlier et al., 2014; Walker & Read, 2002) on stigma reduction. No previous research has investigated the potential destigmatising aspects of providing an epigenetic causal explanation for schizophrenia. Previous theory has suggested that educating people about the causes of an illness should result in a reduction in stigma, however, as is clear from our study and previous research before that, the actual effectiveness of these explanations are varied. The epigenetic condition allowed us to examine the usefulness of causal explanations at reducing stigma from a fresh perspective, and further enabled us to explore the possibility that a new causal measure of stigma reduction may outperform previous methods used in the literature. This addition therefore builds on the current literature which seeks to discern, A) whether causal explanations are the best method when trying to reduce stigma, and B) what the most effective causal explanation is in terms of stigma reduction overall.

The current research was also proficient in that a robust and comprehensive measure of stigma was used. That is, the use of a 47-item questionnaire, including different measures for each separate component of stigma (e.g. stereotypical belief, prejudice, discrimination) should be considered a strong point of the current research. This attention to detail allowed for a precise and sensitive examination of how different causal explanations affect different features of stigma. Most previous research has not employed such comprehensive, inclusive measures of stigma. For example, Lincoln et al., (2008), Schlier et al., (2014) and Walker and Read (2002) each measured aspects of stereotypical belief and discriminatory behaviours, while omitting measures of prejudice from their research. As a plethora of past research identifies that stigma is a multifaceted concept, comprised of several components (Corrigan 2002; Corrigan & Blink 2016) it seems crucial that when designing an experiment aimed at finding the most effective method of stigma reduction overall, a measure of each component of stigma should be included to ascertain what elements of an experiment enhance or diminish specific aspects of stigma in order for us to accurately inform stigma-reduction initiatives. Therefore, as in Cunningham (2016), the inclusion of a multi-component measure has improved the robustness of the research in this area, and further improved on previous studies which have failed to assess the effect of causal interventions on all aspects of stigma.

Limitations

Clearly the participants in the current study are not representative of the general population in regard to age, education and gender, with the majority of the study sample comprising young (aged 17-19), female, university students. Therefore, our results may only be relevant to study samples of similar demographic characteristics such as young female adults with a higher education (i.e. university). Cross-culturally the present findings are also limited, with the majority of respondents in our study identifying as 'New Zealand European' therefore these results cannot be seen as universally informative. It should be noted, however,

that past research has demonstrated that New Zealand students often have similar attitudes to the general population (Blizard, 1968).

Given that all participants in the current study were psychology students, it is also possible that a large number already expressed favourable and sympathetic attitudes towards people with Schizophrenia, therefore explaining the little variance in scores on our stigma measures, and thus resulting in less generalizable findings. Alternatively, being psychology students, who one would generally expect to be potentially more tolerant toward mental illness than your average person, participants may have been motivated by positive self-presentation biases to present themselves in a positive light and appear more understanding and compassionate than the average member of society (e.g. Rosnow, Goodstadt, Suls, & Gitter, 1973) therefore the possibility that a floor effect was occurring in the current study should be considered and accounted for in future research in this area.

This feeds into the third limitation of the study- that is, the lack of implicit measures. While changes in attitude were effectively measured using a multi-component, comprehensive questionnaire, there was no actual measure of behavioural change in the current study. As discrimination is essentially the behavioural component of stigma, the absence of this measure raises a number of issues in regards to reliability of data. Firstly, our behavioural actions and our attitudinal intentions are differing constructs, and previous research has reported attitudes to be poor predictors of behaviour (Ajzen & Fishbein, 2005). Further, attitudinal measures do not exactly indicate whether participants actually intend to engage in the behaviour of note. That is, a participant may report non-stigmatising beliefs and attitudes when completing a self-report measure that are effectively different from them displaying non-stigmatising behaviours. This is the difference between someone reporting that they would be friends, or socialise with an individual with schizophrenia, and then actually doing so. Thus, the current study had no way of measuring whether actual

behavioural change had occurred following the presentation of causal, or non-causal information. So, while in this research stigmatising beliefs and attitudes may have only experienced a minimal positive change (in terms of stereotypical belief) following the teaching of causal explanations, subsequent discriminatory behaviours may have been altered more successfully. To determine this, future research should employ a design or measure of actual behaviour change following a stigma intervention.

Implications

The findings from the current study lead to important theoretical implications; this research somewhat substantiates the idea that stigma is a multi-faceted component, and therefore, the presentation of causal explanations is unlikely to affect all three elements of stigma in the same manner. This was clearly demonstrated in Experiment One, when a psychosocial explanation led to a reduction in stereotypical belief (compared to the creative control) however, not the latter two components- prejudice and discrimination. This was further demonstrated in Experiment Two, where participants in the biogenetic causal condition produced the same pattern; a reduction in stereotypical belief, however not prejudice, or discrimination. This finding implies that while the three components of stigma may be related to each other, they should also be considered stand-alone constructs in the stigma process, as one element can clearly be influenced or altered independently from the others. Further, this study implies that the effectiveness of etiological education on stigma reduction is limited, demonstrating the ability to reduce one aspect of stigma (compared to no-causal information), but failing to improve the other two.

The practical implications of this are important for the ways in which we design anti-stigma programmes and interventions. Consistent with the knowledge that each element of stigma appears to be influenced independently of one another, it would appear that in order to combat every component of stigma and successfully combat the issue as whole, an

intervention would need to target each component of stigma separately, as a technique that improves attitudes towards one component of stigma, may not reduce the other elements as effectively (or at all). A multi-faceted approach would therefore likely be needed to achieve this.

Further, in regard to our study, it appears that etiological information is only helpful for reducing stereotypical belief, moreover, only psychosocial and biogenetic (not epigenetic) explanations were actually proficient in this regard. Therefore, it appears more studies are required in order to ascertain exactly what different kinds of information should go into these destigmatizing campaigns in order to have a positive effect on stigma overall. While causal explanations improve the stereotypical belief associated with stigma, it remains unclear what information we should provide in order to see a reduction in prejudicial belief and discriminatory attitudes as well. In other words, accounting for the findings of the current research, we know that if a destigmitisation programme had the sole aim of reducing stereotypes associated with schizophrenia, this campaign could be primarily concerned with distributing psychosocial or biogenetically based etiological explanations, and likely garner successful stigma-reducing results. However, if the aim of the intervention was to reduce discriminatory attitudes or prejudice, we can assume that neither an epigenetic, biogenetic, or psychosocial condition could achieve the necessary stigma reducing effects. In this regard, it would be most appropriate to explore alternative methods of destigmatisation, unrelated to causal reasons.

It also appears that in comparison to a causal explanation, an explanation based on creativity did not yield any stigma reducing effects, rather, stereotypical belief was actually worse than the psychosocial condition- this implies that information surrounding the potential evolutionary reasons schizophrenia is still present in society, has little effect on stigmatising attitude's, in fact they may actually become worsened by this information (compared with

REDUCING STIGMA TOWARD SCHIZOPHRENIA

psychosocial information). Therefore, future work should not spend any time measuring this potential construct and its ability to reduce stigma. Considering the above points, it is important that future research further investigates the differential effects of causal explanations on stigma as well as other methods of destigmatization.

Future Directions

As mentioned, the findings from the current study reiterate the notion that a multi-faceted destigmatisation programme which is tailored to target each component of stigma separately is likely required in order to successfully reduce stigma overall. Keeping this in mind, it should be a crucial goal for future research to further distinguish what tailored information is best for these programmes, particularly in regard to lowering prejudice and discrimination. As discussed, it does appear that etiological information plays some role in reducing stereotypical beliefs towards mental illness. However, the same cannot be presumed for the latter two elements of the stigma model. In light of this, future experiments should attempt to discern what type of educational information (i.e. explanations about likelihood of recovery as opposed to causal information) is best for reducing these two components of stigma, and, once established combine that information with causal information (which has been found to reduce stereotypical belief) in order to test whether a composite model can produce stigma reducing effects on every component of stigma, rather than just one (i.e. stereotypical belief). Further, given the varied findings in the literature so far regarding the effectiveness of etiological information at reducing stigma, future research should replicate this experiment (and others like it) to certify that the contents of anti-stigma initiatives are based on a robust finding which is established in its usefulness, as opposed to one which is variable in its results. In relation to this point, while the current study was short-term, future research should also attempt to discern whether educational interventions produce long-term improvements in stigmatising attitudes (rather than just temporary changes) and if necessary,

identify ways of maintaining or increasing the benefits over the long term. It is crucial that

we establish what the most effective methods of stigma reduction are. After all, as established

in the current study, causal explanations appear to have a limited impact on stigma. Thus, it is

possible that a method which combines causal information with other educational methods of

stigma reduction, will have a much stronger influence on stigmatising attitudes overall.

References

Addison, S. J., & Thorpe, S. J. (2004). Factors involved in the formation of attitudes towards

those who are mentally ill. *Social Psychiatry and Psychiatric Epidemiology*, *39*(3),

228-234.

Aghukwa, C. N. (2010). Medical students' beliefs and attitudes toward mental illness: effects

of a psychiatric education. *Academic Psychiatry*, *34*(1), 67.

Ajzen, I., & Fishbein, M. (2005). The influence of attitudes on behavior. *The handbook of

attitudes*, *173*(221), 31.

Akinbode, O., & Tolulope, T. (2017). Factors influencing adolescents stigmatising attitudes

and perception of community reaction towards mental illness in Nigeria. *Sudan

Journal of Medical Sciences*, *12*(4), 240-252.

Allport, G. W., Clark, K., & Pettigrew, T. (1954). The nature of prejudice.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental

disorders* (5th ed.). Washington, DC: Author.

Angermeyer, M. C., & Dietrich, S. (2006). Public beliefs about and attitudes towards people

with mental illness: a review of population studies. *Acta Psychiatrica

Scandinavica*, *113*(3), 163-179.

Angermeyer, M. C., & Matschinger, H. (1997). Social distance towards the mentally ill:

results of representative surveys in the Federal Republic of Germany. *Psychological

medicine*, *27*(1), 131-141.

Angermeyer, M. C., & Matschinger, H. (2003). The stigma of mental illness: effects of

labelling on public attitudes towards people with mental disorder. *Acta Psychiatrica

Scandinavica*, *108*(4), 304-309.

Angermeyer, M. C., & Matschinger, H. (2004). The stereotype of schizophrenia and its

impact on discrimination against people with schizophrenia: results from a

representative survey in Germany. *Schizophrenia bulletin*, *30*(4), 1049-1061.

Angermeyer, M. C., & Matschinger, H. (2005). Causal beliefs and attitudes to people with

schizophrenia: trend analysis based on data from two population surveys in

Germany. *The British Journal of Psychiatry*, *186*(4), 331-334.

Angermeyer, M. C., Holzinger, A., & Matschinger, H. (2010). Emotional reactions to people

with mental illness. *Epidemiology and Psychiatric Sciences*, *19*(1), 26-32.

Angermeyer, M. C., Holzinger, A., Carta, M. G., & Schomerus, G. (2011). Biogenetic

explanations and public acceptance of mental illness: systematic review of population

studies. *The British Journal of Psychiatry*, *199*(5), 367-372.

Angermeyer, M. C., Matschinger, H., & Corrigan, P. W. (2004). Familiarity with mental

illness and social distance from people with schizophrenia and major depression:

testing a model using data from a representative population survey. *Schizophrenia

research*, *69*(2-3), 175-182.

Angermeyer, M. C., Matschinger, H., & Holzinger, A. (1998). Gender and attitudes towards

people with schizophrenia. Results of a representative survey in the Federal Republic

of Germany. *International Journal of Social Psychiatry*, *44*(2), 107-116.

Beldie, A., Den Boer, J. A., Brain, C., Constant, E., Figueira, M. L., Filipcic, I., ... &

Karamustafalioglu, O. (2012). Fighting stigma of mental illness in midsize European

countries. *Social psychiatry and psychiatric epidemiology*, *47*(1), 1-38.

Blay, S. L., & Peluso, É. T. P. (2010). Public stigma: the community's tolerance of Alzheimer

disease. *The American Journal of Geriatric Psychiatry*, *18*(2), 163-171.

Blizard, P. J. (1968). Public images of the mentally ill in New Zealand. *The New Zealand medical journal, 68*(438), 297-303.

Bowman, J. W., & West, K. (2018). Prime and prejudice: Brief stereotypical media representations can increase prejudicial attitudes and behaviour towards people with schizophrenia. *Journal of Community & Applied Social Psychology*, *52*(64).

Boysen, G. A., & Vogel, D. L. (2008). Education and mental health stigma: The effects of attribution, biased assimilation, and attitude polarization. *Journal of Social and Clinical Psychology*, *27*(5), 447-470.

Brain, C., Sameby, B., Allerby, K., Quinlan, P., Joas, E., Lindström, E., ... & Waern, M. (2014). Stigma, discrimination and medication adherence in schizophrenia: results from the Swedish COAST study. *Psychiatry research*, *220*(3), 811-817.

Brekke, J. S., Prindle, C., Bae, S. W., & Long, J. D. (2001). Risks for individuals with schizophrenia who are living in the community. *Psychiatric Services*, *52*(10), 1358-1366

Byrd, E. K., & Elliott, T. R. (1985). Feature films and disability: A descriptive study. *Rehabilitation psychology*, *30*(1), 47.

Cangas, A. J., Navarro, N., Parra, J., Ojeda, J. J., Cangas, D., Piedra, J. A., & Gallego, J. (2017). Stigma-Stop: A serious game against the stigma toward mental health in educational settings. *Frontiers in psychology*, *8*, 1385.

Cardwell, M. (1996). Dictionary of Psychology. Chicage IL: Fitzroy Dearborn.

Carter, L., Read, J., Pyle, M., Law, H., Emsley, R., & Morrison, A. (2018). Causal beliefs in people experiencing psychosis: The relationship to treatment accessed and the perceived helpfulness of treatment. *Psychology and Psychotherapy: Theory, Research and Practice*, *91*(3), 332-344.

Casados, A. T. (2017). Reducing the stigma of mental illness: current approaches and future

directions. Clinical Psychology: *Science and Practice, 24(3)*), 306-323.

Catthoor, K., De Hert, M., & Peuskens, J. (2003). Stigma and schizophrenia. a

review. *Journal of Psychiatry*, *45*(2), 87-96.

Chambers, M., Guise, V., Välimäki, M., Botelho, M. A. R., Scott, A., Staniuliene, V., &

Zanotti, R. (2010). Nurses' attitudes to mental illness: A comparison of a sample of

nurses from five European countries. *International journal of nursing studies*, *47*(3),

350-362.

Clement, S., Schauman, O., Graham, T., Maggioni, F., Evans-Lacko, S., Bezborodovs, N., ...

&

Thornicroft, G. (2015). What is the impact of mental health-related stigma on help-seeking?

A systematic review of quantitative and qualitative studies. *Psychological

medicine*, *45*(1), 11-27.

Coker, E. M. (2005). Selfhood and social distance: toward a cultural understanding of

psychiatric stigma in Egypt. *Social Science & Medicine, 61*(5), 920-930.

Collins, R. L., Wong, E. C., Cerully, J. L., Schultz, D., & Eberhart, N. K. (2013).

Interventions to Reduce Mental Health Stigma and Discrimination: A Literature

Review to Guide Evaluation of California's Mental Health Prevention and Early

Intervention Initiative. *Rand health quarterly*, *2*(4).

Conrad, P. (2001). Genetic optimism: Framing genes and mentalillness in the news. *Culture,

medicine and psychiatry*, *25*(2), 225-247.

Cook, T. M., & Wang, J. (2010). Descriptive epidemiology of stigma against depression in a

general population sample in Alberta. *BMC psychiatry*, *10*(1), 29.

Corbiere, M., Samson, E., Villotti, P., & Pelletier, J. F. (2012). Strategies to fight stigma toward people with mental disorders: perspectives from different stakeholders. *The Scientific World Journal*, *2012*.

Corrigan, P. W. (2000). Mental health stigma as social attribution: Implications for research methods and attitude change. *Clinical psychology: science and practice*, *7*(1), 48-67.

Corrigan, P. (2004). How stigma interferes with mental health care. *American psychologist*, *59*(7), 614.

Corrigan, P.W. & Bink, A.B. (2016). The stigma of mental illness. In H.S. Friedman (Ed.) *Encyclopedia of mental health* (*2nd ed.).* Oxford: Academic Press.

Corrigan, P. W., Larson, J. E., & Rüsch, N. (2009). Self-stigma and the "why try" effect: impact on life goals and evidence-based practices. *World psychiatry*, *8*(2), 75-81.

Corrigan, P., Markowitz, F. E., Watson, A., Rowan, D., & Kubiak, M. A. (2003). An attribution model of public discrimination towards persons with mental illness. *Journal of health and Social Behavior*, 162-179.

Corrigan, P. W., & Rao, D. (2012). On the self-stigma of mental illness: Stages, disclosure, and strategies for change. *The Canadian Journal of Psychiatry*, *57*(8), 464-469.

Corrigan, P. W., Roe, D., & Tsang, H. (2011). Challenging the stigma of mental illness. *Hoboken: John Wiley and Sons, Ltd*.

Corrigan, P. W., & Shapiro, J. R. (2010). Measuring the impact of programs that challenge the public stigma of mental illness. *Clinical Psychology Review*, *30*(8), 907-922.

Corrigan, P. W., & Watson, A. C. (2002). Understanding the impact of stigma on people with mental illness. *World psychiatry*, *1*(1), 16.

Couture, S., & Penn, D. (2003). Interpersonal contact and the stigma of mental illness: A review of the literature. *Journal of mental health*, *12*(3), 291-305.

Crisp, A. H., Gelder, M. G., Rix, S., Meltzer, H. I., & Rowlands, O. J. (2000). Stigmatisation of people with mental illnesses. *The British journal of psychiatry*, *177*(1), 4-7.

Crocker, J., & Quinn, D. M. (2000). Social stigma and the self: Meanings, situations, and self-esteem. *The social psychology of stigma*, 153-183.

Cunningham, H. (2016). Destigmatising Schizophrenia: An Investigation into the Effects of Different Causal Explanations upon Stigma. Victoria University, Wellington, New Zelaand.

Dalky, H. F. (2012). Mental illness stigma reduction interventions: Review of intervention trials. *Western journal of nursing research*, *34*(4), 520-547.

De Manzano, Ö., Cervenka, S., Karabanov, A., Farde, L., & Ullen, F. (2010). Thinking outside a less intact box: thalamic dopamine D2 receptor densities are negatively related to psychometric creativity in healthy individuals. *PloS one*, *5*(5), e10670.

Desforges, D. M., Lord, C. G., Ramsey, S. L., Mason, J. A., Van Leeuwen, M. D., West, S. C., & Lepper, M. R. (1991). Effects of structured cooperative contact on changing negative attitudes toward stigmatized social groups. *Journal of personality and social psychology*, *60*(4), 531.

Devine, P. G., & Sharp, L. B. (2009). Automaticity and control in stereotyping and prejudice. *Handbook of prejudice, stereotyping, and discrimination*, *61*, 61.

Dietrich, S., Beck, M., Bujantugs, B., Kenzine, D., Matschinger, H., & Angermeyer, M. C. (2004). The relationship between public causal beliefs and social distance toward mentally ill people. *Australian and New Zealand Journal of Psychiatry*, *38*(5), 348-354.

Durand-Zaleski, I., Scott, J., Rouillon, F., & Leboyer, M. (2012). A first national survey of knowledge, attitudes and behaviours towards schizophrenia, bipolar disorders and autism in France. *BMC psychiatry*, *12*(1), 128.

Eisenberg, D., Downs, M. F., Golberstein, E., & Zivin, K. (2009). Stigma and help seeking

for mental health among college students. *Medical Care Research and Review*, *66*(5),

522-541.

Erlenmeyer-Kimling, L., & Paradowski, W. (1966). Selection and schizophrenia. *The

American Naturalist*, *100*(916), 651-665.

Farina, A., Fisher, J. D., Getter, H., & Fischer, E. H. (1978). Some consequences of changing

people's views regarding the nature of mental illness. *Journal of Abnormal

Psychology*, *87*(2), 272.

Franz, L., Carter, T., Leiner, A. S., Bergner, E., Thompson, N. J., & Compton, M. T. (2010).

Stigma and treatment delay in first-episode psychosis: a grounded theory study. *Early

intervention in psychiatry*, *4*(1), 47-56.

Fresán, A., Robles-García, R., Martínez-López, N., Tovilla-Zárate, C. A., & Madrigal, E.

(2018). Stigma and perceived aggression towards schizophrenia in female students of

medicine and psychology. *Salud mental*, *41*(5), 207-212.

Gaebel, W., & Kerst, A. (2018). The debate about renaming schizophrenia: a new name

would not resolve the stigma. *Epidemiology and psychiatric sciences*, 1-4.

Galletly, C., Castle, D., Dark, F., Humberstone, V., Jablensky, A., Killackey, E., ... & Tran,

N. (2016). Royal Australian and New Zealand College of Psychiatrists clinical

practice guidelines for the management of schizophrenia and related

disorders. *Australian & New Zealand Journal of Psychiatry*, *50*(5), 410-472.

Goffman, E. (1974). *Stigma: Notes on the managment of spoiled identity*. Penguin books.

González-Torres, M. A., Oraa, R., Arístegui, M., Fernández-Rivas, A., & Guimon, J. (2007).

Stigma and discrimination towards people with schizophrenia and their family

members. *Social psychiatry and psychiatric epidemiology*, *42*(1), 14-23.

Gonzalez, J. M., Alegria, M., & Prihoda, T. J. (2005). How do attitudes toward mental health

treatment vary by age, gender, and ethnicity/race in young adults?. *Journal of

community psychology*, *33*(5), 611-629.

Gronholm, P. C., Henderson, C., Deb, T., & Thornicroft, G. (2017). Interventions to reduce

discrimination and stigma: the state of the art. *Social psychiatry and psychiatric

epidemiology*, *52*(3), 249-258.

Gwarjanski, A. R., & Parrott, S. (2018). Schizophrenia in the news: The role of news frames

in shaping online reader dialogue about mental illness. *Health communication*, *33*(8),

954-961.

Hansson, L., Jormfeldt, H., Svedberg, P., & Svensson, B. (2013). Mental health

professionals' attitudes towards people with mental illness: Do they differ from

attitudes held by people with mental illness?. *International Journal of Social

Psychiatry*, *59*(1), 48-54.

Harré, J. R. N. (2001). The role of biological and genetic causal beliefs in the stigmatisation

of'mental patients'. *Journal of mental health*, *10*(2), 223-235.

Haslam, N. (2011). Genetic essentialism, neuroessentialism, and stigma: commentary on Dar-

Nimrod and Heine (2011).

Henderson, C., Robinson, E., Evans-Lacko, S., Corker, E., Rebollo-Mesa, I., Rose, D., &

Thornicroft, G. (2016). Public knowledge, attitudes, social distance and reported

contact regarding people with mental illness 2009–2015. *Acta Psychiatrica

Scandinavica*, *134*, 23-33.

Ho, B. C., & Andreasen, N. C. (2001). Long delays in seeking treatment for

schizophrenia. *The Lancet*, *357*(9260), 898-900.

Holzinger, A., Floris, F., Schomerus, G., Carta, M. G., & Angermeyer, M. C. (2012). Gender

differences in public beliefs and attitudes about mental disorder in western countries:

a systematic review of population studies. *Epidemiology and psychiatric sciences*, *21*(1), 73-85.

Huang, A., Amos, T. B., Joshi, K., Wang, L., & Nash, A. (2018). Understanding healthcare burden and treatment patterns among young adults with schizophrenia. *Journal of medical economics*, *21*(10), 1026-1035.

Jorm, A. F., & Oh, E. (2009). Desire for social distance from people with mental disorders. *Australian and New Zealand Journal of Psychiatry*, *43*(3), 183-200.

Kar, N., & Barreto, S. (2018). Psychosis, creativity and recovery: exploring the relationship in a patient. *BMJ Case Reports*, *2018*, bcr-2017.

Keefe, R. S., Silva, S. G., Perkins, D. O., & Lieberman, J. A. (1999). The effects of atypical antipsychotic drugs on neurocognitive impairment in schizophrenia: a review and meta-analysis. *Schizophrenia bulletin*, *25*(2), 201-222.

Kessler, R. C., Berglund, P. A., Bruce, M. L., Koch, J. R., Laska, E. M., Leaf, P. J., ... & Wang, P. S. (2001). The prevalence and correlates of untreated serious mental illness. *Health services research*, *36*(6 Pt 1), 987.

Kinoshita, Y., Furukawa, T. A., Kinoshita, K., Honyashiki, M., Omori, I. M., Marshall, M., ... & Kingdon, D. (2013). Supported employment for adults with severe mental illness. *Cochrane Database of Systematic Reviews*, (9).

Koschorke, M., Padmavati, R., Kumar, S., Cohen, A., Weiss, H. A., Chatterjee, S., ... & Balaji, M. (2014). Experiences of stigma and discrimination of people with schizophrenia in India. *Social Science & Medicine*, *123*, 149-159.

Kvaale, E. P., Gottdiener, W. H., & Haslam, N. (2013). Biogenetic explanations and stigma: A meta-analytic review of associations among laypeople. *Social science & medicine*, *96*, 95-103.

Kvaale, E. P., Haslam, N., & Gottdiener, W. H. (2013). The 'side effects' of medicalization: A meta-analytic review of how biogenetic explanations affect stigma. *Clinical psychology review*, *33*(6), 782-794.

Kyaga, S., Lichtenstein, P., Boman, M., Hultman, C., Långström, N., & Landen, M. (2011). Creativity and mental disorder: family study of 300 000 people with severe mental disorder. *The British Journal of Psychiatry*, *199*(5), 373-379.

Lam, D. C., Salkovskis, P. M., & Warwick, H. M. (2005). An experimental investigation of the impact of biological versus psychological explanations of the cause of "mental illness". *Journal of Mental Health*, *14*(5), 453-464.

Lannin, D. G., Vogel, D. L., Brenner, R. E., Abraham, W. T., & Heath, P. J. (2016). Does self-stigma reduce the probability of seeking mental health information?. *Journal of Counseling Psychology*, *63*(3), 351.

Latalova, K., Kamaradova, D., & Prasko, J. (2014). Perspectives on perceived stigma and self-stigma in adult male patients with depression. *Neuropsychiatric disease and treatment*, *10*, 1399.

Lauber, C., Anthony, M., Ajdacic-Gross, V., & Rössler, W. (2004). What about psychiatrists' attitude to mentally ill people?. *European Psychiatry*, *19*(7), 423-427.

Lauber, C., Nordt, C., Falcato, L., & Rössler, W. (2004). Factors influencing social distance toward people with mental illness. *Community mental health journal*, *40*(3), 265-274.

Lebowitz, M. S., Ahn, W. K., & Nolen-Hoeksema, S. (2013). Fixable or fate? Perceptions of the biology of depression. *Journal of consulting and clinical psychology*, *81*(3), 518.

Lee, S., Chiu, M. Y., Tsang, A., Chui, H., & Kleinman, A. (2006). Stigmatizing experience and structural discrimination associated with the treatment of schizophrenia in Hong Kong. *Social Science & Medicine*, *62*(7), 1685-1696.

Lincoln, T. M., Arens, E., Berger, C., & Rief, W. (2007). Can antistigma campaigns be improved? A test of the impact of biogenetic vs psychosocial causal explanations on implicit and explicit attitudes to schizophrenia. *Schizophrenia Bulletin*, *34*(5), 984-994.

Link, B. G., & Phelan, J. C. (2001). Conceptualizing stigma. *Annual review of Sociology*, *27*(1), 363-385

Link, B. G., Cullen, F. T., Frank, J., & Wozniak, J. F. (1987). The social rejection of former mental patients: Understanding why labels matter. *American journal of Sociology*, *92*(6), 1461-1500.

Link, B. G., Phelan, J. C., Bresnahan, M., Stueve, A., & Pescosolido, B. A. (1999). Public conceptions of mental illness: labels, causes, dangerousness, and social distance. *American journal of public health*, *89*(9), 1328-1333.

Link, B. G., Struening, E. L., Neese-Todd, S., Asmussen, S., & Phelan, J. C. (2001). Stigma as a barrier to recovery: The consequences of stigma for the self-esteem of people with mental illnesses. *Psychiatric services*, *52*(12), 1621-1626.

Link, B. G., Struening, E. L., Rahav, M., Phelan, J. C., & Nuttbrock, L. (1997). On stigma and its consequences: evidence from a longitudinal study of men with dual diagnoses of mental illness and substance abuse. *Journal of health and social behavior*, *38*, 177-190.

Link, B. G., Yang, L. H., Phelan, J. C., & Collins, P. Y. (2004). Measuring mental illness stigma. *Schizophrenia bulletin*, *30*(3), 511-541.

Livingston, J. D., & Boyd, J. E. (2010). Correlates and consequences of internalized stigma for people living with mental illness: A systematic review and meta-analysis. *Social science & medicine*, *71*(12), 2150-2161.

Longdon, E., & Read, J. (2017). 'People with Problems, Not Patients with Illnesses': Using Psychosocial Frameworks to Reduce the Stigma of Psychosis. *The Israel journal of psychiatry and related sciences*, *54*(1), 24-28.

Lyndon, A. E., Crowe, A., Wuensch, K. L., McCammon, S. L., & Davis, K. B. (2016). College students' stigmatization of people with mental illness: familiarity, implicit person theory, and attribution. *Journal of mental health*, 1-5.

Madianos, M. G., Madianou, D., Vlachonikolis, J., & Stefanis, C. N. (1987). Attitudes towards mental illness in the Athens area: implications for community mental health intervention. *Acta Psychiatrica Scandinavica*, *75*(2), 158-165.

Magliano, L., Read, J., & Marassi, R. (2011). Metaphoric and non-metaphoric use of the term "schizophrenia" in Italian newspapers. *Social Psychiatry and Psychiatric Epidemiology*, *46*(10), 1019-1025.

Magliano, L., Read, J., Rinaldi, A., Costanzo, R., De Leo, R., Schioppa, G., & Petrillo, M. (2016). The influence of causal explanations and diagnostic labeling on psychology students' beliefs about treatments, prognosis, dangerousness and unpredictability in schizophrenia. *Community mental health journal*, *52*(3), 361-369.

Major, B., & O'brien, L. T. (2005). The social psychology of stigma. *Annu. Rev. Psychol.*, *56*, 393-421.

Mann, C. E., & Himelein, M. J. (2004). Factors associated with stigmatizationof persons with mental illness. *Psychiatric Services*, *55*(2), 185-187.

Manning, C., & White, P. D. (1995). Attitudes of employers to the mentally ill. *Psychiatric Bulletin*, *19*(9), 541-543.

Martin, J. K., Pescosolido, B. A., & Tuch, S. A. (2000). Of fear and loathing: The role of'disturbing behavior,'labels, and causal attributions in shaping public attitudes toward people with mental illness. *Journal of health and social behavior*, 208-223.

Maruta, T., & Matsumoto, C. (2018). Renaming schizophrenia. *Epidemiology and psychiatric sciences*, 1-3.

McCrone, P., Knapp, M., Henri, M., & McDaid, D. (2010). The economic impact of initiatives to reduce stigma: demonstration of a modelling approach. *Epidemiology and Psychiatric Sciences*, *19*(2), 131-139.

Millan, M. J., Fone, K., Steckler, T., & Horan, W. P. (2014). Negative symptoms of schizophrenia: clinical characteristics, pathophysiological substrates, experimental models and prospects for improved treatment. *European Neuropsychopharmacology*, *24*(5), 645-692.

Monahan, J., & Appelbaum, P. S. (2000). Reducing violence risk. In *Violence among the Mentally III* (pp. 19-34). Springer, Dordrecht.

Morgan, A. J., Reavley, N. J., Ross, A., San Too, L., & Jorm, A. F. (2018). Interventions to reduce stigma towards people with severe mental illness: Systematic review and meta-analysis. *Journal of psychiatric research*.

Nettle, D., & Clegg, H. (2005). Schizotypy, creativity and mating success in humans. *Proceedings of the Royal Society B: Biological Sciences*, *273*(1586), 611-615.

Nevid, J. S., & Morrison, J. (1980). Attitudes toward mental illness: The construction of the libertarian mental health ideology scale. *Journal of Humanistic Psychology*, *20*(2), 71-85.

Nordt, C., Rössler, W., & Lauber, C. (2006). Attitudes of mental health professionals toward people with schizophrenia and major depression. *Schizophrenia bulletin*, *32*(4), 709-714.

O'Keeffe, D., Turner, N., Foley, S., Lawlor, E., Kinsella, A., O'Callaghan, E., & Clarke, M. (2016). The relationship between mental health literacy regarding schizophrenia and psychiatric stigma in the Republic of Ireland. *Journal of Mental Health*, *25*(2), 100-108.

Owen, P. R. (2012). Portrayals of schizophrenia by entertainment media: a content analysis of contemporary movies. *Psychiatric Services*, *63*(7), 655-659.

Parrott, S., & Parrott, C. T. (2015). Law & disorder: The portrayal of mental illness in US crime dramas. *Journal of Broadcasting & Electronic Media*, *59*(4), 640-657.

Peluso, É. T. P., & Blay, S. L. (2011). Public stigma and schizophrenia in São Paulo city. *Brazilian Journal of Psychiatry*, *33*(2), 130-136.

Penn, D. L., Guynan, K., Daily, T., Spaulding, W. D., Garbin, C., & Sullivan, M. (1994). Dispelling the stigma of schizophrenia: What sort of information is best? Schizophrenia Bulletin, 20, 567–578.

Penn, D. L., Kohlmaier, J. R., & Corrigan, P. W. (2000). Interpersonal factors contributing to the stigma of schizophrenia: social skills, perceived attractiveness, and symptoms. *Schizophrenia research*, *45*(1-2), 37-45.

Pescosolido, B. A., Martin, J. K., Long, J. S., Medina, T. R., Phelan, J. C., & Link, B. G. (2010). "A disease like any other"? A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. *American Journal of Psychiatry*, *167*(11), 1321-1330.

Pescosolido, B. A., Monahan, J., Link, B. G., Stueve, A., & Kikuzawa, S. (1999). The public's view of the competence, dangerousness, and need for legal coercion of persons with mental health problems. *American journal of public health*, *89*(9), 1339-1345.

Phelan, J. C. (2005). Geneticization of deviant behavior and consequences for stigma: The

case of mental illness. Journal of Health & Social Behavior, 46, 307–322.

Phelan, J. C., Cruz-Rojas, R., & Reiff, M. (2002). Genes and stigma: The connection between

perceived genetic etiology and attitudes and beliefs about mental illness. *Psychiatric*

Rehabilitation Skills, *6*(2), 159-185.

Phelan, J. C., Link, B. G., & Dovidio, J. F. (2008). Stigma and prejudice: One animal or

two?. *Social science & medicine*, *67*(3), 358-367.

Pilgrim, D. (2005). *Key concepts in mental health*. London, United Kingdom: SAGE

Publications Ltd.

Pinfold, V., Toulmin, H., Thornicroft, G., Huxley, P., Farmer, P., & Graham, T. (2003).

Reducing psychiatric stigma and discrimination: evaluation of educational

interventions in UK secondary schools. *The British Journal of Psychiatry*, *182*(4),

342-346.

Read, J. (2007). Why promoting biological ideology increases prejudice against people

labelled "schizophrenic." *Australian Psychologist*, *42*(2), 118–128.

doi:10.1080/00050060701280607

Read, J., & Law, A. (1999). The relationship of causal beliefs and contact with users of

mental health services to attitudes to the'mentally ill'. *International Journal of Social*

Psychiatry, *45*(3), 216-229.

Read, J., Haslam, N., Sayce, L., & Davies, E. (2006). Prejudice and schizophrenia: a review

of the 'mental illness is an illness like any other'approach. *Acta Psychiatrica*

Scandinavica, *114*(5), 303-318.

Reavley, N. J., & Jorm, A. F. (2011). Stigmatizing attitudes towards people with mental

disorders: findings from an Australian National Survey of Mental Health Literacy and

Stigma. *Australian and New Zealand Journal of Psychiatry*, *45*(12), 1086-1093.

Reavley, N. J., Mackinnon, A. J., Morgan, A. J., & Jorm, A. F. (2014). Stigmatising attitudes towards people with mental disorders: A comparison of Australian health professionals with the general community. *Australian & New Zealand Journal of Psychiatry*, *48*(5), 433-441.

Reinke, R. R., Corrigan, P. W., Leonhard, C., Lundin, R. K., & Kubiak, M. A. (2004). Examining two aspects of contact on the stigma of mental illness. *Journal of social and clinical psychology*, *23*(3), 377-389.

Rosnow, R. L., Holper, H. M., & Gitter, A. G. (1973). More on the reactive effects of pretesting in attitude research: Demand characteristics or subject commitment?. *Educational and Psychological Measurement*, *33*(1), 7-17.

Ross, C. A., & Goldner, E. M. (2009). Stigma, negative attitudes and discrimination towards mental illness within the nursing profession: a review of the literature. *Journal of psychiatric and mental health nursing*, *16*(6), 558-567

Rössler, W. (2016). The stigma of mental disorders: A millennia-long history of social exclusion and prejudices. *EMBO reports*, *17*(9), 1250-1253.

Roth, T. L., Lubin, F. D., Sodhi, M., & Kleinman, J. E. (2009). Epigenetic mechanisms in schizophrenia. *Biochimica et Biophysica Acta (BBA)-General Subjects*, *1790*(9), 869-877.

Rüsch, N., Angermeyer, M. C., & Corrigan, P. W. (2005). Mental illness stigma: Concepts, consequences, and initiatives to reduce stigma. *European psychiatry*, *20*(8), 529-539.

Rüsch, N., Müller, M., Lay, B., Corrigan, P. W., Zahn, R., Schönenberger, T., ... & Rössler, W. (2014). Emotional reactions to involuntary psychiatric hospitalization and stigma-related stress among people with mental illness. *European Archives of Psychiatry and Clinical Neuroscience*, *264*(1), 35-43.

Rüsch, N., Todd, A. R., Bodenhausen, G. V., & Corrigan, P. W. (2010). Biogenetic models of

psychopathology, implicit guilt, and mental illness stigma. *Psychiatry research,*

179(3), 328-332.

Schlier, B., Schmick, S., & Lincoln, T. M. (2014). No matter of etiology: Biogenetic,

psychosocial and vulnerability-stress causal explanations fail to improve attitudes

towards schizophrenia. *Psychiatry research*, *215*(3), 753-759.

Schulze, B., & Angermeyer, M. C. (2003). Subjective experiences of stigma. A focus group

study of schizophrenic patients, their relatives and mental health professionals. *Social*

science & medicine, *56*(2), 299-312.

Schulze, B., Richter-Werling, M., Matschinger, H., & Angermeyer, M. C. (2003). Crazy? So

what! Effects of a school project on students' attitudes towards people with

schizophrenia. *Acta Psychiatrica Scandinavica*, *107*(2), 142-150.

Shrivastava, A., Johnston, M., & Bureau, Y. (2012). Stigma of mental illness-1: Clinical

reflections. *Mens sana monographs*, *10*(1), 70.

Singh, A., Mattoo, S. K., & Grover, S. (2016). Stigma associated with mental illness:

conceptual issues and focus on stigma perceived by the patients with schizophrenia

and their caregivers. *Indian Journal of Social Psychiatry*, *32*(2), 134.

Staiger, T., Waldmann, T., Rüsch, N., & Krumm, S. (2017). Barriers and facilitators of help-

seeking among unemployed persons with mental health problems: a qualitative

study. *BMC health services research*, *17*(1), 39.

Stuart, H. (2016). Reducing the stigma of mental illness. *Global Mental Health*, *3*.

Svensson, B., & Hansson, L. (2016). How mental health literacy and experience of mental

illness relate to stigmatizing attitudes and social distance towards people with

depression or psychosis: A cross-sectional study. *Nordic journal of psychiatry*, *70*(4),
309-313.

Tandon, R., Nasrallah, H. A., & Keshavan, M. S. (2009). Schizophrenia,"just the facts" 4.
Clinical features and conceptualization. *Schizophrenia research*, *110*(1-3), 1-23.

Thornicroft, G., Brohan, E., Rose, D., Sartorius, N., Leese, M., & INDIGO Study Group.
(2009). Global pattern of experienced and anticipated discrimination against people
with schizophrenia: a cross-sectional survey. *The Lancet*, *373*(9661), 408-415.

Thornicroft, G., Mehta, N., Clement, S., Evans-Lacko, S., Doherty, M., Rose, D., ... &
Henderson, C. (2016). Evidence for effective interventions to reduce mental-health-
related stigma and discrimination. *The Lancet*, *387*(10023), 1123-1132.

Thornicroft, G., Rose, D., Kassam, A., & Sartorius, N. (2007). Stigma: ignorance, prejudice
or discrimination?. *The British Journal of Psychiatry*, *190*(3), 192-193.

Thornton, J. A., & Wahl, O. F. (1996). Impact of a newspaper article on attitudes toward
mental illness. *Journal of community psychology*, *24*(1), 17-25.

Thorsteinsson, E. B., Bhullar, N., Williams, E., & Loi, N. M. (2018). Schizophrenia literacy:
the effects of an educational intervention on populations with and without prior health
education. *Journal of Mental Health*, 1-9.

Van Dorn, R. A., Swanson, J. W., Elbogen, E. B., & Swartz, M. S. (2005). A comparison of
stigmatizing attitudes toward persons with schizophrenia in four stakeholder groups:
perceived likelihood of violence and desire for social distance. *Psychiatry:
Interpersonal and Biological Processes*, *68*(2), 152-163.

Vezzoli, R., Archiati, L., Buizza, C., Pasqualetti, P., Rossi, G., & Pioli, R. (2001). Attitude
towards psychiatric patients: a pilot study in a northern Italian town. *European
psychiatry*, *16*(8), 451-458.

Wahl, O. F., & Harman, C. R. (1989). Family views of stigma. *Schizophrenia Bulletin*, *15*(1),

131-139.

Walker, I., & Read, J. (2002). The differential effectiveness of psychosocial and biogenetic

causal explanations in reducing negative attitudes toward "mental

illness". *Psychiatry: Interpersonal and Biological Processes*, *65*(4), 313-325.

Walsh, E., Moran, P., Scott, C., McKenzie, K., Burns, T., Creed, F., ... & Fahy, T. (2003).

Prevalence of violent victimisation in severe mental illness. *The British Journal of

Psychiatry*, *183*(3), 233-238.

Watson, A. C., Corrigan, P., Larson, J. E., & Sells, M. (2007). Self-stigma in people with

mental illness. *Schizaophrenia bulletin*, *33*(6), 1312-1318.

Wiesjahn, M., Jung, E., Kremser, J. D., Rief, W., & Lincoln, T. M. (2016). The potential of

continuum versus biogenetic beliefs in reducing stigmatization against persons with

schizophrenia: an experimental study. *Journal of behavior therapy and experimental

psychiatry*, *50*, 231-237.

Wood, A. L., & Wahl, O. F. (2006). Evaluating the effectiveness of a consumer-provided

mental health recovery education presentation. *Psychiatric rehabilitation

journal*, *30*(1), 46.

Ye, J., Chen, T. F., Paul, D., McCahon, R., Shankar, S., Rosen, A., & O'Reilly, C. L. (2016).

Stigma and discrimination experienced by people living with severe and persistent

mental illness in assertive community treatment settings. *International Journal of

Social Psychiatry*, *62*(6), 532-541.

Young, M., Read, J., Barker-Collo, S., & Harrison, R. (2001). Evaluating and overcoming

barriers to taking abuse histories. *Professional Psychology: Research and

Practice*, *32*(4), 407.

Yuan, Q., Abdin, E., Picco, L., Vaingankar, J. A., Shahwan, S., Jeyagurunathan, A., ... &

Subramaniam, M. (2016). Attitudes to mental illness and its demographic correlates

among general population in Singapore. *PloS one*, *11*(11), e0167297.

Appendix A

Content Check Questions for Video Clip

The following includes a list of the two multi choice content-check questions that were given to participants across both experiments in order to assess whether these individuals had been paying sufficient attention to the contents of the video material. Participants were able to select one answer only, the order they were displayed in for the experiment, is the same order as they are presented in below. The correct answers for each question are indicated by an asterisk.

1. 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

2. In the second half of the clip, who attends the therapy session with Gerald?

   - His mother *

   - His brother

   - His friend

Appendix B

Written Material: Information about the symptoms of schizophrenia

The following page contains the information participants read about the symptoms of schizophrenia immediately following the content check questions regarding the material in the video clip (Appendix A). Irrespective of condition, participants in both experiments read this text.

"There are three key symptoms in schizophrenia- hallucinations, delusions & disorganised thought/disorganised behaviour. These behaviours were all evident in the previous clip, please read the following text as it explains the symptoms of schizophrenia more clearly, when you are done proceed to the next page:

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 much like 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, in the previous clip 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 the previous video as well. For example, 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 and Two Educational Information:

Below is a complete copy of the information provided to participants in the Creative Novel

Condition, the Epigenetic Causal Condition, the Psychosocial Causal Condition and the

Biogenetic Causal Condition. These causal explanations were displayed to participants

immediately following the presentation of the text exert which explained the symptoms of

schizophrenia (Appendix B). It should be noted however, that the creative information was

only used in Experiment One of the current research, and the epigenetic information was only

utilised for Experiment Two. The Biogenetic and Psychosocial explanations were the same

across both Experiments in this study.

Biogenetic Condition Causal Explanation

Schizophrenia is caused by BIOLOGY:

Schizophrenia is a debilitating and severe brain disease. More specifically, popular
research suggests that schizophrenia is a biological disorder whereby certain genes cause
brain abnormalities to develop, these malfunctions then cause numerous symptoms, such as
those displayed in the video you have just watched. Below you will read an exert which
describes this notion in further detail:

Researchers have found two key differences between the brain functioning and structure of
those individuals with schizophrenia, and those without. Firstly, scientists have suggested that
an imbalance of specific natural chemicals in the brain may play a pivotal role in the
development of schizophrenia. These chemicals are otherwise referred to as
neurotransmitters, and essentially allow brain cells to communicate with one another. A
disproportionate number of these neurotransmitters mean the brain will fail to function and
converse properly. Schizophrenic individuals are known to have abnormal quantities of the
neurotransmitter dopamine. The positive symptoms of schizophrenia- including
hallucinations and delusions- are thought to be a result of abnormal dopamine levels in the
brains of schizophrenic patients.

The second biological difference between schizophrenics and non-schizophrenics, is the
dissimilarity between the size and shape of certain brain structures. For instance, ventricular
enlargement is thought to be one of the most consistent and reliable findings among
schizophrenic patients compared to non-patients. That is, researchers have discovered
differences in the size of a certain brain structure called the ventricles. Brain scans taken from
twin studies show that in comparison to a healthy individual, the ventricles of an individual
with schizophrenia are much larger in size! This can be seen in the photograph below:

Experts believe that these malfunctions and imbalances in the brain are most likely caused by an individual's genetic make up. This means that people with schizophrenia are, to a certain extent, born that way. These people haven't done anything specifically which would cause them to develop schizophrenia; they were merely born with different genes to the rest of us.

To summarise, there exists a large volume of scientific research which presents evidence that schizophrenia is a biological disease which affects the brain, and further, that this disease is passed through individuals via genetic inheritance. While there is still a lot of research to be done on schizophrenia, there is one thing we know for sure: **schizophrenia is an illness much like any other medical sickness and it is caused purely by biological factors and genetics.**

Psychosocial Condition Causal Explanation:
Schizophrenia is caused by PSYCHOSOCIAL (lifestyle) factors:

One of the most popular views regarding the causes of schizophrenia **emphasizes the effect of social factors and stressors on an individual when developing this illness**. Below you will read an exert which describes this notion in further detail, **please pay as much attention to this as possible as you will be asked content check questions following this exercise:**

Schizophrenia is a type of mental unpredictability that is triggered by negative factors or destructive events in a person's social life. Widespread research has been carried out by researchers to identify the kinds of events and enduring factors which ultimately culminate in a diagnosis of schizophrenia. More specifically, it has been suggested that the unusual thoughts, behaviours and sometimes, delusions experienced by those with schizophrenia are a result of the exposure to adverse events earlier in an individual's life. Whilst there are suggestions that the majority of these incidents are severe and would produce psychological distress for most individuals, scientists have also suggested that a lower level negative life experience (sustained over a long period of time) may also result in the onset of schizophrenia. Over the past few decades, researchers have investigated the differences between individuals with schizophrenia and those with no history of mental instability. This research has aided in recognising the numerous adverse life events which are responsible for causing schizophrenia.
Some of these life events include: extreme exposure to emotional, physical, or sexual abuse in childhood or adolescence; experiencing high volumes of stress, being placed in traumatic situations, and/or being socially isolated. Additionally, whilst not as life changing as the former reasons, experiencing things such as: bullying, discrimination (sexism, racial prejudice etc.) , a damaged relationship with parents, lower income, job instability and income inequality can all additionally increase the likelihood an individual will develop schizophrenia.

Whilst some of these factors may not seem substantial enough to contribute to the onset of schizophrenia alone, there has been a great deal of research over the past few decades which has proved these factors are strong contributors to an individual developing this illness. More specifically, this fact is proven in the diagram below which shows levels of income inequality compared with levels of schizophrenia among individuals in numerous locations:

(IMAGE REMOVED DUE TO COPYRIGHT)

Income inequality is a measure of the 'rich-poor gap' in any given society. It reflects the extent to which a society is unequal in terms of income distribution. The research above found that as income inequality increased, so did the amount of individuals with schizophrenia in each location. To summarize, there exists a large collection of scientific research which provides evidence that schizophrenia is directly caused by severely negative events in an individual's life. While there is still a large amount of research to be conducted regarding schizophrenia, one thing is known for sure: **it is the psychosocial factors (social experiences and environment) that an individual grows up around which ultimately determines whether someone will develop schizophrenic thoughts and behaviour**

Epigenetic Condition Causal Explanation:

Schizophrenia is caused by EPIGENETICS (a combination of genetics AND lifestyle factors):
Like all mental illnesses, the causes of schizophrenia (SCZ) are extremely complex and variable. For example, although certain genes are associated with the development of schizophrenia, the presence of these genes does not necessarily mean they will be 'expressed' by the individual. To expand on this idea, researchers have consistently suggested that it is the interaction between certain candidate genes and stressful life events which are pivotal to the onset of schizophrenia.

In simpler terms, researchers suggest that the development of schizophrenia is influenced by both an individual's genes AND their environment. Over recent years, researchers have begun to understand how particular life experiences influence the expression of certain genes to make an individual either more or less vulnerable towards developing schizophrenia. One of the key discoveries researchers have made is that even if an individual has particular genes which have been implicated in schizophrenia, these are not necessarily active without outside factors. This is because genes can be switched 'on' or 'off' (much like a light bulb) depending on the outside influence of certain lifestyle factors. The study of these factors is referred to as epigenetics (the way that genes interact with the environment, in order to produce individual phenotypes). To describe this process at its simplest: you may be born with a capacity to be tall and confident, but if you are undernourished and abused as a child, you are likely to turn into a stunted and fearful young adult, regardless of your genetic makeup.

(IMAGE REMOVED DUE TO COPYRIGHT)

There is increasing experimental evidence which tells us that environmental lifestyle factors (stress, socio-economic status, history of abuse) can alter the epigenetic status of specific genes and therefore the presence of schizophrenia in an individual. For example, research now proves that environmental factors such as; poor diet, drug use, negative social interactions and stress levels can all serve as epigenetic agents, altering DNA and ultimately leading to the onset of schizophrenia. That is, these lifestyle factors each play a role in whether the genes associated with schizophrenia are turned on or not. In simpler terms, genes may be compared to a heating system in your home, where various other factors influence the

thermostat. The heater is always there, however it is not always active, it is the settings on the thermostat that determine whether the heat will be turned on or not. Comparatively, the genes we are born with are always there, it is the presence of environmental factors which regulates whether these genes are altered and switched on or not.

To summarize, there exists a large volume of scientific research which provides evidence that schizophrenia is caused by a gene X environment interaction. That is, the expression of an individual's genes may be altered according to their exposure to various environmental factors. Whilst there is still a large amount of research to be conducted regarding schizophrenia, one thing is known for sure: both an individual's genetic make-up AND their exposure to certain lifestyle factors ultimately determine whether someone will develop schizophrenia or not.

Novel Creative/ Evolutionary Condition Information:

Schizophrenia exists due to the benefits it holds in society today:

One of the most popular views regarding the cause of schizophrenia emphasises that the **symptoms characteristic of schizophrenia constitute one end of a spectrum of primarily adaptive strategie**s. Below you will read an exert which describes this notion in further detail:

One of the primary features of schizophrenia comprises of positive symptoms. More specifically, delusions and hallucinations are often experienced very severely among individuals with schizophrenia. Whilst these symptoms have typically been viewed as 'harmful' and 'out of touch with reality'- recent evidence suggests that, these symptoms of schizophrenia, and the illness overall, are in fact an adaptive trait that has been moulded by natural selection over the course of evolution, and can be foretelling of positive outcomes in certain settings.

Psychiatric studies have suggested that individuals with schizophrenia are more creative and imaginative than the general population. This has raised the idea that in the past, and over the course of evolution, schizophrenic genes actually helped carriers to solve survival problems and potentially even attract mating partners. Essentially, these positive symptoms, which are characteristic of schizophrenia, have helped us historically, acting as a means to aid survival and allowing the development of superior problem solving skills, creativity and adaptability over time.

There is evidence which also supports the notion that creativity and schizophrenia are undeniably linked to each other. A study of the medical records of 150,000 individuals has found that the relatives of patients diagnosed with schizophrenia or bipolar disorder are more likely than the general public to be creative professionals (such as actors, dancers, musicians, visual artists or writers). The findings from these medical records were then compared with various other studies in the Netherlands and Sweden involving a further 35,000 people. This revealed that members of creative professions were 25 per cent more likely than other professions to carry the DNA variants linked with schizophrenia. This research re-emphasises the possible adaptive features of schizophrenia over time, such as an increase in artistic capacity and talent.

Additionally, a number of well-known and creative individuals have suffered from schizophrenia in the past, and ultimately succeeded in their field due to their enhanced creativity. John Nash is one example of this- he received the Nobel Prize in economics for "The pioneering analysis of equilibria in the theory of non-competitive games" (game theory). His work would essentially change history in both mathematical and engineering fields. It is examples like these which allow us to understand the advantages of schizophrenia as an adaptive trait that has been cultivated and shaped during natural selection to enhance creativity and flexibility among the population. While there is still a lot of work to be conducted regarding schizophrenia, a large collection of research points to one undeniable fact: **schizophrenia results from increasing levels of creativity and thinking which has developed throughout evolutionary history, and when put to use in the correct circumstances can be a definite advantage.**

Appendix D

Scales Measuring Stigma: Comprehensive List of all 47 Items

Below is a list comprising all four of the questionnaires (and one subscale) as compiled by Cunningham (2016). Every participant in the current research was required to answer these.

Schizophrenia Stereotype Questionnaire (SSQ)

This questionnaire measures the degree to which individuals subscribe to a range of stereotypical beliefs about individuals with schizophrenia. The SSQ is comprised of 15 items, and an additional eight from 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 to indicate this.

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 popular stereotypical beliefs regarding the dangerousness of individuals with schizophrenia. A higher score on the SPDS suggests a higher level of the perception 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 <u>would</u> 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 <u>would</u> 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)

The ERQ measures the extent to which participants experience prejudicial emotional reactions when intermingling with or thinking about people with schizophrenia. A higher score on the ERQ is indicative of a higher level of prejudice. Items one, four, and eight of the ERQ are reverse-scored to reflect this.

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 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 stigmatising attitudes. 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 are reverse-scored so that 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?