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Educating medical students about anorexia nervosa: A potential method for reducing the volitional stigma associated with the disorder?

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Abstract

It is frequently reported clinicians across a range of professional disciplines experience strong negative reactions toward patients with anorexia nervosa (AN). The present study aimed to develop, evaluate, and compare the effectiveness of two different educational programs, based on an etiological framing model. Participants were medical students ($N = 41$) from an Australian University, who were randomly assigned to one of three conditions (biogenetic intervention vs multifactorial intervention vs control). Outcome attitudinal/stigma data were collected pre- and post-intervention, and at eight weeks follow-up. Results indicated intervention participations exhibited significantly lower volitional stigma scores compared to the control group, who exhibited no change in attitudes or stigma. Specifically, intervention participants had significantly lower total ED stigma scores, level of blame, perceptions of AN as a selfish/vain illness, and viewed sufferers' as less responsible for their illness, at post-intervention. These reductions were maintained at follow-up. Overall, the study provides preliminary evidence brief targeted interventions can assist in reducing levels of volitional stigma toward AN.

Keywords: eating disorders, anorexia nervosa, stigma, volition, medical education

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The concept of stigma has attracted increased attention in recent years among the general population, health professionals, and policy makers, entering the vocabulary of public culture in an attempt to describe the social impact of specific illnesses (Weiss & Ramakrishna, 2006). For individuals suffering from an eating disorder (ED), particularly anorexia nervosa (AN), the experience of stigma is an unfortunate and pervasive reality, inflicted not only by the general population (Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000), but also the health profession (Lauber, Anthony, Ajdacic-Gross, & Rossler, 2004).

Research has indicated clinicians across a range of health-related disciplines, particularly entry-level physicians, frequently report negative reactions in response to ED sufferers (Thompson-Brenner, Satir, Franko, & Herzog, 2012). Researchers suggest these reactions are due to pervasive stigma, particularly perceptions of volition (Crisafulli, Von Holle, & Bulk, 2008; Holliday, Wall, Treasure, & Weinman, 2005; Mond, Robertson-Smith, & Vetere, 2006; Stewart, Keel, & Schiavo, 2006). For example, despite the severity of AN, the condition is often trivialised as self-inflicted (Crisp et al., 2000), a “teenage fad” (Holliday et al., 2005), overenthusiastic dieting, an attempt to copy celebrity icons (Tierney, 2008), or attention seeking in nature (Mond et al., 2006).

This perception of AN as a voluntary, or self-inflicted illness, has been theorised as a form of stigma referred to as “volitional stigma”, as individuals with AN are often blamed for their illness. However, unlike traditional mental illness stigma, which proposes individuals are set apart from “normals”, volitional stigma is suggested to involve being judged by normal behavioural standards (Easter, 2012), possibly due to the ego-syntonic nature of AN. That is, individuals (i.e., friends, family members, treating professionals) may recognise the severity of the condition, yet assign desirable, almost enviable attributes, to disordered eating

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behaviour (e.g., weight control). This is an aspect of stigma unique to AN, as similar attitudes are non-existent across other mental disorders (Roehrig & McLean, 2010).

Over the past two decades, numerous studies have examined health professionals' attitudes toward EDs, revealing alarming findings. In a sample of medical and nursing staff (N = 352) from a general hospital in Australia, 59.4 percent strongly agreed patients with AN were personally responsible for their condition and many described AN sufferers as less likeable than other patients (Fleming & Szmukler, 1992). Similarly, first-year medical residents in medicine, psychiatry and paediatrics have been found to experience greater negative affect (e.g., anger, irritation, and hostility) when treating patients with AN compared to patients with diabetes or obesity, with researchers suggesting this may be due perceptions of AN as intentional and destructive (Brotman, Stern, & Herzog, 1984).

Physicians and general medical nurses have also been found in numerous studies to communicate clear messages to ED patients their time and expertise is more effectively spent caring for patients who are 'really sick', 'more deserving', and who have not 'self-inflicted' illness given scarce health resources, and the sufferer is merely 'occupying/blocking a bed' (Happell, 2005; Mavundla, 2000). Given that sensitivity to criticism and perfectionism are very frequently associated with EDs (Becker, Arrindell, Perloe, Fay, & Striegel-Moore, 2009), it is probable even an occasional encounter with a health professional demonstrating these stigmatising attitudes (or the knowledge individuals holding these beliefs exist) will be distressing for AN sufferers and may have strong detrimental effects (e.g., shame).

Do health professionals possess adequate ED knowledge?

While health professionals are typically believed to possess a greater level of mental health literacy than the general population, research indicates the expected dichotomy between lay and professional beliefs falls more along a continuum, with little distinction between groups (Jorm, 2000). It is well documented increased practitioner knowledge

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translates to improved detection and treatment of EDs in primary care; however, deficits in primary care physicians ED knowledge have been consistently highlighted, affecting both attitudes and clinical behaviour (e.g., diagnosis, follow-up appointments, referrals to specialist services) (Currin, Waller, & Schmidt, 2009).

Studies assessing the attitudes of medical professionals (e.g., general practitioners, paediatricians, medical registrars, gynecologists and obstetricians) have also demonstrated health professionals lack confidence, or feel a lack of competence, in treating EDs, particularly AN (Boulé & McSherry, 2002; Linville, Benton, O'Neil, & Sturm, 2010). Many physicians have been found to feel uncomfortable in the treatment and management of ED patients, and often report their undergraduate studies and entry-level training did not provide adequate education in relation to the etiology, treatment, and management of EDs (Walker & Lloyd, 2012). As a result, physicians often decline to treat individuals with EDs and frequently respond to sufferers in a negative manner, significantly affecting treatment alliances and future help-seeking behaviour (Pereira, Lock, & Oggins, 2006).

For example, a study (e.g., de la Rie, Noordenbos, Donker, & van Furth, 2006) which evaluated ED treatment experiences from the perspective of current and previous sufferers (*N* = 156), revealed over 80 percent of the sample reported consultation with an inexperienced general practitioner (GP), or treatment within a non-specialist hospital was "unhelpful" and "traumatic", due to a lack of knowledge, punitive treatment methods (e.g., isolation, forced feeding), poor empathy and understanding, or delayed referral to specialised services due to illness trivialisation. Findings of the study also revealed 21 percent of the sample ceased treatment seeking for more than five years following a negative treatment experience with a GP and more than 70 percent reported doctor delay due to fear of stigmatisation.

Development of negative attitudes and volitional stigma

Extensive research suggests negative attitudes toward particular mental illnesses

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develop early in an individual's medical training and career (Mukherjee, Fialho, Wijetunge, Checinski, & Surgenor, 2002; Williams & Leichner, 2006). In relation to AN, research has indicated medical students and early-career physicians, who have little to no experience in treating EDs, often report a lack of empathy and strongly endorse beliefs of self-infliction (Walker & Lloyd, 2012), despite the wealth of literature highlighting the contribution of multiple factors (e.g., biogenetic, psychological, and social) in the development and maintenance of AN.

While there has traditionally been strong endorsement of the biomedical approach within the medical profession, the lack of application of this model in respect to the etiology of AN is perplexing, raising concerns about the nature of education received, transmission of attitudes/beliefs in relation to the condition, and/or the degree to which students practice based on personal assumptions developed from inaccurate media representations (e.g., sociocultural explanations of vanity and societal thin ideals) within the public domain. As the basic structure of physicians' clinical knowledge and attitudes appears to be developed and shaped early in their careers, the importance of adequately educating students about EDs appears vital.

Researchers (e.g., Crisafulli et al., 2008; Currin et al., 2009; Thompson-Brenner et al., 2012) have suggested incorporating didactic education, regarding the etiology and prognosis of AN, into the training of medical students may positively affect clinical attitudes and behaviour toward sufferers; however, there is limited research investigating the optimal nature and frequency of this training, nor whether effects would be enduring over time. In recent years, efforts to alleviate stigma via education in other highly stigmatised conditions (e.g., HIV/AIDS, leprosy, schizophrenia) have been successful, highlighting the possibility similar outcomes could be achieved for AN.

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The effect of etiological framing

As stigma and perceived etiology are taken to be conceptually similar, people's beliefs about the etiology of mental disorders heavily influence their perceptions of individuals with particular disorders (Read, Haslam, Sayce, Davies, 2006). According to attribution theory of stigma (Weiner, Perry, & Magnusson, 1988), controllability is closely linked to blame. The more a condition is seen as controllable, the more sufferers are seen as responsible for their situation, a common perception in the origin and maintenance of EDs. As biological explanations often promote the belief individuals are incapable of controlling their behaviour, framing the etiology of AN as biogenetic has been hypothesised to be an effective method for reducing volitional stigma (Link et al., 2004).

Preliminary research suggests this strategy may indeed be beneficial, with various etiological framing models examined (e.g., biogenetic, sociocultural, environmental, and multifactorial explanations) in female nursing students (Crisafulli et al., 2008), psychology students (Crisafulli et al., 2010), and a general undergraduate sample (Bannatyne & Abel, 2014). Consistent with attribution theory, the studies revealed participants who received a biologically-framed etiological explanation tended to view individuals suffering from AN as less responsible for their condition, exhibited lower levels of blame and vanity related stigma, and greater intention to engage in helping behaviour, compared to those who received a sociocultural explanation. However, concerns regarding the impact of traditional multifactorial explanations of AN were raised as the multifactorial group in both Bannatyne and Abel (2014) and Crisafulli et al. (2010) produced increased perceptions of illness responsibility. Bannatyne and Abel (2014) suggested attitude accessibility (Roskos-Ewoldsen, Bichsel, & Hoffman, 2002), whereby individuals attend to the most salient information (sociocultural factors in the case of AN) and ignore competing evidence (e.g., biogenetic information), might be a possible explanation for the finding.

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The current study

Based on previous research, the empirical objective of the current study was to examine the effect of education and various disease explanations of AN in reducing negative attitudes and volitional stigma. The current study developed and assessed the impact and effectiveness of a traditional multifactorial educational intervention compared to a biogenetic educational intervention, and a control group, over several time points to determine retention effects. Consistent with previous research and the propositions of attribution theory, it was hypothesised that:

1. Participants who received either the multifactorial or biogenetic intervention would exhibit a significant decrease in both negative attitudes and volitional stigma measures from pre-intervention to post-intervention, and any effects would be maintained over time (i.e., 8 weeks). That is, follow-up scores for the intervention groups would be significantly different from pre-intervention measurement.
2. Participants in the intervention groups would have significantly lower negative attitudes and volitional stigma scores compared to participants who received no education (control group) at follow-up (Time 3).
3. The biogenetic intervention would demonstrate a greater stigma reduction effect over participants in the traditional multifactorial intervention, immediately post-intervention and at follow-up; however, the multifactorial group would still have significantly lower scores than the control group.

Method

Participants

A purposive sample comprised of 41 fourth-year Medicine students was recruited through School of Medicine at the authors' institution. Consistent with the reasoning provided by Crisafulli et al. (2008), that brief information regarding the etiology of AN would be

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unlikely to compete with previous experiences of the illness, one female participant with a self-reported history of AN was removed from the data-set. The final sample comprised of 40 fourth-year medical students aged 20 to 55 years (M = 25.33, SD = 6.70), with no self-reported history of AN. Males comprised 60 percent (n = 24) of the sample, whilst females comprised 40 percent (n = 16). In terms of ethnicity, 20 participants identified as Caucasian, 14 identified as Asian, and six identified as "Other", typically stating Middle Eastern or Indian. Based on random assignment, 11 participants attended the multifactorial educational intervention, 15 participants received the biogenetic educational intervention, and 14 participants received no intervention, forming the control group. All participants gave informed consent. The research was approved by the university ethics committee and the study was registered under the Australia New Zealand Clinical Trials Registry.

Materials

Demographic Questions. Participants were asked to supply demographic information for the purpose of describing the sample. Participants were asked whether he/she felt, or had been told by a health professional, he/she had suffered from AN.

Educational Interventions. Participants were randomly assigned to one of three conditions (biogenetic education vs traditional multifactorial education vs no education) via a computer-generated randomisation procedure, completed by administrative staff at the School of Medicine. The educational interventions were developed by the first author on the basis of existing literature and current evidence-based research, in addition to patient reports obtained through semi-structured qualitative questionnaires (not described in the current paper due to word limit restrictions).

The interventions were identical in all respects, except information regarding the etiology of AN. Consistent information presented to both groups included classification diagnostic criteria of EDs (including proposed DSM-5 changes); comprehensive assessment

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strategies; the impact of starvation (e.g., Minnesota Starvation Study); prevalence rates; mean illness duration; mortality rates; comorbid physical and psychological conditions; myth busting; physical and psychological comorbidities; medical complications and management; importance of early identification and treatment; multidisciplinary treatment options; and empathic communication skills.

In terms of differing etiological information, the biogenetic intervention emphasised factors such heritability rates, candidate genes, structural and neurochemical changes, inherit temperament, neurobiological differences (e.g., attentional biases, weak central coherence), endophendotypes (i.e., symptom overlap with Autism Spectrum Disorders and Anxiety Disorders), and the cycle of risk (e.g., prenatal factors, obstetric complications, and post-natal influences). In the traditional multifactorial intervention, the interaction between the biogenetic, psychological, and social factors was emphasised in the context of a gene-environment (diathesis-stress) model.

Prior to intervention delivery, the interventions were reviewed by four independent sources to ensure any errors were identified, comprehensibility was assessed, and corrections could be made. This process was repeated twice. A copy of the interventions may be obtained by request.

Causal Attributions. Participants were asked to indicate on a 7-point Likert scale (1 = *does not contribute at all* to 7 = *main contributing factor*) the extent to which nine different factors contribute to the development of AN. In line with previous research (e.g., Bannatyne & Abel, 2014), the Causal Attributions Scale (Crisafulli et al., 2008) was separated into two subscales for analysis: biogenetic attributions and sociocultural attributions. An average score for each subscale was created for analysis. Previous research (e.g., Bannatyne & Abel, 2014) has reported good internal consistencies ranging from .82 (sociocultural) to .84 (biogenetic). Reliability analyses for the current study revealed similar internal consistencies (.86 and .83,

respectively).

Opinions. Participants' attitudes toward individuals with AN were assessed using the Opinions Scale (Stewart et al., 2006). Participants were asked to indicate their degree of agreement with five stigmatising statements on a 5-point Likert scale (1 = *strongly disagree* to 5 = s*trongly agree*). The five items reflected stigmata such as responsibility (e.g., "*are to blame for their condition*"), as well as fear and exclusion (e.g., "*are a danger to others*"). Higher scores were indicative of more negative opinions. The Opinions Scale was analysed at a subscale level, with average scores for each subscale used in analysis. Previous research (e.g., Bannatyne & Abel, 2014) has reported internal consistencies of .68 for Fear and Exclusion, and .86 for Responsibility. Reliability analyses for the current study revealed similar internal consistencies (.69 and .81, respectively).

Eating Disorder Stigma. Participants were asked to complete the Eating Disorder Stigma Scale (Crisafulli et al., 2010), a self-report measure designed to assess a variety of beliefs people may hold about AN. Participants were asked to indicate the extent to which they agreed with each statement on a 5-point Likert scale (1 = *strongly disagree* to 5 = *strongly agree*). The ED Stigma Scale contained 20-items reflecting stigmata such as trivialisation, selfish/vain, weak, and blame. Research has indicated the ED Stigma Scale is a psychometrically sound instrument with internal consistencies ranging from .90 for the full scale, and .80 to .89 for the subscales (Bannatyne & Abel, 2014, Crisafulli et al., 2010). For the purpose of the study, the ED Stigma Scale was analysed at the full scale and subscale level, with reliability analyses revealing similar internal consistencies (.81 to .95) to previous research.

Procedure

Participants were recruited via the Bond University School of Medicine, with the educational interventions delivered during structured teaching periods in the first week of an

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eight-week clinical paediatrics rotation. Selection of this rotation was based on the likelihood of interaction with ED sufferers. Prior to the first author delivering the educational interventions, participants were asked to read an explanatory statement, provide informed consent, and complete a pre-intervention package consisting of demographic questions and items from the dependent variables. To enable data matching, each participant developed a de-identified code. The total intervention period lasted for approximately three hours, consisting of didactic and exploratory learning methods.

For the intervention groups, participants were asked to complete a post-intervention questionnaire, identical in all respects to the pre-test questionnaire, with the addition of a treatment evaluation. Contact information for counselling services at various locations was provided and participants were given the option to have their responses withdrawn. The first author returned eight-weeks later, with participants asked to complete a follow-up questionnaire, identical in all respects to the post-test questionnaire (excluding the treatment evaluation). A debrief statement was also provided. For participants in the control group, the first author followed the same procedure, attending the structured teaching period pre-intervention and at follow-up (8-weeks later). Participants in the control group were advised the researcher was investigating "attitudes and perceptions of mental illness" and were asked to complete an identical questionnaire to the intervention groups.

Results

The data were analysed using SPSS version 21. An alpha level of .05 was utilised to determine the statistical significance of all results. Due to word limit restrictions, only significant findings are presented. The means and standard deviations for each group (across measurement points) can be seen in Table 1. Between groups comparisons can also be found in Table 1. Within groups comparisons are shown in Table 2 (only variables with a significant univariate effect listed).

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INSERT TABLE 1 HERE

INSERT TABLE 2 HERE

Stigmatisation MANOVA

With the use of Wilk's criterion, a significant multivariate interaction between Time and Intervention Type was revealed $F(12, 64) = 1.86$, $p = .038$, partial $\eta^2 = .27$, power = .88.

Blame. Univariate analyses revealed a significant interaction effect on blame. No significant differences between groups were found at pre-intervention in terms of level of blame; however, significant differences between groups were observed at follow-up. Post-hoc Tukey's analyses revealed the biogenetic ($p = < .001$) and multifactorial ($p = .003$) groups exhibited significantly lower levels of blame toward AN than the control group; however, no significant differences between the biogenetic and multifactorial groups were observed at follow-up ($p = .595$). Similarly, there were no significant differences in blame between the biogenetic and multifactorial groups immediately post-intervention. Across time, there were no significant differences in level of blame from pre-intervention to follow-up for the control group. For the biogenetic intervention, significant differences over time were observed. Pairwise comparisons with Sidak adjustment revealed the level of blame assigned to AN sufferers reduced significantly from pre-intervention to post-intervention, with this reduction maintained at follow-up. Significant reductions in blame were also found for the multifactorial group from pre-intervention to post-intervention, with this decrease in blame maintained at follow-up.

Selfish/Vain. Univariate analyses revealed a significant interaction effect on the selfish/vain subscale. No significant differences in the perception of AN as a selfish/vain illness were observed between groups at pre-intervention. Significant differences were, however, revealed between groups at follow-up. Post-hoc Tukey's analyses revealed the control group perceived AN to be a selfish and vain illness to a significantly greater extent

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than the biogenetic (p = < .001) and multifactorial (p = .001) groups. No significant differences between the biogenetic and multifactorial groups were observed at follow-up (p = .181). Results did, however, reveal a significant difference between the biogenetic and multifactorial groups immediately post-intervention, with the biogenetic group exhibiting significantly lower selfish/vain scores than the multifactorial group. Across time, no significant changes in selfish/vain scores were observed from pre-intervention to follow-up for the control group. Significant differences in selfish/vain scores were found for the biogenetic intervention, with perceptions of AN as a selfish or vain illness decreasing significantly from pre-intervention to post-intervention, and maintained at follow-up. Unlike the biogenetic intervention, results of the multifactorial intervention revealed the decreases in selfish/vain scores over time were non-significant.

Responsibility. Univariate analyses revealed a significant interaction effect on responsibility. At pre-intervention, no significant differences were observed between groups in terms of perceived responsibility; however, significant differences between groups were found at follow-up. Post-hoc Tukey's analyses revealed the control group exhibited a greater perception of illness responsibility compared to the biogenetic (p = < .001) and multifactorial (p = .004) groups. No significant differences were observed between the biogenetic and multifactorial groups at follow-up (p = .172), or immediately following delivery of the interventions.

Across time, no significant differences in perceptions of responsibility were observed for the control group. For the biogenetic group, significant differences across time were found. Pairwise comparisons with Sidak adjustment revealed perceptions of responsibility did not significantly reduce from pre-intervention to post-intervention; however, responsibility scores were significantly lower at follow-up than pre-intervention. For the multifactorial group, significant differences across time were observed, with perceptions of responsibility

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decreasing significantly from pre-intervention to post-intervention. While a significant increase in responsibility scores was observed in the multifactorial group between post-intervention and follow-up, the follow-up scores were still significantly lower than pre-intervention (p = .045).

Total ED Stigma Mixed ANOVA

Due to a high level of multicollinearity with the EDSS subscales, the total ED stigma scale score was evaluated in a single mixed ANOVA. Results revealed a significant multivariate interaction between Time and Intervention. No significant differences in total ED stigma were observed between groups at pre-intervention. Significant differences were, however, revealed between groups at follow-up. Post-hoc Tukey's analyses revealed the biogenetic (p = < .001) and multifactorial (p = .001) groups, exhibited significantly lower total ED stigma scores compared to the control group. No significant differences in total ED stigma scores were observed between the biogenetic and multifactorial groups at post-intervention or follow-up (p = .180).

In the control group, no significant decreases in total ED stigma were observed from pre-intervention to follow-up, with results revealing scores actually appeared to increase over time; however, this increase was non-significant. For the biogenetic intervention, a significant difference in total ED stigma across time was found. Pairwise comparisons with Sidak adjustment revealed a significant decrease in total ED stigma from pre-intervention to post-intervention, with this effect maintained at follow-up. Unlike the biogenetic intervention, no significant differences across time were observed for the multifactorial intervention, however scores did appear to decrease from pre-intervention to post-intervention and follow-up.

Causal Attributions MANOVA

With the use of Wilk's criterion, a significant multivariate interaction between Time

and Intervention Type was revealed $F(4, 72) = 3.62$, $p = .010$, partial $\eta^2 = .17$, power = .85.

Sociocultural Attributions Univariate analyses revealed a significant interaction effect on sociocultural attribution. Between groups, no significant differences in the endorsement of sociocultural etiological factors were observed at pre-intervention or post-intervention. A significant difference between groups was observed at follow-up. Post-hoc Tukey's analyses revealed no significant differences between groups, however differences between the control and biogenetic group approached significance ($p = .053$). Across time, no significant differences in the endorsement of sociocultural etiologic factors were observed for the control group. Significant differences in sociocultural causal attributions were, however, observed for the biogenetic group. Pairwise comparisons with Sidak adjustment revealed a significant reduction in sociocultural attributions from pre-intervention to post-intervention, with this effect maintained at follow-up. For the multifactorial group, significant reductions in sociocultural causal attributions were also observed from pre-intervention to post-intervention, with this effect maintained at follow-up.

Biogenetic Attributions. Univariate analyses revealed a significant interaction effect on biogenetic attribution. Between groups, no significant differences in the endorsement of biogenetic factors were observed at pre-intervention. Significant differences between groups were found at follow-up, with the biogenetic ($p = .002$) and multifactorial ($p = .030$) groups making significantly stronger biogenetic causal attributions compared to the control group. No significant differences in the endorsement of biogenetic factors were observed between the biogenetic and multifactorial at post-intervention or follow-up ($p = .995$). Across time, no significant differences in the endorsement of biogenetic factors were seen for the control group. For the biogenetic intervention, significant increases in biogenetic causal attributions were observed from pre-intervention to post-intervention, with the effect maintained at follow-up. Significantly greater biogenetic attributions were also observed for the

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multifactorial group from pre-intervention to post-intervention, with this effect maintained at follow-up.

Discussion

The present study contributes to the growing body of literature highlighting the need for, and benefit of, greater ED education and training for health professionals, particularly entry-level clinicians. The results, which partially supported hypotheses, indicated participants in both intervention conditions viewed AN differently following an educational workshop. Findings of the current study also contribute to empirical research evaluating the use of etiological framing as a potential stigma reduction method. Partially consistent with expectations, the results suggest education from a biogenetic or multifactorial framework is equally effective and more beneficial than an absence of ED education altogether.

Consistent with previous research, participants, as a group, displayed a high level of ambivalence in terms of specific ED stigma at pre-intervention (e.g., perceptions of responsibility and blame, consideration of the illness as selfish/vain and trivial), highlighting the level of volitional stigma students/entry-level clinicians may enter into a clinical environment holding. Similarly, participants had a general propensity to attribute sociocultural factors as the primary etiology for AN at pre-intervention, with biogenetic factors contributing only "occasionally". In medicine, beliefs regarding the etiology of conditions are typically established through scientific method and decision theory (Hamm, 2009), however despite recent empirical research consistently highlighting the contribution of biological and genetic factors in the development of AN, there appeared to be a general lack of knowledge and/or poor endorsement of these factors in the current sample, consistent with findings of previous research (e.g., Bannatyne & Abel, 2014; Crisafulli et al., 2008; Crisafulli et al., 2010).

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In understanding the impact of the interventions, it was found that participants who received either the multifactorial or biogenetic intervention exhibited significantly lower volitional stigma at follow-up, compared to participants who received no education (control group). Specifically, intervention participants had significantly lower total ED stigma scores, levels of blame, perceptions of AN as a selfish/vain illness, and viewed sufferers' as less responsible for their illness. Intervention participants also demonstrated greater endorsement and acknowledgement of biogenetic factors in the development of AN, and a reduction in sociocultural attributions, which may explain the observed decrease in volitional stigma scores, consistent with the propositions of attribution theory. Possibly educating students about biogenetic etiological factors promoted the belief individuals with AN are less capable of controlling their behaviour, thus reducing interpretations of the illness as a 'choice' or behavioural issue (Link et al., 2004).

While significant differences were revealed on several elements of stigma, it should be noted no significant differences were observed for the weak subscale of the ED Stigma Scale; however, participants, as a group, displayed very minimal endorsement of this subscale at pre-intervention. Similarly, the non-significant difference observed for fear and exclusion is consistent with previous research and supports the proposition EDs are less likely to trigger fear-based stigma compared to other psychological conditions (e.g., schizophrenia), due to perceptions of control; however, are significantly more likely to elicit blame-based stigma (Link et al., 1997).

Given the paucity of literature regarding the optimal frequency of education, it was important to determine whether interventions effects were maintained over time (i.e., 8 weeks) and if one intervention had a greater lasting impact. As expected, intervention effects observed in the biogenetic group (e.g., lower total ED stigma, reduced blame, lower selfish/vain scores, reduced responsibility, greater biogenetic attribution, lower sociocultural

attribution) were maintained over time, suggesting good retention of information and/or modification of attitudes and beliefs. For the multifactorial intervention, this hypothesis was only partially supported, with temporal stability observed for reduced blame, and greater biogenetic attribution. An increase in responsibility scores was observed from post-intervention to follow-up, however follow-up scores for this group were still significantly lower than pre-intervention and participants were still within a range of disagreement. Similarly, scores of the multifactorial group were still significantly lower than the control group, as expected.

Whilst stigmatising attitudes toward mental illness may be influenced by deficits in knowledge (Dyduch & Grzywa, 2009; Wolff, Pathare, Craig, & Leff, 1999), it has also been suggested greater contact with individuals suffering from mental illness can result in more positive attitudes and empathic understanding (Addison & Thorpe, 2004; Ng, Martin, & Romans, 1995). Results of the current study were inconsistent with this suggestion, revealing that participants in the control group showed no change in the level of volitional stigmatisation over the course of the eight-week clinical rotation, where exposure to AN sufferers was obtained. Of interest, a six point increase in overall ED stigma was observed from pre-intervention to follow-up, indicating overall stigma scores actually worsened in the control group; however, this increase was non-significant. Possibly contact challenges other forms of stigma, but does not extend to the volitional stigma associated with AN.

Although results of the current study indicate a greater stigma-reduction effect was achieved by the biogenetic intervention, the difference between groups was non-significant. That is, the two interventions were deemed to be equally effective in terms of stigma-alleviation, both immediately post-intervention and at follow-up. There was one exception to this at post-intervention, with the biogenetic group displaying significantly lower selfish/vain scores; however, the scores of the multifactorial group were still low and the difference was

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non-significant at follow-up. Overall, it appears some form of education in relation to AN is beneficial, rather than no education.

While previous studies (e.g., Bannatyne & Abel, 2014; Crisafulli et al., 2010) have highlighted potentially harmful effects of presenting AN as a multifactorial illness (e.g., increase in perceptions of blame and responsibility), due to attitude accessibility and biased processing of information, results of the current study were inconsistent with these findings. Several factors could account for this, including the nature of the study design (e.g., workshop vs vignettes), which allowed the presenter to ensure participants consolidated all causal factors, rather than processing only sociocultural information presented in the multifactorial explanation. It is possible, however, other presenters may take a more global and unspecified approach (e.g., "AN is caused by numerous factors"), which may be harmful if all factors are not explained in equal detail. It is also important to note, the etiological section of the interventions, which served as the framing approach, formed only a component of the larger intervention, making causal relationships for the destigmatisation effect difficult to determine. Results of the current study are preliminary, therefore replication by other presenters is needed to determine specific program versus presenter effects, and further investigation of specific program components (i.e., the unique impact of each element).

Other limitations are also noted. Firstly, all participants were medical students from the same university and single cohort, therefore generalisability of the results to other medical programs and cohorts is limited, however pre-intervention findings were consistent with a large body of literature highlighting the volitional stigmatisation of AN in entry-level clinicians. Similarly, the sample size was relatively small, thus reducing statistical power, which may explain some of the non-significant differences between and within groups. Future research should endeavour to rectify these sampling limitations by evaluating

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educational programs/interventions in a larger sample, with medical students from a range of programs.

It is also possible results of the current study were influenced by social desirability concerns, given the self-report format of assessing stigma. It has also been argued self-report responses to questionnaires measuring stigma do not always predict real-world behaviours (Link et al., 2004). This was a primary limitation of the study, as the measures employed did not assess participants' knowledge prior to, or following the interventions, therefore changes in knowledge could not be ascertained. Future research should address this concern by including a measure of ED knowledge pre- and post intervention. Similarly, there was no opportunity to observe, measure or evaluate changes in clinical behaviour. It would be important for future research to assess stigma through other methods less affected by social desirability and more predictive of real world behaviours, which may include observations of medical students interacting with ED patients during clinical rounds and discussing ED clients within case-conferencing environments.

Overall, our findings indicated volitional stigma was present within a small sample of medical students, consistent with previous studies, highlighting the clear need for more comprehensive training in EDs, at both a medical program and post-graduate level. Our study also provides preliminary evidence that brief, targeted interventions can assist in significantly reducing levels of volitional stigma, and this effect can be maintained over an eight-week period. Future research is, however, needed to determine the optimal nature, frequency, and format, of this education. Given the wealth of literature highlighting the impact of therapeutic relationships on patient satisfaction and adherence, providing our doctor's of tomorrow with the necessary tools to screen for, diagnose, and treat EDs in humane and dignified manner is a vital step in building clinician competence, removing treatment barriers, and improving future patient care, for this underserved population.

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Table 1

Means and standard deviations for the dependent variables between groups across time, including simple effects analyses for group (i.e., between-groups comparisons).

Variables and Measurement Points	Biogenetic Intervention (n = 15)	Multifactorial Intervention (n = 11)	Control (no intervention) (n = 14)	
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*p*
Trivialisation				.449
1.	1.91 (1.03)	1.91 (.46)	2.19 (.55)	ns
2.	1.78 (.75)	1.91 (.90)	–	ns
3.	1.75 (.47)	1.89 (.60)	2.34 (.47)	ns
Selfish/Vain				.009
1.	2.59 (.95)	2.61 (.66)	3.18 (.82)	.303
2.	1.72 (.51)	2.29 (.83)	–	.042
3.	1.80 (.49)	2.26 (.60)	3.30 (.47)	< .001
Weak				.062
1.	2.27 (.77)	2.18 (.68)	2.26 (.77)	ns
2.	1.79 (.69)	1.98 (.75)	–	ns
3.	1.87 (.48)	1.96 (.70)	2.50 (.57)	ns
Blame				.001
1.	3.00 (.60)	2.89 (.65)	3.20 (.41)	.369
2.	2.10 (.55)	2.27 (.78)	–	.513
3.	2.17 (.53)	2.39 (.79)	3.21 (.39)	< .001
Total ED Stigma				.003
1.	48.67 (14.82)	47.64 (8.83)	50.27 (11.68)	.321
2.	37.20 (11.19)	41.36 (14.89)	–	.423
3.	37.33 (8.72)	42.36 (12.00)	56.86 (7.23)	< .001
Responsibility				.021
1.	2.40 (.60)	2.73 (.72)	2.89 (.88)	.205
2.	2.00 (.73)	1.86 (.39)	–	.581
3.	1.83 (.70)	2.18 (.40)	2.96 (.96)	< .001
Fear & Exclusion				.739
1.	2.23 (.68)	2.73 (.56)	3.04 (.84)	ns
2.	1.97 (.74)	2.73 (.39)	–	ns
3.	1.87 (.69)	2.46 (.52)	2.54 (.72)	ns

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Sociocultural Attribution				.027
1.	5.43 (.99)	5.70 (.64)	5.46 (1.09)	.715
2.	3.71 (1.72)	4.66 (.86)	–	.175
3.	3.70 (1.49)	4.59 (.96)	4.80 (1.09)	.050
Biogenetic Attribution				.042
1.	4.40 (1.14)	4.50 (.89)	4.39 (1.27)	.967
2.	5.37 (1.06)	5.55 (.99)	–	.666
3.	5.38 (.93)	5.41 (1.26)	4.14 (1.34)	.011

Note. 1 = pre-intervention, 2 = post-intervention, 3 = follow-up. p = significance value (to 3 decimal points). ns = non-significant (> .05).

Table 2

Within-groups comparisons for each dependent variable (simple effects analyses for time).

	Biogenetic Intervention	Multifactorial Intervention	Control (no intervention)
Variables and Measurement Points	*p*	*p*	*p*
Blame	< .001	.027	.856
1 → 2	.002	.033	ns
1 → 3	< .001	.047	ns
2 → 3	ns	. ns	ns
Selfish/Vain	< .001	.264	.393
1 → 2	.001	ns	ns
1 → 3	.007	ns	ns
2 → 3	ns	ns	ns
Responsibility	.028	.001	.655
1 → 2	.145	.001	ns
1 → 3	.002	.045	ns
2 → 3	ns	.026	ns
Total ED Stigma	< .001	.159	.072
1 → 2	.008	ns	ns
1 → 3	.008	ns	ns
2 → 3	ns	ns	ns
Sociocultural Attributions	< .001	< .001	.093
1 → 2	.004	.001	ns
1 → 3	.001	< .001	ns
2 → 3	ns	ns	ns
Biogenetic Attributions	.006	.023	.534
1 → 2	.029	.013	ns
1 → 3	.016	.036	ns
2 → 3	ns	ns	ns

Note. 1 = pre-intervention, 2 = post-intervention, 3 = follow-up. *p* = significance value (to 3 decimal points). ns = non-significant (> .05).