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Can a brief biologically-based psychoeducational intervention reduce stigma and increase help-seeking intentions for depression in young people? A randomised controlled trial

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There is disagreement in the literature as to whether biological attribution increases or decreases stigma. This study investigated the effect of an online biological intervention on stigma and help-seeking intentions for depression among adolescents. A three-arm, pre-post test, double-blind randomised controlled trial (RCT) was used to compare the effects of a biological and a psychosocial intervention delivered online. Participants comprised secondary school students ($N = 327$) aged 16–19 years. Outcome measures included anticipated self-stigma for depression (primary), personal stigma, help-seeking intention for depression, and biological and psychosocial attribution. Neither the biological nor the psychosocial educational intervention significantly reduced anticipated self-stigma or personal stigma for depression relative to the control. However, a small increase in help-seeking intention for depression relative to the control was found for the biological educational condition. The study was undertaken over a single session and it is unknown whether the intervention effect on help-seeking intentions was sustained or would translate into help-seeking behaviour. A brief online biological education intervention did not alter stigma, but did promote a small increase in help-seeking intentions for depression among adolescents. This type of intervention may be a practical means for facilitating help-seeking among adolescents with current or future depression treatment needs.

Introduction

Depression is the leading cause of disability worldwide, and the leading contributor to burden of disease in young people aged 18–24 years (AIHW, 2007; WHO, 2016). Given that approximately half of all lifelong mental disorders begin before the age of 14, and three-quarters by the age of 24 (Rickwood, Deane, & Wilson, 2007; Zubrick, Silburn, Burton, & Blair, 2000), targeting mental health prevention and intervention initiatives at an early stage in the lifespan (e.g., adolescence) is a major public health priority. However, one of the biggest challenges for effective early intervention is the reluctance of young people to seek help for their mental health problems. Young people are considerably less likely than any other group in the population to seek professional help for depression or anxiety (Rickwood et al., 2007). As delays in seeking treatment have a negative impact on treatment outcomes for mental disorders (Boyd et al., 2007), understanding and addressing the barriers to help-seeking for depression among adolescents is critical for improving mental health outcomes.

One barrier to help-seeking for depression is the stigma associated with the disorder itself (Barney, Griffiths, Jorm, & Christensen, 2006; Gulliver, Griffiths, & Christensen, 2010). Educational interventions have decreased the stigma associated with depression in adult populations (Griffiths, Carron-Arthur, Parsons, & Reid, 2014). However, little is known about what types of educational content are most effective in destigmatisation interventions (Griffiths & Christensen, 2004). One mechanism theorised to play a role is that of attributing one's depression to biological factors (i.e., biological attribution) (Han, Chen, Hwang, & Wei, 2006). Biological attribution may help decrease stigma by reducing the perceived controllability of the condition and thereby increase willingness to seek professional help. In contrast, several investigators have argued that biological explanations of mental disorders are counterproductive and likely to increase stigma by reinforcing fear, prejudice, and a desire for distance (Read, Haslam, Sayce, & Davies, 2006). However, most of the foregoing debate has been informed by cross-sectional studies of the relationship between biological attribution and stigma. These studies have yielded mixed findings, with some reporting that biological attribution is associated with greater depression stigma (Breheny, 2007), and others demonstrating either no relationship (Jorm & Griffiths, 2008), or that biological attribution is associated with lower stigma (Cook & Wang, 2011).

Only four studies have investigated the effect of changing biological attributions on stigma and/or help-seeking outcomes, with mixed findings (Boucher & Campbell, 2014; Han & Chen, 2014; Han et al., 2006; Rusch, Kanter, & Brondino, 2009). For example, one intervention study conducted among university students reported that a brief biologically-based educational intervention decreased public stigma (measured via social distance) relative to a no-intervention control condition (Han & Chen, 2014). Another found a greater increase in participants' willingness to seek professional help for depression among university students receiving a biological explanation of depression compared to an intervention group receiving a psychosocial explanation (Han et al., 2006). In contrast, experimental and intervention studies have reported that biologically-based anti-stigma messages (Boucher & Campbell, 2014) and biomedical educational interventions (Rusch et al., 2009) are ineffective in reducing stigma or increasing informal and formal help-seeking among university students.

However, a major limitation of empirical studies to date is that they have focused mainly on the link between causal attribution and public attitudes to depression or mental illness rather than on internalised stigma (i.e., the views the person holds about their *own* illness). This is an important distinction, as researchers have raised the possibility that causal attributions might impact differently on internalised stigma by simultaneously increasing the desire for social distance among members of the community, whilst decreasing self-stigma among consumers with depression, leading to increased help-seeking behaviour (Griffiths & Christensen, 2004). Moreover, it is clear that self-stigma plays a particularly critical role in help-seeking (Barney et al., 2006; Clement et al., 2015).

In addition, although adolescence has been identified as a period of high risk for first onset of mental health problems, to date no study has examined whether attributing depression to biological causes has an effect on stigmatising and help-seeking intentions or behaviours in school-aged students.

This study aimed to undertake a randomised controlled trial (RCT) to determine the effect of a brief online educational intervention, designed to increase biological causal attribution for depression, on anticipated self-stigma (anticipated negative attitudes to depression in oneself), personal stigma (negative attitudes to depression in general), and help-seeking for depression among adolescents. The intervention was based on that used in the study by Han et al. (2006) and comprised a vignette of a person with major depression together with information on the biological causes of depression. To investigate whether any changes in stigma or intended help-seeking for depression were specific to a biological explanation for depression, a second intervention comprised information on psychosocial causes of depression outside the control of the individual. Both interventions were compared to a control condition that contained neutral information. Finally, since research evidence exists that suggests that self-stigma can adversely affect attitudes towards help-seeking, and that this in turn can directly affect help-seeking intentions (Bathje & Pryor, 2011; Vogel, Wade, & Hackler, 2007), a mediation analysis was undertaken to investigate if any changes in help-seeking associated with the biological education intervention were attributable to changes in self-stigma.

Methods

Trial design

This study used a double-blind, parallel-group trial design with randomisation and control. The trial was conducted at two sites within Australia, and consisted of three educational conditions: (1) a biological condition which comprised educational information describing the biological causes of depression; (2) a psychosocial condition which comprised educational information describing the psychosocial causes of depression; and (3) a control condition containing neutral information on the symptoms of depression adapted from an online information page for depression (<http://bluepages.anu.edu>) for site 1, and information on physical health (<http://beactive.com.au>) for site 2. Each of the interventions comprised one page of text delivered online. The text incorporated a vignette of a person who met the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria for major depression together with educational information on the causes of depression. Each condition incorporated either a male or a female vignette, resulting in six possible condition combinations. Participants were randomly assigned to a condition by means of a sealed envelope containing one of six access codes. A research assistant, not otherwise involved in the project, prepared the envelopes based on a computer-generated list. These in turn were distributed in the classroom in a fixed order by KH. Interim analyses for efficacy were undertaken after the trial was completed at site 1. These raised the possibility that the control condition had an effect on the outcome measures. Based on these interim analyses, the site 2 control condition was modified to provide general health information rather than information on depression.

Participants and procedure

Participants consisted of 327 senior high school students aged 16 years and over, recruited from 2 independent Catholic schools in the Australian Capital Territory (site 1; 18 June 2009) and regional New South Wales (site 2; 21 August 2009). The study comprised an online baseline survey, the online intervention or control condition, and a post-intervention online survey. Each participant completed all 3 components of the study in a single 40-minute session during school class time. Passive parental consent was obtained by providing parents with an information sheet about the study, and a form through which they could decline consent for their child's participation. Students also provided online informed consent before study participation. The relevant institutional review board approved the study (trial registration ID: ACTRN12609000497202).

Power calculation

Our planned sample size of 330 was estimated to detect an effect size of 0.4 with at least 80% power and an alpha of 0.05, allowing for 10% attrition (i.e., final sample of 300). This power calculation was updated using the study data (i.e., final group sizes, baseline data on means and standard deviations for each measure, and correlations between the pre- and post-test scores, for the relevant conditions) to confirm there was at least 80% power to detect a between group difference of 0.3 with an alpha of 0.05.

Measures

Outcomes and related variables were assessed online immediately before the intervention (pre-test) and immediately after completing the intervention (post-test). The online survey comprised measures of self-stigma (primary outcome), personal stigma, help-seeking intentions, biological attribution, and psychosocial attribution. In addition, level of depressive symptoms and demographic status were measured at baseline.

Stigma

Anticipated internalised stigma was measured using the 16-item Self-Stigma for Depression Scale (SSDS; Barney, Griffiths, Christensen, & Jorm, 2010) (primary outcome measure). The items required the participant to indicate how they would think or feel if they were depressed. Each item scored on a 4-point Likert scale from 1 to 4 (strongly agree to strongly disagree) with total scores ranging from 16 to 80, and higher scores indicating greater self-stigma. Cronbach's alpha for the

SSDS in our study was 0.88 at baseline indicating good internal reliability. Personal stigma (also described in the literature as public stigma) was measured using the personal subscale of the Depression Stigma Scale (DSS; Griffiths, Christensen, & Jorm, 2008). Each item required students to indicate their attitude to a person with depression. Cronbach's alpha for the DSS-Personal in this study was 0.79 at baseline, indicating adequate internal reliability.

Help-seeking intentions

Intentions to seek help for depression were measured using the General Help-seeking Questionnaire (GHSQ; Wilson, Deane, Ciarrochi, & Rickwood, 2005). This measure comprises nine items in which respondents rate the likelihood that they would seek help from a particular source on a seven-point scale ranging from 1 ("extremely unlikely") to 7 ("extremely likely"). The sources of help were: (1) partner (e.g., significant boyfriend or girlfriend); (2) friend (not related to you); (3) parent; (4) other relative/family member; (5) mental health professional (e.g., school counsellor, psychologist, psychiatrist); (6) phone help line (e.g., Lifeline, Kids Help Line); (7) family doctor/general practitioner; (8) teacher (year adviser, classroom teacher); and (9) help from someone else not listed. Higher scores indicate greater help-seeking intention with potential scores ranging from 7 to 63. Cronbach's alpha for the GHSQ in our study was 0.73 at baseline, indicating acceptable internal reliability.

Causal attribution

To confirm that the intervention was modifying participants' biological and psychosocial causal attribution for depression, these constructs were measured using an adaptation of the Han et al. (2006) Biological Attribution Scale (BAS) and Psychological Blame Scale (PBS) respectively. These scales comprise five items each scored on a five-point Likert scale, with higher scores indicating higher attribution. Cronbach's alphas for the Biological Attribution and Psychosocial Attribution scales were 0.60 and 0.86, indicating poor and good internal reliabilities respectively.

Depressive symptoms

Depressive symptoms were assessed online before the intervention using the Center for Epidemiological Studies Depression scale (CES-D; Radloff, 1977), a 20-item self-report scale which yields scores ranging from 0 to 60 with higher scores indicating more severe depressive symptoms.

Demographic status

Age, years of schooling, and gender were measured before the intervention.

Statistical analysis

Data were analysed using Stata Version 11.1. The effect of each intervention on each outcome measure (GHSQ, SSDS, DSS, BAS and PBS scores) was established using a time (pre-test versus post-test) by intervention (intervention group versus control group) interaction in a random effects regression model with robust standard errors. A random intercept was used for individuals over time and variance was clustered on school. A between group effect size was calculated for each intervention relative to control by computing the difference between the within group Cohen's *d* effect sizes for the intervention and the control groups. Skewed variables were normalised using a Box-Cox transformation implemented with the Stata `lnskew0` command.

The study also aimed to investigate if any increase in help-seeking intention for depression produced by the biological causal attribution intervention was mediated by reduced self-stigma for depression. According to the principles established by Baron and Kenny (1986), the identification of change in self-stigma as a mediator of help-seeking intention change requires evidence of the following: (a) a significant relationship between the biological causal attribution intervention and anticipated self-stigma (SSDS score) for depression (i.e., pathway A in Figure 1); (b) a significant relationship between the biological causal attribution intervention for depression and help-seeking (GHSQ score; pathway C in Figure 1); and (c) a significant relationship between anticipated self-stigma (SSDS score) and help-seeking intention (GHSQ score) while adjusting for the

biological causal attribution for depression intervention (pathway B in Figure 1). In addition, (d) the relationship between the biological causal attribution intervention for depression and help-seeking intention (pathway C in Figure 1) needed to be reduced (or eliminated) by adjusting for anticipated self-stigma. The biological intervention was a fixed (time-invariant) effect in the analysis. Both the anticipated self-stigma scores and the help seeking scores were time-varying (included in the analysis at both time points). The mediation effect was tested using the following analyses: pathway C was tested using a biological causal attribution intervention (intervention vs. control condition) \times time (pre- versus post-test) interaction effect on anticipated self-stigma (SSDS score), to determine if the biological causal attribution intervention decreased anticipated self-stigma (as per the main analysis). Pathway A was tested using a biological causal attribution intervention (intervention vs. control condition) \times time (pre- versus post-test) interaction effect on help-seeking (GHSQ score), to determine if the biological causal attribution intervention increased help-seeking (as per the main analysis). Pathway B was tested using a self-stigma (SSDS score) \times time (pre- versus post-test) interaction effect on help-seeking score (GHSQ score) to determine if any reduction in stigma at post-test increased help-seeking. Finally, we tested whether the relationship between the biological causal attribution intervention and help-seeking intention (pathway C in Figure 1) was attenuated or eliminated by adjusting for self-stigma by including self-stigma as a covariate in the analysis used to test pathway C. All tests were two-sided with significance set at $p < 0.05$.

Results

Participants

Of the 351 students who were eligible to participate, 1 did not consent to the study, 19 failed to provide baseline data on the primary outcome variable, and a further 4 dropped out at post-test (Figure 2). The final sample ($N = 327$) had a median age of 16 years (range 16–19 years); most (80%) were in year 11, and 47% were female. A total of 39% exceeded the CES-D cut-off for clinically significant depressive symptoms (score 16+), with around half of these, or 17% of the entire sample, exceeding the cut-off for major depression (score of 27 or greater). Neither CES-D scores nor other demographics, differed significantly between the intervention conditions (Table 1).

Changes in causal attribution for depression

The mean (SD) BAS and PBS scores for each condition pre-test and post-test are presented in Table 2. As expected, there was a significant increase in biological and psychosocial attribution for depression within the biological and psychosocial conditions respectively (Table 3). Conversely, there was no significant change in biological attribution in either the psychosocial condition or the control condition, nor was there a significant change in psychosocial attribution in either the biological condition or the control condition (Table 3). The between group difference in the pre-post effect sizes (Cohen's d) for the biological intervention and control condition was 0.79 for biological attribution and

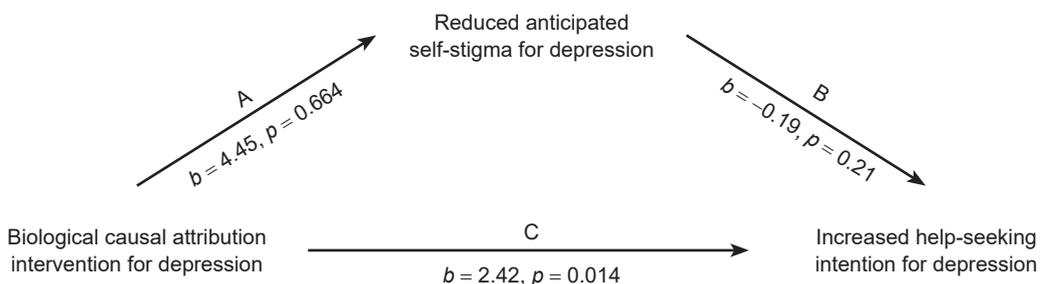


Figure 1: Causal diagram showing the relationship between biological causal attribution for depression, anticipated self-stigma for depression and help-seeking for depression

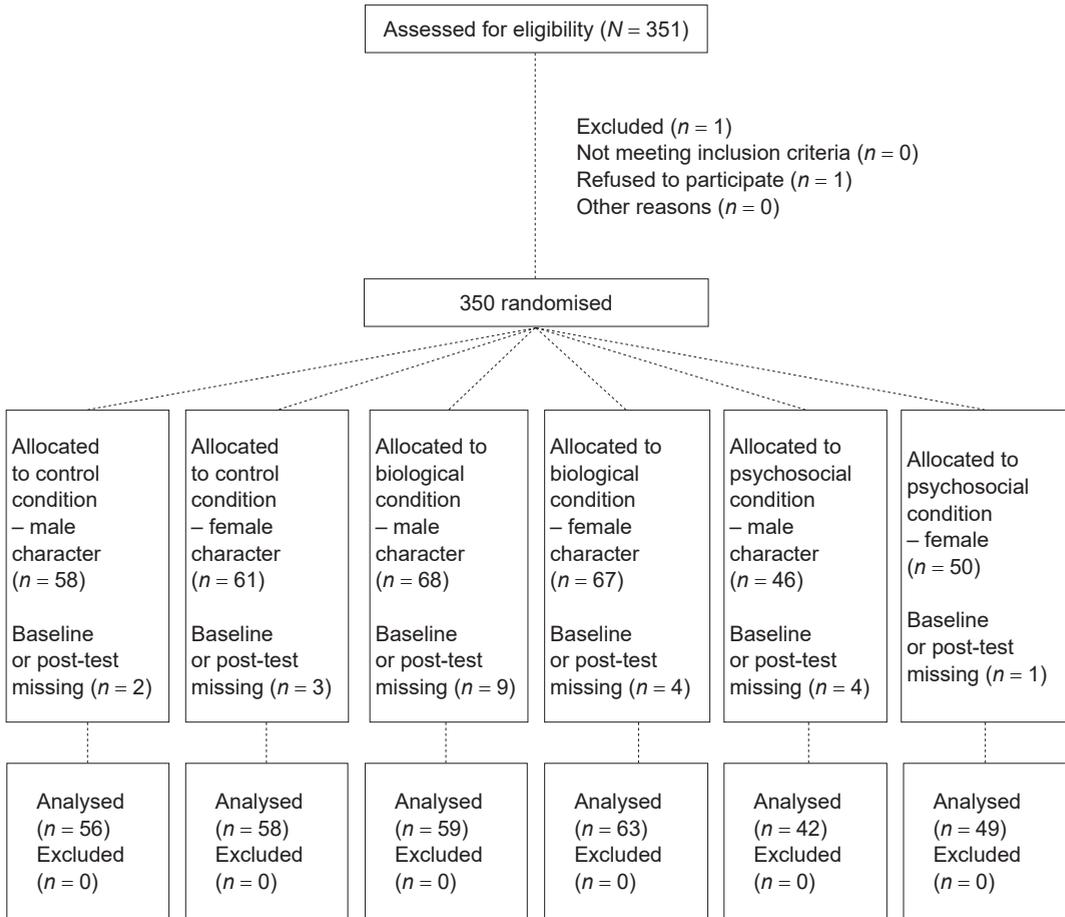


Figure 2: Participant flow through the trial

Table 1: Characteristics of participants by condition

	Control condition (n = 114)	Biological condition (n = 122)	p	Psychosocial condition (n = 91)	p	Total sample (N = 327)
Female (%)	45	47	0.760	51	0.408	47
School 2, JPC (%)	29	39	0.120	37	0.202	35
Year 12 (%)	18	17	0.808	24	0.315	20
Age (median)	16	16	0.854	17	0.416	16
CES-D score (mean)	15.7	13.8	0.125	15.8	0.881	12
SSDS score (mean)	11.6	12.8	0.105	12.1	0.470	12.2
GHSQ score (mean)	29.8	28.3	0.200	26.7	0.010	28.4
DSS score (mean)	55.9	55.4	0.740	56.3	0.813	55.8

Comparisons relative to the control condition. Dichotomous variables were compared using a Pearson’s chi square test; medians were compared using an equality of medians test, and means using a t-test. CES-D scores were normalised.

0.06 for psychosocial attribution. The corresponding between group effects for the psychosocial and control conditions were 0.25 and -0.03 for psychosocial and biological attribution respectively.

Stigma associated with depression

There was an overall reduction in anticipated self-stigma (Figure 3) and personal stigma at post-test in all three conditions (Table 2). However, neither the biological nor the psychosocial conditions had a significant effect on anticipated self-stigma or personal stigma relative to the control group (Table 4).

Table 2: Mean levels of biological and psychosocial attribution for the cause of depression, self-stigma, personal stigma, and help-seeking before and after the interventions

	Pre-test Mean (SD)	Post-test Mean (SD)
Biological attribution (BAS score)		
Control condition	16.2 (3.2)	16.6 (3.8)
Biological condition	16.2 (2.9)	19.3 (3.9)
Psychosocial condition	16.5 (3.1)	16.8 (4.0)
Psychosocial attribution (PBS score)		
Control condition	20.0 (3.7)	20.3 (3.8)
Biological condition	20.0 (3.3)	20.2 (3.6)
Psychosocial condition	20.1 (4.0)	21.1 (4.1)
Self-stigma (SSDS score)		
Control condition	55.89 (11.25)	54.17 (12.77)
Biological condition	55.43 (9.97)	54.16 (10.21)
Psychosocial condition	56.25 (10.68)	55.34 (12.00)
Personal stigma (DSS score)		
Control condition	11.55 (5.34)	10.92 (6.03)
Biological condition	12.77 (6.10)	12.25 (6.43)
Psychosocial condition	12.14 (6.31)	11.56 (6.22)
Help-seeking (GHSQ score)		
Control condition	29.8 (8.4)	31.0 (10.2)
Biological condition	28.3 (9.6)	31.9 (9.9)
Psychosocial condition	26.7 (8.7)	30.7 (10.5)

SD = standard deviation

Table 3: Regression results for the effect of the biological and psychosocial conditions on biological attribution and psychological attribution

	B (Robust SE)	p
Biological attribution (BAS score)		
Biological condition	-0.0 (0.3)	0.934
Time	0.4 (0.4)	0.365
Biological condition x time	2.7 (0.7)	<0.001
Psychological condition	0.3 (0.8)	0.696
Time	0.4 (0.4)	0.365
Psychological condition x time	-0.1 (0.2)	0.482
Psychological attribution (PBS score)		
Biological condition	0.0 (0.9)	0.972
Time	0.3 (0.1)	0.002
Biological condition x time	-0.1 (0.2)	0.616
Psychological condition	0.1 (0.9)	0.925
Time	0.3 (0.1)	0.002
Psychological condition x time	0.8 (0.2)	<0.001

SE = standard error

Help-seeking for depression

There was an overall increase in help-seeking at post-test in all three conditions (Figure 3). However, the biological condition produced a small, yet significant increase in help-seeking ($d = 0.24$) relative to the control condition at post-test (Table 4). The psychosocial condition produced a similar magnitude of effect ($d = 0.28$) relative to the control group, but this effect was

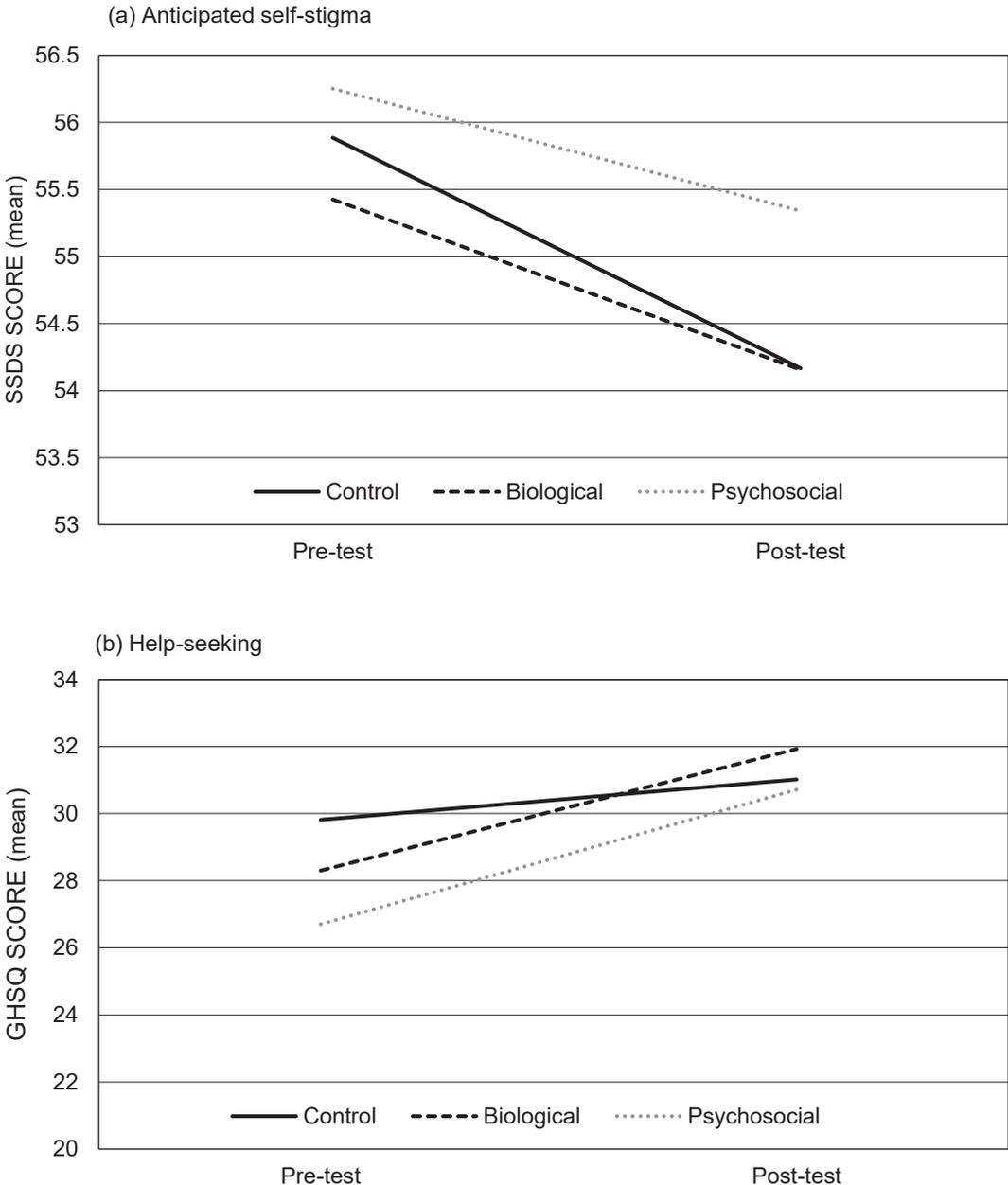


Figure 3: The effect on the intervention on anticipated self-stigma and help-seeking intentions

not statistically significant (Table 4). Adjusted results presented in Table 4 show that these effects persisted after adjustment for age, sex, school and baseline depression (normalised score on the CES-D). The effect of the biological condition on help-seeking was not moderated by participants' level of depression, as evidenced by the lack of a significant three-way interaction between the

Table 4: Regression results for the effect of the biological and psychosocial conditions on self-stigma, personal stigma, and help-seeking relative to the control group

	Unadjusted		Adjusted	
	B (Robust SE)	p	B (Robust SE)	p
Help-seeking (GHSQ)				
Biological condition	-1.5 (0.6)	0.010	-1.7 (0.6)	0.007
Time	1.2 (0.4)	0.004	1.2 (0.4)	0.004
Biological condition × time	2.4 (1.0)	0.014	2.4 (1.0)	0.015
Age (years)			-0.3 (1.9)	0.867
Sex (female)			-1.1 (0.9)	0.199
School (site 2)			0.5 (0.0)	<0.001
Normalised CES-D score			-2.1 (0.3)	<0.001
Psychological condition	-3.1 (0.4)	<0.001	-3.1 (0.2)	<0.001
Time	1.2 (0.4)	0.004	1.2 (0.4)	0.004
Psychological condition × time	2.8 (1.8)	0.109	2.8 (1.8)	0.111
Age (years)			-0.7 (2.0)	0.739
Sex (female)			-0.8 (0.4)	0.030
School (site 2)			1.2 (0.2)	<0.001
Normalised CES-D score			-2.3 (0.7)	<0.001
Self-stigma (SSDS)				
Biological condition	-0.5 (0.1)	<0.001	-0.5 (0.0)	<0.001
Time	-1.7 (0.7)	0.012	-1.7 (0.7)	0.012
Biological condition × time	0.4 (1.0)	0.664	0.4 (1.0)	0.665
Age (years)			-1.5 (1.5)	0.303
Sex (female)			2.6 (1.9)	0.186
School (site 2)			0.9 (0.1)	<0.001
Normalised CES-D score			0.2 (0.7)	0.748
Psychological condition	0.4 (1.2)	0.758	0.4 (1.8)	0.834
Time	-1.7 (0.7)	0.012	-1.7 (0.7)	0.012
Psychological condition × time	0.8 (1.3)	0.548	0.8 (1.4)	0.550
Age (years)			-0.9 (1.3)	0.484
Sex (female)			2.9 (3.0)	0.331
School (site 2)			-0.8 (0.2)	<0.001
Normalised CES-D score			2.0 (0.2)	<0.001
Personal stigma (DSS)				
Biological condition	1.2 (1.0)	0.228	1.4 (0.9)	0.136
Time	-0.6 (0.2)	<0.001	-0.6 (0.2)	<0.001
Biological condition × time	0.1 (0.3)	0.741	0.1 (0.4)	0.742
Age (years)			0.6 (1.3)	0.672
Sex (female)			-2.0 (1.1)	0.074
School (site 2)			-1.0 (0.1)	<0.001
Normalised CES-D score			0.2 (0.3)	0.468
Psychological condition	0.6 (1.4)	0.683	0.6 (1.4)	0.692
Time	-0.6 (1.2)	0.000	-0.6 (0.2)	<0.001
Psychological condition × time	0.0 (0.4)	0.908	0.0 (0.4)	0.908
Age (years)			0.9 (1.2)	0.418
Sex (female)			-1.4 (1.3)	0.259
School (site 2)			-0.4 (0.1)	<0.001
Normalised CES-D score			0.4 (0.1)	<0.001

SE = standard error; model adjusted for baseline covariates of age, sex, school and normalised CES-D score

effect of baseline depression (normalised score on the CES-D) and the intervention effect (e.g., pre- versus post-test interaction with condition) ($p > 0.05$).

Mediation of effects by anticipated self-stigma for depression

Anticipated self-stigma was not a mediating factor in the relationship between biological attribution for depression and intention to seek help for depression. Specifically, although there was a significant relationship between the biological attribution condition and help seeking (pathway C in Figure 3) there was no significant relationship between the biological causal attribution for depression (e.g., the biological condition) and anticipated self-stigma (SSDS scores) and (pathway A in Figure 1), as noted earlier in the results section. There was a significant negative relationship between anticipated self-stigma (SSDS scores) and help-seeking (GHSQ scores) while adjusting for biological causal attribution for depression (pathway C in Figure 1) ($b = -0.19$, $SE = 0.08$, $p = 0.021$). However, there was no reduction in the relationship between causal attribution for depression and help-seeking when adjusting for self-stigma (adjusted $b = 2.51$, $SE = 0.83$, $p = 0.002$).

Discussion

The question of whether biological attribution increases or decreases stigma carries significant implications for mental health awareness campaigns and public health interventions. In this study, brief online psychoeducational interventions providing information on the biological or psychological causes of depression were found to neither increase nor decrease anticipated self and personal stigma in adolescents. However, the biological intervention significantly increased biological attribution and help-seeking intentions for depression, and the psychosocial intervention significantly increased psychosocial causal attribution. Although the effect size for psychosocial causal attribution on help-seeking intentions was comparable to that for the biological intervention, the effect was not statistically significant, likely due to a lack of statistical power in this condition (i.e., smaller sample size and higher variance).

The finding that stigma levels were unaffected by a biologically-based psychoeducational intervention is inconsistent with proposals by other authors (Dietrich et al., 2004; Read, 2007; Read et al., 2006), who argue that promoting a belief in biological causes will increase stigma by reinforcing the view that people with a mental disorder lack control. Nor are they consistent with the hypothesis that increased biological attribution will decrease stigma (Weiner, 1995; Weiner, Perry, & Magnusson, 1988). The lack of impact of the biological and psychosocial condition on anticipated self-stigma for depression suggests that other factors are important in mediating the reductions in stigma seen with psychoeducational interventions.

The finding that the biological attribution was effective in increasing help-seeking is consistent with the findings of Han et al. (2006) in a university student sample, and the suggestion by Griffiths and Christensen (2004) that biological informational material may be capable of promoting help-seeking. However, the finding that this increase in help-seeking was not mediated by decreased stigma was unexpected. This suggests that other factors are important in mediating the help-seeking seen following psychoeducation about the biological causes of depression. For example, a belief in the biological aetiology of depression may lead to the view that the condition requires a biological intervention (e.g., prescription of antidepressants), or that the condition is more serious than previously perceived and warrants professional intervention (cf. self-care or no treatment). It is also possible that changes in help-seeking were attributable to the message promoting help-seeking rather than causal explanations. However, a recent online study investigating the effect of help-seeking messages alone found no evidence that such information promotes help-seeking for depression in young people (Gulliver et al., 2012).

The finding that a brief online psychoeducation intervention, consisting of one page of online text about the causes of depression, can significantly increase adolescents' intentions to seek help for depression potentially has significant public health implications. Such interventions might, for example, facilitate help-seeking in adolescents with current treatment needs, as well as in those who develop depression in the future. Although the change in help-seeking intention was small,

this modest benefit is offset by the low cost with which such an intervention could be implemented en masse. However, consideration needs to be given to the limitations of this study and how they impact on the potential translation of findings to a real-world setting.

First, the findings pertain to a non-clinical population and may not extend to adolescents who are experiencing clinical levels of depression in the community. Second, we cannot draw any inferences about the potential impact of the intervention on actual help-seeking as we only assessed intention to seek help. Since the association between help-seeking intentions and behaviour is relatively small (Rickwood, Deane, Wilson, & Ciarrochi, 2005), our findings may not translate into increased help-seeking. Third, for pragmatic reasons, the baseline and post-intervention assessments were undertaken immediately before and after the intervention which may have weakened the size of the observed effects. In addition, the sustainability of the intervention effects over time is unknown.

Conclusions

Providing adolescents with online information on the biological causes of depression may be a low-cost strategy to increase their help-seeking for depression. Importantly, we found that reduced self-stigma or personal stigma for depression was not necessary to increase help-seeking intention, and findings were not consistent with the claim that increasing biological causal attribution for depression increases stigma. Further research is needed to determine whether the modest increases in help-seeking intention found in this study translate into increases in actual help-seeking and are sustainable over time. Further research is also needed to clarify the mechanisms underpinning the increased help-seeking intentions seen after adolescents are provided with information on the biological causes of depression.

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