

Regional update

Anxiety symptoms mediate the relationship between exposure to stressful negative life events and depressive symptoms: A conditional process modelling of the protective effects of resilience



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ABSTRACT

Background: Resilience has provided a useful framework that elucidates the effects of protective factors to overcome psychological adversities but studies that address the potential contingencies of resilience to protect against direct and indirect negative effects are lacking. These obvious gaps have also resulted in oversimplification of complex processes that can be clarified by moderated mediation associations. This study examines a conditional process modelling of the protective effects of resilience against indirect effects.

Method: Two separate samples were recruited in a cross-sectional survey from Australia and Norway to complete the Patient Health Questionnaire –9, Generalized Anxiety Disorder, Stressful Negative Life Events Questionnaire and the Resilience Scale for Adults. The final sample sizes were 206 (females = 114; males = 91; other = 1) and 210 (females = 155; males = 55) for Australia and Norway respectively. Moderated mediation analyses were conducted across the samples.

Results: Anxiety symptoms mediated the relationship between exposure to stressful negative life events and depressive symptoms in both samples. Conditional indirect effects of exposure to stressful negative life events on depressive symptoms mediated by anxiety symptoms showed that high subgroup of resilience was associated with less effect of exposure to stressful negative life events through anxiety symptoms on depressive symptoms than the low subgroup of resilience.

Limitations: As a cross-sectional survey, the present study does not answer questions about causal processes despite the use of a conditional process modelling.

Conclusions: These findings support that, resilience protective resources can protect against both direct and indirect – through other channels – psychological adversities.

1. Introduction

Resilience has provided a useful framework that elucidates the effects of protective factors to overcome psychological adversities such as the relationship among exposure to stress, anxiety and depressive symptoms (Anyan and Hjemdal, 2016). However, significant gaps in the literature remain. Studies that address the potential contingencies of the effects of resilience protective resources to protect against direct and indirect negative effects associated with anxiety and depressive symptoms as result of exposure to stressful negative life events are lacking. These obvious gaps have also resulted in oversimplification of complex processes that can be clarified by moderated mediation associations in the resilience literature (see Hayes, 2013). A broad

literature review shows that resilience protective resources either mediate (Anyan and Hjemdal, 2016; Klibert et al., 2014) or moderate (Ai and Hu, 2016; Besser et al., 2015; Chen et al., 2016; Hjemdal et al., 2006; Niu et al., 2016) the relationship between stressful negative life events and adverse outcomes. A search found no studies with a conditional process modelling that combines mediation and moderation using resilience protective resources in the associations among exposure to stressful negative life events and, anxiety and depressive symptoms.

Hankin and Abela (2005) in a review of depression from childhood through adolescence and adulthood provide perspectives that suggest that exposure to stress precede and contribute to depressive symptoms. These perspectives, together with complementary models, have been

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expounded, refined and have become more integrative. The stress-depression relationship has been found not to be unidirectional but transactional and bidirectional (Hankin and Abela, 2005). Prospective studies have shown that the relationship between stressful negative life events and depressive symptoms is not unidirectional but bi-directional over time (Grant et al., 2003; Grant and McMahon, 2005). For example, stressful negative experiences predicted depression; depression in turn predicted increases in stressful negative experiences (Grant et al., 2004b).

Building on previous models, the general conceptual model of the role of stressors in contributing to symptoms of psychopathology such as anxiety and depressive symptoms was proposed and has become broadly applicable across the lifespan (Grant et al., 2003; Grant and McMahon, 2005). According to the general conceptual model, while mutually inclusive and operating at once and in dynamic interaction:

- i. Stressors contribute to symptoms of psychopathology
- ii. Mediators explain the relation between stressors and symptoms of psychopathology
- iii. Moderators influence the relation between stressors and symptoms of psychopathology
- iv. There is specificity in the relations among stressors, moderators, and mediators
- v. Relations among stressors, moderators, mediators, and symptoms of psychopathology are reciprocal and dynamic.

1.1. Stressors contribute to anxiety and depressive symptoms

The extant literature in child, adolescent and adult mental health in view of the general conceptual model hypothesizes that stressors contribute to symptoms – (the present study focuses on anxiety and depressive symptoms) of psychopathology. They include major and minor stressful negative life events as well as chronic conditions (Grant et al., 2003; Grant and McMahon, 2005). A recent cross-sectional study by Schofield et al. (2016) investigated the prevalence of depressive symptoms among Australian university students and the characteristics of those with depressive symptoms. In all, 13% of the students reported severe or extremely severe depressed range. The authors found that psychosocial stress was a key variable associated with higher depressive symptoms (Schofield et al., 2016). In another cross-sectional study by Hjemdal et al. (2012) occurrence of stressful negative life events showed moderate positive associations with depressive, anxiety and somatization symptoms among Norwegian samples.

1.2. Mediators explain the relationship between stressors and depressive symptoms

Mediators and moderators are different because effects of the moderators exist prior to the effects of stress exposure whereas mediators become reactionary to the effects of stress exposure which increases or decreases as result of the effects of stressor. Mediators are mainly biological, psychological and social processes or factors (Grant and McMahon, 2005). A recent study (Kok et al., 2016), that among other objectives, investigated the mediating effect of anxiety symptoms in the relationship between stress exposure and depression, found that anxiety symptoms fully mediated the relationship between exposure to stress and depression in the total sample as well as in subgroups (males and females).

1.3. Moderators influence the relationship between stressors and depressive symptoms

Moderators of the relationship between stressors and depressive symptoms explain an increase or decrease likelihood of depressive symptoms as result of the moderator variable which may be a protective or vulnerability factor (Grant et al., 2003; Grant and

McMahon, 2005). Moderators are mainly personal characteristics or factors that exist in the environment or contexts (Grant and McMahon, 2005). A previous prospective study found that individuals who scored higher on resilience at baseline were not as negatively affected when exposed to stressful negative life events. Whereas individuals who reported lower levels of resilience at baseline developed higher levels of psychiatric symptoms at follow-up when exposed to stressful negative life events even when controlling for gender, age and psychiatric symptoms at pre-test and number of life events (Hjemdal et al., 2006). In this study higher access to sufficient resilience resources highlight the functional utility of resilience as a moderator in overcoming psychological adversities.

2. The current study

Mediation analyses typically answer the question of *how* the correlation between two variables (i.e. an independent variable and a dependent variable) is related to a third variable, the mediator. Moderation analyses seek to determine *when* the association between the independent and the dependent variables in one way or another depend on the moderator variable (Hayes, 2012). Mediation and moderation do not individually say something about whether the mediated effects (i.e. an indirect effect) remains constant, varies systematically across different groups of individuals or contexts. As Hayes (2013) shows, these obvious shortfalls in mediated and moderated models result in oversimplification of complex processes involving indirect mechanisms by ignoring potential contingencies of an effect. Moderated mediation refers to when the strength of an indirect effect is estimated to depend on the level of some variable (Preacher et al., 2007). Moderated mediation combines mediation and moderation in the so called conditional process modelling.

Construction of a conditional process modelling – *moderated mediation model* – will allow to determine the pathways through which the indirect effects of exposure to stressful negative life events mediated by anxiety symptoms on depressive symptoms vary systematically across subgroups of resilience namely high, average and low levels of resilience. In this way, we go beyond explaining mediated pathways through which the relationship between exposure to stressful negative life events and depressive symptoms unfold, to specify subgroups of resilience – *high versus low resilience* – that is likely to overcome psychological adversities that do not directly but indirect affect a person.

2.1. Hypotheses

- i. Exposure to stressful negative life events will contribute to the explained variance in anxiety and depressive symptoms.
- ii. Anxiety symptoms will mediate the relationship between exposure to stressful negative life events and depressive symptoms.
- iii. Resilience protective resources will moderate the relationship between anxiety symptoms and depressive symptoms
- iv. The effect of exposure to stressful negative life events on depressive symptoms through (i.e. mediated by) anxiety symptoms will be less for high subgroup of resilience than the low subgroup of resilience

3. Methods

3.1. Participants and procedure

3.1.1. Australian sample

A total sample of 231 adults was recruited but a final sample size of 206 was used for analyses due to missing responses. The sample comprised undergraduate students of the Research School of Psychology, graduate students of Graduate House and University House of the Australian National University (ANU), waiting passengers at bus stops in Canberra and visitors to a psychology clinic in Sydney.

Eighty-four respondents were aged between 18 and 25 years, 48 respondents were aged between 26 and 30 years and 82 respondents were aged 31 years and above. One hundred and fourteen were females, 91 were males and one reported 'other' as gender. This study was approved by the Human Research Ethics Committee of the ANU. Respondents completed a paper-and-pen questionnaire.

3.1.2. Norwegian sample

A total sample of 352 adults was recruited but a final sample size of 210 was used for the analyses due to missing responses. The sample consisted of undergraduate students in the Norwegian University of Science and Technology. One-hundred and ninety-four respondents were aged between 18 and 25 years, seven respondents were aged 26 to 30 years and nine respondents were aged 31 years or more. One hundred and fifty-five were females while 55 were males. The Regional Committee for Medical Research Ethics (REK) in Norway approved this study. Respondents completed an online survey, which used the existing Norwegian versions of the questionnaires completed by the Australian sample.

3.1.3. Data preparation

SPSS 22 was used for data preparation. In both samples, item-level analyses of more than 5% missing data were removed prior to the analyses. Due to missing responses in the Australian sample, from an original sample size of 231, seven cases were removed from the resilience measure, and ten cases from the stressful negative life events measure and eight respondents who did not report their gender were removed from the analyses. No cases were removed from the other measures. In the Norwegian sample, from 352 sample size, 98 cases were removed from the resilience measure, 41 cases were removed from the measure of depressive symptoms and three cases were removed from the stressful negative life events measure due to missing data. No cases were removed from the other measures.

In both samples, the less strict model-based imputation technique, Expectation Maximization (EM) with 50 iterations, based on maximum likelihood (ML) algorithm (Enders, 2001) was used to replace missing data. This has advantage over single imputation techniques which assume data is missing completely at random and also tends to yield biased estimates under missing at random (Kline, 2005). Expectation maximization uses more information to generate more than one score for each missing observation (Kline, 2005).

3.1.4. Measures

3.1.4.1. Patient health questionnaire (PHQ). The PHQ-9 (Kroenke et al., 2001) is a nine-item self-report measure that assesses the frequency of depressive symptoms over the past two weeks. All items are answered using a 4-point Likert-type scale format ranging from 0 (*not at all*) to 3 (*nearly every day*) with total scores from 0 to 27. Higher scores indicate more depressive symptoms reported by the participants. Example items include "Little interest or pleasure in doing things" "Trouble concentrating on things, such as reading the newspaper or watching television". It has previously been used in a Norwegian sample with a Cronbach's alpha of 0.88 (Solem et al., 2015).

3.1.4.2. Generalized anxiety disorder (GAD – 7). The GAD – 7 (Kroenke et al., 2001; Spitzer et al., 2006) is seven-item self-report measure that assesses anxiety related symptoms in primary care. All items are answered using a 4-point Likert-type scale format ranging from 0 (*not at all*) to 3 (*nearly every day*) with total scores from 0 to 21. Higher scores indicate more anxiety symptoms reported by the participants. Example items include "Feeling nervous, anxious or on the edge" and "Trouble relaxing". The GAD – 7 has previously been used in a Norwegian sample with a Cronbach's alpha of 0.89 (Solem et al., 2015).

3.1.4.3. Stressful negative life events questionnaire (SLE). The SLE (Hjemdal et al., 2006) is self-report questionnaire designed to

measure exposure to stressful life events such as spouse's death, divorce or separation, having been bullied, serious personal accident or injury, exposure to violence or physical attack, having been threatened with a weapon, sexual assault, subjected to other violent or unpleasant sexual activities, and an unhappy childhood. The total number of events is summed up in one sum score for each individual. The SLE has been use in a previous study in Norway (Hjemdal et al., 2006). The Norwegian sample completed the 25-item SLE. The responses were categorized as 0 (*Has not happened*) and 1 (*Negative*). Total score range from 0 to 25. The Australian sample completed a 27-item SLE. The two additional items were "Unable to pay university fees/charges" and "Parents/relatives asking me (to marry) about marriage". Response categories were 0 (*No*) and 1 (*Yes*). Total scores ranged from 0 to 27.

3.1.4.4. Resilience scale for adults (RSA). The RSA is a 33-item self-report scale for measuring resilience to psychosocial adversities among adults (Friborg et al., 2003; Hjemdal et al., 2001). The RSA has been found to have cross-cultural validity and uses a 7-point semantic differential scale format (Capanna et al., 2015; Friborg et al., 2006; Hjemdal et al., 2006). Each item has two opposite attributes at each end of the scale continuum to prevent acquiescence bias (Friborg et al., 2005; Friborg et al., 2006). Higher scores indicate higher levels of protective resources associated with resilience.

3.1.4.5. Socioeconomic status. Socioeconomic status (SES) was assessed by eight items which asked questions about the current employment status of participants, and/or their partners, household income after tax and an item that asked participants to indicate their level of SES from low to high SES. Example employment status items included "Are you employed", "Is your partner employed" and "How would you rate your own socioeconomic status". A composite score was then computed for levels of SES. Total score ranged from 8 to 25 with higher scores indicating better socioeconomic circumstances.

3.1.4.6. Statistical analyses. Statistical analyses were conducted using Stata 14 (StataCorp, 2015). In both samples frequencies and mean scores were analyzed on all measures. Pearson product-momentum correlation was used to explore bivariate associations between the variables in the study. A multi-group path analysis with stressful negative life events as the predictor variable, anxiety symptoms as the mediator variable and depressive symptoms as the criterion variable was conducted to examine possible group differences in the paths that may be statistically significant from each other across the two groups. When at least, one parameter was found to be different and the difference(s) was statistically significant across the two groups, separate moderated mediation models were estimated for the samples.

The mediating effect of anxiety symptoms (Fig. 1) was tested using SEM modelling with maximum likelihood, ML to estimate the indirect, direct and total effects in the multi-group path analysis that tested both the first and second hypotheses. A significant mediating effect was established when the 95% bias-corrected bootstrap confidence interval based on 1000 bootstrap samples did not contain zero. This procedure provides bootstrap confidence interval and standard errors for the mediated effects and has advantages over the traditional approaches in testing mediation (Hayes, 2012, 2015; Preacher et al., 2007) such as the causal steps approach or the test of joint significance approach popularized by (Baron and Kenny, 1986) and the product of coefficients approach (Sobel, 1982, 1986). As multivariate non normality was observed in both samples, applying bootstrapping was apposite remedial strategy for statistical inference about the standard errors and confidence intervals of the conditional indirect effects (see Brown, 2015; Hayes, 2013; Preacher et al., 2007). Additionally, no assumptions about the sampling distribution of the indirect effect are made (Hayes, 2012; Preacher and Hayes, 2008; Preacher et al., 2007).

As the multi-group path analysis showed statistical significance in

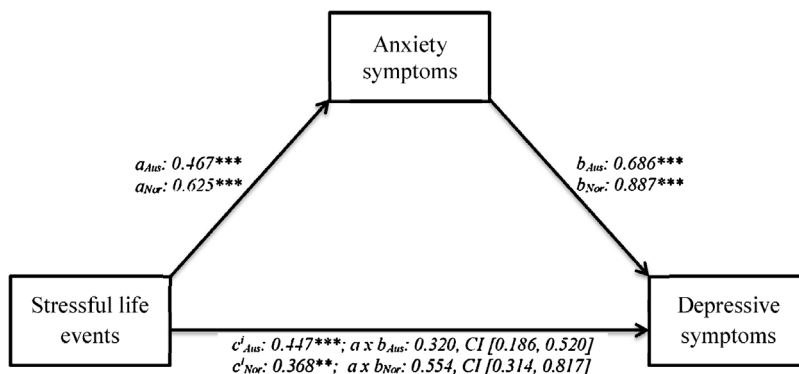


Fig. 1. Statistical model for the mediating effect of anxiety symptoms in the relationship between stressful life events and depressive symptoms among Australian and Norwegian samples.*p < 0.05; **p < 0.01; ***p < 0.001.

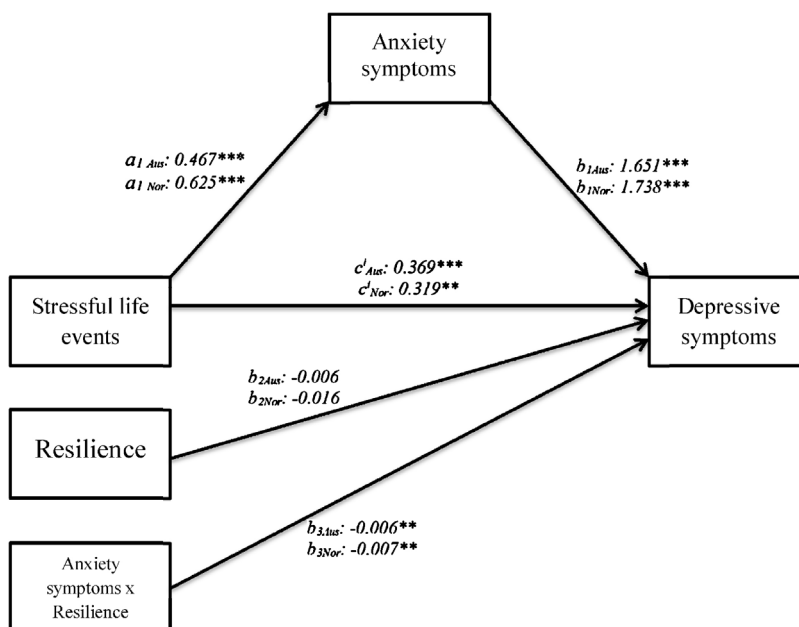


Fig. 2. The conditional process model displayed in statistical form Note. Values are unstandardized path coefficients.*p < 0.05; **p < 0.01; ***p < 0.001.

the difference in at least one path, we conducted two separate moderated mediation analyses using SEM with ML. To test the third hypothesis, resilience was added to the model as a moderator variable of the mediated effect by anxiety symptoms to examine contingencies of the conditional indirect effects of exposure to stressful negative life events on depressive symptoms at high, average and low levels of resilience (see Fig. 2). The following categories were constructed for high (1 SD above the mean), average, and low (1 SD below the mean) levels of resilience. Also using 95% bias-corrected bootstrap confidence interval based on 1000 bootstrap samples, moderation of the indirect effects (i.e. conditional indirect effects) at levels of resilience were examined. Evidence of moderated mediation was shown when a 95% bias-corrected bootstrap confidence interval did not contain zero for the conditional indirect effects (Hayes, 2013, 2015).

Even though some researchers suggest that structural equation modelling (SEM) is robust against multicollinearity other researchers offer methods that can be used to detect multicollinearity (see Belsley, 1991; Belsley et al., 1980; Kaplan, 1994). The methods include condition index and variance decomposition proportion, inspection of correlation matrix and variance inflation factors. All correlations in this study were below the cut-off ($r > 0.80$) by (Field, 2013) in both samples. The variance inflation factor ranged between 1.03 to 1.26 and 1.03 to 1.57 for the Australian and Norwegian samples respectively. The tolerance statistics ranged between 0.79 to 0.97 and 0.64 to 0.97

for the Australian and Norwegian samples respectively. Belsley (1991), and Belsley et al. (1980) suggest a condition index greater than 30 as indicating strong dependency. In the present study the condition indices were below 30. The condition indices were 17.03 and 19.95 for the Australian and Norwegian samples respectively. These test statistics show that multicollinearity was not a problem in either of the samples. Because of the overlap between predictors of depressive symptoms and predictors of protective factors in the literature it was necessary to adjust for control variables namely age, gender and SES to offer adequate insight in demonstrating unique relationships.

4. Results

4.1. Preliminary correlation analyses

Table 1 displays the means, standard deviations and Cronbach's alphas of the measures used and their inter-correlations. In both samples, age significantly positively correlated with socioeconomic status, and significantly positively with resilience but significantly negatively with anxiety and depressive symptoms in the Australian sample. Gender significantly negatively correlated with exposure to stressful negative life events and anxiety symptoms in the Norwegian sample. Socioeconomic status significantly negatively correlated with exposure to stressful negative life events in the Australian sample. In

Table 1
Table of Means, Standard deviations, Cronbach's alpha estimates(α) and Correlations for All the Measures.

Variable	Australia (N = 206)		Norway (N = 210)									
	M(SD)	α	M(SD)	α	1	2	3	4	5	6	7	
1 Age					–	–0.013	0.150*	–0.051	0.045	–0.003	0.012	
2 Gender					–0.001	–	–0.071	0.007	–0.171*	–0.191**	–0.082	
3 Socioeco-nomic status	16.04(2.68)		13.37(1.95)		0.301***	0.082	–	0.059	0.143*	0.033	0.005	
4 Resilience	5.30(0.73)	0.89	5.13(0.72)	0.9	0.168*	–0.074	0.034	–	–0.306***	–0.529***	–0.611***	
5 Stressful life events	2.80(2.71)	0.7	3.07(2.51)	0.64	–0.127	–0.064	–0.205**	–0.321***	–	0.398***	0.430***	
6 Anxiety symptoms	5.11(4.14)	0.87	6.18(4.12)	0.86	–0.196**	–0.06	0.022	–0.364***	0.304***	–	0.741***	
7 Depressive symptoms	5.13(4.77)	0.87	6.70(5.30)	0.91	–0.320***	–0.072	–0.052	–0.484***	0.451***	0.713***	–	

Note: Intercorrelations among variables are shown below the diagonal for Australian samples and above the diagonal for Norwegian samples.

- * $p < 0.05$.
- ** $p < 0.01$.
- *** $p < 0.001$

both samples, resilience significantly negatively correlated with exposure to stressful negative life events, anxiety and depressive symptoms. Also in both samples, exposure to stressful negative life events significantly positively correlated with anxiety and depressive symptoms.

The differential significant correlations of age, gender and socioeconomic status particularly with resilience, anxiety and depressive symptoms across the samples support adjusting the effects of these variables in subsequent analyses.

Hypothesis 1. Exposure to stressful negative life events contribute to the explained variance in anxiety and depressive symptoms

In support of the first hypothesis, exposure to stressful negative life events was significantly positively associated with anxiety symptoms (Australia: *unstandardized* B = 0.467, 95% CI = 0.268, 0.665, $p < 0.001$; Norway: *unstandardized* B = 0.625, 95% CI = 0.419, 0.832, $p < 0.001$). Exposure to stressful negative life events was also significantly positively associated with depressive symptoms (Australia: *unstandardized* B = 0.686, 95% CI = 0.579, 0.793, $p < 0.001$; Norway: *unstandardized* B = 0.887, 95% CI = 0.762, 1.011, $p < 0.001$).

Hypothesis 2. Anxiety symptoms mediate the relationship between exposure to stressful negative life events and depressive symptoms

Table 2 displays the estimates of 95% bias-corrected bootstrap CI and results summary for the relationship between exposure to stressful negative life events and depressive symptoms mediated by anxiety symptoms displayed in the left column for Australian sample and in the right column for Norwegian sample. In both samples, anxiety symptoms partially mediated the relationship between exposure to stressful negative life events and depressive symptoms in partial support of our second hypothesis. This is showed by (i) the direct effects were smaller than the total effects of stressful negative life events on depressive symptoms, and (ii) there was no zero in the 95% bias-

Table 2
Mediating effect of Anxiety symptoms in the relationship between Stressful life events and Depressive symptoms in Australian and Norwegian samples.

Effect	Australia (N = 206)			Norway (N = 210)		
	B(SE)	p-value	Bias-corrected bootstrap 95% CI	B(SE)	p-value	Bias-corrected bootstrap 95% CI
a	0.467(0.101)	< 0.001		0.625(0.105)	< 0.001	
b	0.686(0.055)	< 0.001		0.887(0.064)	< 0.001	
c	0.767(0.106)	< 0.001		0.922(0.144)	< 0.001	
c ^d	0.447(0.084)	< 0.001		0.368(0.107)	0.001	
a x b	0.320(0.074)	< 0.001	[0.186, 0.520]	0.554(0.126)	< 0.001	[0.314, 0.817]

Note: CI = confidence interval.

a = effects of stressful life events on anxiety symptoms. b = effects of anxiety symptoms on depressive symptoms after adjusting for stressful life events. c = total effects of stressful life events on depressive symptoms. c^d = direct effects of stressful life events on depressive symptoms. a x b = mediating effects of anxiety symptoms in the relationship between stressful life events and depressive symptoms (i.e. the indirect effect of stressful life events mediated by anxiety symptoms on depressive symptoms).

corrected bootstrap confidence interval for the indirect effects. In addition, a statistically significant difference was found for the paths that predicted symptoms of depression from symptoms of anxiety across the two samples $\chi^2(1) = 5.703, p = 0.016$ in the multi-group path analysis analyses.

4.2. Moderated mediation

As the multi-group path analysis showed a statistically significant difference in at least one path across the two samples, we conducted two separate bias-corrected bootstrapped moderated mediation analyses using SEM with ML.

Hypothesis 3. Resilience protective resources moderate the relationship between anxiety symptoms and depressive symptoms.

Table 3 displays the model coefficients for the moderated mediation analyses in both samples. The interaction between anxiety symptoms and resilience significantly negatively predicted depressive symptoms in both samples. In support of the third hypothesis, higher scores on resilience was associated with lower direct negative effects of anxiety symptoms on depressive symptoms whereas lower scores on resilience was associated with higher direct negative effects of anxiety symptoms on depressive symptoms.

Hypothesis 4. Effect of exposure to stressful negative life events on depressive symptoms mediated by anxiety symptoms is less for high subgroup of resilience compared to low subgroup of resilience.

Table 4 displays the results for the conditional indirect effect of exposure to stressful negative life events on depressive symptoms mediated by anxiety symptoms for subgroups of high, average and low resilience. As a statistically significant interaction (between the mediator – anxiety symptoms, and the moderator – resilience) does not imply evidence of moderated mediation (i.e. evidence of conditional indirect effects). Further analyses were conducted to derive and

Table 3
Model Coefficients for the Conditional Process Modelling (i.e. Moderated mediation).

Predictor variable	Outcome variable					
		Anxiety symptoms		Depressive symptoms		
		B(SE)	p-value	B(SE)	p-value	
Australia (N = 206)						
Stressful life events	a ₁	0.467(0.101)	< 0.001	0.369(0.081)	< 0.001	
Anxiety symptoms	–	–	–	b ₁	1.651(0.310)	< 0.001
Resilience	–	–	–	b ₂	–0.006(0.013)	0.637
Anxiety symptoms × Resilience	–	–	–	b ₃	–0.006(0.002)	0.001
Age	a ₂	–0.934(0.315)	0.003	b ₄	–0.918(0.237)	< 0.001
Gender	a ₃	–0.431(0.530)	0.416	b ₅	–0.590(0.396)	0.136
Socioeconomic status	a ₄	0.232(0.107)	0.03	b ₆	0.059(0.079)	0.456
Norway (N = 210)						
Stressful life events	a ₁	0.625(0.105)	< 0.001	c ⁱ	0.319(0.097)	0.001
Anxiety symptoms	–	–	–	b ₁	1.738(0.313)	< 0.001
Resilience	–	–	–	b ₂	–0.016(0.017)	0.348
Anxiety symptoms × Resilience	–	–	–	b ₃	–0.007(0.002)	0.001
Age	a ₂	–0.166(0.596)	0.781	b ₄	–0.088(0.502)	0.861
Gender	a ₃	–1.208(0.598)	0.043	b ₅	0.573(0.509)	0.261
Socioeconomic status	a ₄	–0.060(0.135)	0.659	b ₆	0.006(0.115)	0.956

Note: B = Unstandardized path coefficients; SE = Standard Error.
a = effects of stressful life events on anxiety symptoms.
ci = effects of stressful life events on depressive symptoms.
b1 = effects of anxiety symptoms on depressive symptoms.
b2 = effects of resilience on depressive symptoms.
b3 = the interaction of anxiety symptoms and resilience on depressive symptoms.

quantify the conditional indirect effects of exposure to stressful negative life events on depressive symptoms through anxiety symptoms for subgroups of resilience in both samples as shown in Table 4.

With a negative index of moderated mediation, the results further show that high resilience was associated with less effects of exposure to stressful negative life events through anxiety symptoms on depressive symptoms whereas low resilience was associated with more effects of exposure to stressful negative life events through anxiety symptoms on depressive symptoms. This was statistically significant in both samples as shown in the 95% bias-corrected bootstrap confidence interval.

5. Discussions

This study tested major hypotheses from the overarching general conceptual model of the role of stressors in symptoms of psychopathology (Grant et al., 2003; Grant and McMahon, 2005) across two samples, from Australia and Norway. In addition, the functional utility of resilience resources (among subgroups) in protecting against the indirect negative effect of exposure to stressful negative life events through anxiety symptoms on depressive symptoms was tested. Firstly, exposure to stressful negative life events contributed to the explained variance in anxiety and depressive symptoms in both samples. Secondly, anxiety symptoms partially mediated the relationship between

exposure to stressful negative life events and depressive symptoms in both samples. Thirdly, resilience moderated the relationship between anxiety symptoms and depressive symptoms in both samples. And last but not least, the results showed that the relation between exposure to stressful negative life events and depressive symptoms mediated by anxiety symptoms was less for high subgroup of resilience compared to low subgroup of resilience in both samples. The protective factors measured by resilience were based on the overarching categories of positive personal dispositions, family cohesion and external social support outside the family (Werner and Smith, 1992).

5.1. Exposure to stressful negative life events contribute to the explained variance in anxiety and depressive symptoms across Australian and Norwegian samples

Consistent with our predictions exposure to stressful negative life events contributed to the explained variance in anxiety and depressive symptoms which supports previous studies (Grant et al., 2003; Grant and McMahon, 2005; Hankin and Abela, 2005) and in a related sample in Australia (Schofield et al., 2016) and in Norway (Hjemdal et al., 2012). Recently, researchers have suggested that this relationship reflect a situation of prolonged stressful experiences especially among university students (Schofield et al., 2016). This finding supports the

Table 4
Derivation and Quantification of, and Inference about the Conditional Indirect Effects of Stressful Life Events on Depressive Symptoms at High, Average and Low Resilience.

a ₁	a ₁ b ₃	Resilience	a ₁ θ _(resilience → depressivesymptoms) = a ₁ (b ₁ + b ₃ *Resilience)	SE	Bias-corrected bootstrap 95% CI
Australia (N = 206)					
0.467	–0.003	High	0.195	0.081	[0.081, 0.408]
0.467	–0.003	Average	0.265	0.073	[0.159, 0.442]
0.467	–0.003	Low	0.334	0.079	[0.210, 0.516]
Norway (N = 210)					
0.625	–0.004	High	0.281	0.125	[0.102, 0.583]
0.625	–0.004	Average	0.381	0.119	[0.199, 0.654]
0.625	–0.004	Low	0.480	0.124	[0.274, 0.769]

Note: * = multiply; SE = Standard Error for the conditional indirect effects; CI = Confidence interval for the conditional indirect effects; a₁ = effect of stressful life events on anxiety symptoms (unconditional); a₁b₃ = Index of moderated mediation; aθ_(resilience → depressivesymptoms) = conditional indirect effects at levels of resilience.

general conceptual model for the role of exposure to negative life events in contributing to symptoms of psychopathology (Grant et al., 2003; Grant and McMahon, 2005).

Exposure to stressful negative life events has been implicated in prospective studies in contributing to the development of hopelessness and loss, which may account for the predictability in depressive symptoms while appraising stressful negative life events as uncontrollable may result in apprehensive expectations about several events that may account for the predictability in anxiety symptoms. Hankin and Abela (2005) on the stress-depression relationship affirmed that exposure to stressful negative life events are associated with generation of stressful circumstances and additional negative events. This can advance further increases in anxiety symptoms or depressive symptoms that will feed back into elevated exposure to stressful negative life events that shows a transactional and bidirectional relationship.

5.2. Anxiety symptoms mediate the relationship between exposure to stressful negative life events and depressive symptoms across Australian and Norwegian samples

The present findings, like many others, implicate exposure to stressful negative life events as an important contributor to depressive symptoms. It is also clear that, in the mediated model anxiety symptoms emerged as an underlying contributory factor to the hypothesized positive relationship between the effects of exposure to stressful negative life events and depressive symptoms. This shows that exposure to stressful negative life events both directly and indirectly (through anxiety symptoms) contributes to depressive symptoms. As with previous studies (Kok et al., 2016), anxiety symptoms mediated the relationship between stress exposure and depressive symptoms.

In both samples, it can be argued that the negative effects of the association between exposure to stressful negative life events and depressive symptoms is in part, accounted for by scores on anxiety symptoms. This lends support to cognitive and emotion theories which suggests a relationship between anxiety and depression in the development of symptoms of psychopathology. Higa-McMillan et al. (2014) suggested that anxiety plays a central role in negative emotions, that may in turn, as a risk factor contribute to depression. It may also be true to argue that the pathways that explain the association between exposure to stressful negative life events and depressive symptoms may be associated with the consequences of anxiety-related cognitions due to scores on anxiety symptoms, which in turn may be positively associated with negative beliefs that characterizes depressive symptoms.

5.3. The functional utility of resilience against direct effects of anxiety symptoms and indirect effects of exposure to stressful negative life events on depressive symptoms across Australian and Norwegian samples

The findings in this study demonstrate a novel functional utility of resilience protective resources. Derivation and quantification of the conditional indirect effects of exposure to stressful negative life events on depressive symptoms through anxiety symptoms showed that high subgroup of resilience was associated with less effect of exposure to stressful negative life events through anxiety symptoms on depressive symptoms than the low subgroup of resilience. Additionally, higher scores on resilience were associated with lower direct negative effects of anxiety symptoms whereas lower scores on resilience protective resources were associated with higher direct negative effects of anxiety symptoms on depressive symptoms. As with a previous study (Hjemdal et al., 2006), resilience buffered the effects of stressful negative life events associated with anxiety and depression, and at follow-up, individuals who reported higher access to resilience resources at baseline were not negatively affected when exposed to stressful negative life events. This is consistent with the findings in the present study. However, the study by Hjemdal et al. (2006) did not examine the

utility of resilience in subgroups. Additionally, the study by Hjemdal et al. (2006) and several other studies (Ai and Hu, 2016; Besser et al., 2015; Chen et al., 2016; Niu et al., 2016) do not investigate the utility of resilience protective resources in protecting against indirect effects.

Taken together, more access to and availability of, as well as the magnitude of resilience protective resources support resilience as having a buffering effect that can protect people who score higher on resilience and those who have available high resilience against psychological adversities that may be direct or indirect. These are supporting findings for the functional utility of resilience protective resources in relation to psychological adversities. It may not be farfetched to hypothesize that more access to resilience or people who have available high resilience resources can protect against direct negative life circumstances as well as those that may confront them indirectly through other channels of adversities. To the best of our knowledge, the present study is the first study to investigate the utility of resilience in a conditional process model of the indirect effect of exposure to stressful negative life events through anxiety symptoms on depressive symptoms among subgroups of resilience. Such conditional process models have the ability to clarify complex processes involving indirect pathways that do not ignore the potential contingencies of the effect of the putative relationship between (two or more) variables. In the present study we found that the effect of exposure to stressful negative life events through anxiety symptoms on depressive symptoms *actually depended on* – as shown by the bias-corrected C.I of $a_1(b_1 + b_3 * Resilience)$ – whether or not the person belonged to a high or low resilience subgroup.

6. Conclusions

This study has clarified salient indirect and conditional pathways across countries that improve international perspectives on the relationship between exposure to stressful negative life events and anxiety and depressive symptoms as well as the literature on protective factors. We tested whether the mediated effect by anxiety symptoms in the relationship between exposure to stressful negative life events and depressive symptoms can be extenuated depending on a person's available and strength of resilience protective resources. In addition to explaining the pathways through which anxiety symptoms contribute to the relationship between exposure to stressful negative life events and depressive symptoms unfolds. We have also clarified subgroups of resilience protective resources that may be protected against the indirect effect of exposure to stressful negative life events through anxiety symptoms on depressive symptoms.

Support was found for the hypotheses that were tested based on the general conceptual model of the role of stressors in contributing to symptoms of depression. Exposure to stressful negative life events contributed to the explained variance in anxiety and depressive symptoms. High scores on exposure to stressful negative life events were associated with high anxiety symptoms, which in turn, were positively associated with depressive symptoms. Resilience provided buffering effects that protected against depressive symptoms.

6.1. Limitations

The measures of stressful negative life events were not identical in their number of items across the two samples. The two additional items for the Australian sample were intended to capture additional financial and sociocultural stress. Differences in the stressful negative life events questionnaire may affect general assessment of exposure to stressful negative life events in the samples. However, it is also important that a measure of exposure to stressful negative life events be contextualized in a way that it can capture relevant stressors in the context that it is utilized.

The different modes of data collection – online versus paper-and-pen survey – may affect the results in terms of response rate and data

reliability across the two samples. Therefore, limiting extrapolation of study results. Hohwü et al. (2013) and Zuidgeest et al. (2011) contended that paper-and-pen is preferred and results in higher response rate and data reliability than online surveys, as it is more convenient to answer paper-and-pen and in several rounds. This may explain removing about 40% of the respondents from the Norwegian sample who completed online survey as compared to removing about 11% of the respondents from Australian sample who completed paper-and-pen survey due to more than 5% missing data.

The use of cross-sectional survey fails to show causal process and temporal changes over time. While we have provided strong evidence of pathways by use of the bootstrap (bias-corrected) method in a conditional process modelling to explain the associations among exposure to stressful negative life events, anxiety and depressive symptoms as well as the association with protective factors. We acknowledge that the use of a cross-sectional survey is limited in answering questions about protective processes that lead to successful adaption over time from a lifespan developmental perspective.

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