Longitudinal Associations between Family Identification, Loneliness, Depression, and Sleep Quality

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Abstract

Objectives: The prevalence of depression and loneliness are increasing in Western nations, and both have been shown to cause poor sleep quality, with evidence suggesting that loneliness also predicts depression. The Social Cure perspective can shed light on these relationships, and thus informs the present study. Specifically, it was hypothesised that the extent of participants' identification with a significant social group, their family, would positively predict sleep quality, and that this relationship would be mediated by loneliness and depression.

Design: A two-wave longitudinal online survey was used.

Methods: Participants completed an online survey at T1 ($N = 387$) and one year later at T2 ($N = 122$) assessing the extent to which they identified with their family. Their loneliness, depressive symptomology, and sleep quality/insomnia severity were also measured.

Results: Consistent with predictions, cross-sectional and longitudinal serial mediation models indicated that family identification was a negative predictor of loneliness, which in turn was a positive predictor of depression, which predicted poor sleep quality/insomnia.

Conclusions: This is the first Social Cure study to explore the mediated relationship between social identification and sleep quality. As well as advancing the Social Cure perspective, these results have implications for how health professionals understand, prevent, and treat sleep problems.

Introduction

The World Health Organisation notes that depression is the world's biggest cause of disability, affecting over 300 million people (WHO, 2017). Depression's prevalence has also increased dramatically, leading some to suggest we are experiencing an epidemic (e.g., Whitaker, 2005). While depression causes numerous negative health outcomes, sleep disturbance is especially common, with around 90% of patients experiencing poor sleep quality and insomnia (Tsuno, Besset, & Ritchie, 2005). In turn, sleep problems have the potential to exacerbate mental health conditions, creating a vicious cycle (Kaneita et al., 2007).

As well as depression, another increasingly common cause of sleep problems is loneliness (Mushtag, Shoib Shah, & Mushtag, 2014). As with depression, there have been suggestions that many industrialised societies are facing a loneliness epidemic, with up to one-third of people affected (Cacioppo & Cacioppo, 2018a; King, 2018). The loneliness-depression link is well-established (Hakulinen et al., 2018; Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015), with research showing that loneliness predicts depression over time, regardless of initial depression levels (Cacioppo, Hawkley, & Thisted, 2010).

As mentioned, researchers have also explored the relationship between loneliness and sleep. Recruiting students, Cacioppo et al. (2002) found that lonely participants had poorer sleep quality and more night-time waking than non-lonely participants: a finding strengthened by more recent longitudinal research exploring populations such as older adults (e.g., McHugh & Lawlor, 2013), and meta-analytic work indicating a robust positive relationship between social support and sleep quality (de Grey, Uchino, Trettevik, Cronan, & Hogan, 2018). In their Evolutionary Theory of Loneliness (ETL) Cacioppo and Caciopppo (2018b) propose that loneliness leads to poor sleep quality and insomnia (as well as other outcomes) because it signals that one exists in an environment where others are likely to behave selfishly. This triggers a physiological response to the perceived possibility of social threat (even when such threat does not exist), including increased production of cortisol, ordinarily secreted at waking, which negatively affects sleep onset and is unconducive to high-quality sleep. This was supported by Matthews et al. (2017), who found that the association between

loneliness and poor sleep quality in young adults was especially pronounced for those who had experienced trauma (e.g., violence/neglect). The authors concluded that such individuals would be even more vigilant regarding social threats than lonely people without such experiences, leading to even poorer quality sleep. While loneliness has thus been shown to be an important predictor of both depression and poor sleep quality, the variables that might predict loneliness itself have not been fully explored.

Social Groups, Depression, and Sleep Quality

One possible predictor of loneliness (and thus ultimately a predictor of depression and sleep quality) is the strength of one's social networks: how connected one is to others. Indeed, much work within sociology and epidemiology has shown how stronger social networks predict better mental health (Berkman, Glass, Brissette, & Seeman, 2000). Sleep has also been investigated: although they did not explore loneliness, Cheng, Malhotra, Chan, Østbye, and Lo (2018) investigated the longitudinal relationship between strength of social networks and poor sleep quality. Recruiting a sample of elderly Taiwanese people, they showed that weak social networks predicted restless sleep, an depressed mood mediated this relationship.

Cheng and colleagues' work is promising; however, it is important to note that the authors defined 'social networks' as the number of friends participants interact with at least once a month, and how often they interact with relatives. This definition is consistent with traditional sociological measures of *social integration*, which focus on the quantitative aspects of one's social landscape (e.g., Berkman et al., 2000; Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997). In recent years, researchers aligned with the Social Identity Approach within social psychology (e.g. Sani, Madhok, Norbury, Dugard, & Wakefield, 2015) have highlighted the limitations of this focus: most notably, that it neglects the subjective and psychological nature of social group memberships, and thus fails to consider how it *feels* to belong to a group.

The Social Cure Perspective

Through placing emphasis on the subjective and contextual aspects of people's social landscapes, the Social Identity Approach gave rise to the Social Cure perspective (e.g., Jetten, Haslam, & Haslam, 2012). This perspective posits that social group memberships have a key role to play in people's well-being, but only if they subjectively *identify* with the group/s in question (i.e., experience a sense of belonging to the group, Sani, et al., 2015). Being a member of such a group has been shown to confer many benefits (see Wakefield, Bowe, Kellezi, McNamara, & Stevenson, 2019, for a review). In turn, these benefits have been shown to enhance health/well-being across numerous populations and contexts, from people with multiple sclerosis (Wakefield, Bickley, & Sani, 2013) and detainees (Kellezi, Bowe, Wakefield, McNamara, & Bosworth, 2019) to people recovering from stroke (Haslam et al., 2008) and new mothers (Seymour-Smith, Cruwys, Haslam, & Brodribb, 2017).

Importantly for the present study, there is evidence from the Social Cure literature to support the idea that group identification predicts reductions in feelings of loneliness. For instance, in their investigation of the efficacy of the Social Cure-inspired Groups4Health intervention, Haslam, Cruwys, Haslam, Dingle, and Chang (2016) showed that reductions in loneliness were driven by participants' increased identification with their Groups4Health support group. Additionally, Jose and Lim (2014) showed a negative relationship between group identification and loneliness over time in an adolescent sample.

Moreover, there is also evidence to support the idea that group identification predicts reductions in depressive symptomology: for instance, as well as Haslam et al. (2016) and Jose and Lim (2014) finding reductions in depression being driven by group identification in their studies mentioned above, Cruwys et al. (2014) showed that interventions designed to increase group identification are successful at reducing depression in clinical and community settings.

One group that has been shown to be particularly important for mental health/well-being is the family. For most individuals, the family is the 'first group': the one with which they are likely to experience most contact (Elliott & Umberson, 2004). Numerous studies have highlighted the important relationship between family identification and health/well-being: Herrera, Sani and Bowe (2011), Sani, Herrera, Wakefield, Boroch, and Gulyas (2012), and Wakefield et al. (2017) reported a

positive relationship between family identification and mental well-being cross-sectionally, and this relationship persists longitudinally (Wakefield, Sani, Herrera, Khan, & Dugard, 2016). Additionally, Naughton, O'Donnell, and Muldoon (2015) showed the protective effects that family identification can have on the mental health of young people who witness intimate partner violence. Moreover, Sani, Wakefield, Herrera, and Zeybek (2017) showed a negative association between family identification and paranoid ideation in non-clinical individuals. This is particularly relevant for the present study, as there are well-established links between paranoia and loneliness (Riggio & Kwong, 2011).

The Present Study

Combining these strands of literature, a longitudinal survey study was conducted to investigate the relationship between the extent of participants' family identification and their sleep quality and insomnia severity. Rather than just measuring insomnia severity, sleep quality was also assessed in order to ensure rigour within the research: while sleep quality scales often measure insomnia severity, they provide a more holistic assessment of participants' sleep experiences (e.g., Pilcher, Ginter, & Sadowsky, 1997). This relationship was tested both cross-sectionally and longitudinally, with the prediction that this association would be serially mediated by loneliness and depression. No research has yet explored the relationship between group identification and sleep quality/insomnia severity, so the present study represents an important contribution to the literature. Combining evidence from the Social Cure literature and the loneliness literature leads to the following predictions:

1. There will be a negative relationship between participants' family identification and poor sleep quality/insomnia severity, both a) cross-sectionally, and b) longitudinally.

2. There will be a negative relationship between participants' family identification and loneliness, both a) cross-sectionally, and b) longitudinally.

3. There will be a negative relationship between participants' family identification and depression, both a) cross-sectionally, and b) longitudinally.

4. There will be a positive relationship between participants' loneliness and poor sleep quality/insomnia severity, both a) cross-sectionally, and b) longitudinally.

5. There will be a positive relationship between participants' depression and poor sleep quality/insomnia severity, both a) cross-sectionally, and b) longitudinally.

6. Loneliness and depression will mediate the relationship between family identification and poor sleep quality/insomnia severity, so that stronger family identification will predict lower loneliness, which in turn will predict lower depression, which in turn will predict better sleep quality/less severe insomnia, both a) cross-sectionally, and b) longitudinally.

Method

Participants and Procedure

Five-hundred and twenty-seven participants completed the first wave of an online survey. Informed consent was obtained, and participants were debriefed at the end of the survey. One-hundred and forty of these participants were removed from the data-file because they completed too little of the survey to produce analysable results (i.e., the vast majority-or even all-of their responses were blank). This led to a time 1 (T1) sample of three-hundred and eighty-seven (246 females, 62 males, 79 unknown; M_{age} = 33.67 years, SD = 12.60, $range$ = 18-76). Participants were recruited globally via social media platforms (predominantly Facebook groups, after gaining permission from the group moderators), and locally via posters with survey links. We wished to recruit participants as widely as possible, so as to reduce the likelihood of any findings being bound by culture. Of the 299 participants who stated their nationality, 229 (76.59%) were British/Irish, and 33 (11.04%) were American. The other 12.37% was comprised of 23 nationalities, including Singaporean (2.01%) and German (1.34%).

One year later (M = 352.79 days, SD = 9.93, $range$ = 324.16-374.43 days), participants were contacted via email and asked to complete the study's second wave. A time-period of one year was selected in order to avoid seasonal changes in light levels (which can affect sleep: Anderson et al., 1994) affecting the T2 results. One hundred and forty-nine participants responded, but data for 27 participants were removed from the data-file because they completed too little of the survey to

produce analysable results. This led to a time 2 (T2) sample of one hundred and twenty-two (99 females, 23 males; M_{age} = 34.82 years, SD = 12.39, $range$ = 18-73). Of the 120 participants who stated their nationality, 103 (85.83%) were British/Irish, and 6 (5%) were American. The other 9.17% was comprised of 10 nationalities, including German (0.83%).

An analysis of variance was conducted to compare the T1 participants who did vs. did not complete the T2 survey. These groups did not differ significantly in terms of age, highest level of education, family identification, loneliness, depression, sleep quality, or insomnia severity (ps ranged from .07 to .92). A chi-square analysis was also conducted, which revealed that the groups did not differ significantly in terms of the number of males vs. females, the number of participants in a relationship vs. not in a relationship, and the number of participants looking after a young child vs. not looking after a young child (ps ranged from .37 to.43). Based on these analyses, it was concluded that the participants who completed the T2 survey were a good representation of the sample as a whole.

Measures

Participants' *family identification* was measured with the four-item Group Identification Scale (GIS; Sani et al., 2015, e.g., "I feel a sense of belonging to my family"). Respondents specified their agreement with each item using a scale ranging from 1 ("I strongly disagree") to 7 ("I strongly agree"). The items were averaged to create an overall measure of family identification (T1: α = .90, T2: α = .89), where higher values indicate higher identification. Participants were asked to define 'family' in any way that was meaningful for them. The group-level intraclass correlation coefficient (ICC) across the two time-points was .90, which is above the 'excellent' reliability cut-off of .75 (Fleiss, 1986).

Participants' *loneliness* was measured with Gierveld and Tilburg's (2006) six-item Short Loneliness Scale. Participants rated their agreement with each item (e.g., "I miss having people around") on a scale ranging from 1 ("I strongly disagree") to 7 ("I strongly agree"). The items were averaged to create an overall measure of loneliness (T1: α = .84, T2: α = .88), where higher values indicate higher loneliness. The ICC was .89.

Participants' *sleep quality* was measured with the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Participants completed 19 items on a range of scales (e.g., "During the past month"… "When have you usually gone to bed?"), which were used to compute an overall score of sleep quality ranging from 0 to 21 (T1: α = .74; T2: α = .75), with higher scores indicating poorer sleep quality (see Buysse et al., 1989 for computation details). The ICC was .82.

Participants' *insomnia severity* was measured with the Insomnia Severity Index (Bastien, Vallières, & Morin, 2001). Participants completed 7 items on a range of 0-4 scales (e.g., "How satisfied/dissatisfied are you with your current sleep pattern?"). The sum of the items was found in order to create an overall measure of insomnia severity ranging from 0 to 28 (T1: α = .89; T2: α = .90), with higher values indicating greater severity. The ICC was .80.

Participants' *depression* was measured with the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). Participants rated the frequency with which they had experienced seven depressive symptoms (e.g., "I felt as if I am slowed down") during the past week on a 0-3 scale (e.g., "not at all" to "most of the time"). Participants' responses were summed, creating a value between 0 and 21 (T1: α = .85; T2: α = .84), where higher values indicate more depressive symptoms. The ICC was .85.[1]

A number of relevant *control variables* were also measured: participants' *age*, *gender* (0 = female, 1 = male), *relationship status* (0 = no, 1 = yes), *highest level of education* on a scale ranging from 1 (GCSEs) to 5 (doctorate), and whether they are currently *caring for a young child* (0 = no, 1 = yes). These variables were selected for inclusion due to the potential impact they might have on sleep quality/insomnia.

Results

Table 1 shows the variable inter-correlations. Supporting hypotheses 1a, 2a, and 3a, family identification T1 correlated negatively with poor sleep quality, insomnia, loneliness, and depression T1 (*p*s < .001), while family identification T2 correlated negatively with poor sleep quality, insomnia, loneliness, and depression T2 (*p*s < .01). Moreover, supporting hypotheses 1b, 2b, and 3b, family identification T1 correlated negatively with poor sleep quality, insomnia, loneliness, and depression

T2 ($ps < .001$). Meanwhile, supporting hypotheses 4a, 4b, 5a, and 5b, loneliness and depression both correlated positively with poor sleep quality and insomnia at T1, T2, and across T1 and T2 ($ps < .001$). Depression and loneliness also correlated positively at T1, T2, and across T1 and T2 ($ps < .001$).

(TABLE 1)

Cross-Sectional Mediation Analyses

Model six in version 3.0 of Hayes' (2017) PROCESS macro was used to test Hypothesis 6a: that loneliness and depression will mediate the relationship between family identification and sleep quality/insomnia at T1, so that stronger family identification will predict lower loneliness, which in turn will predict lower depression, which in turn will predict both better sleep quality and insomnia severity. The analyses involved 5,000 bootstrapping samples with 95% confidence intervals (LLCI/ULCI), using the percentile method. Values were mean centred for the construction of products. Participants' gender, age, relationship status, highest level of education, and whether or not they were looking after a young child were controlled for. For completeness, both analyses were re-run with the positions of the mediators reversed (i.e., depression placed before loneliness). The resultant models were both non-significant.

T1 Analysis: Predicting Sleep Quality

Supporting Hypothesis 6a, a significant effect of family identification T1 on poor sleep quality T1 though loneliness T1 and depression T1 was found, *Effect* = -0.50, *Boot SE* = .10, *Boot LLCI* = -.69, *Boot ULCI* = -.34. Family identification was a negative predictor of loneliness, *Coeff* = -.51, *SE* = .05, *t* = -10.54, *p* < .001, *LLCI* = -.60, *ULCI* = -.41, while loneliness was a positive predictor of depression, *Coeff* = 1.93, *SE* = .17, *t* = 11.66, *p* < .001, *LLCI* = 1.61, *ULCI* = 2.27, which in turn was a positive predictor of poor sleep quality, *Coeff* = .51, *SE* = .06, *t* = 8.98, *p* < .001, *LLCI* = .40, *ULCI* = .62. The total effect of family identification on poor sleep quality was significant, *Effect* = -.70, *SE* = .15, *t* = -4.68, *p* < .001, *LLCI* = -.99, *ULCI* = -.40, and this became non-significant when loneliness and depression were accounted for (i.e., the direct effect), indicating full mediation, *Effect* = -.13, *SE* = .15, *t* = -.91, *p* = .37, *LLCI* = -.43, *ULCI* = .16. See Figure 1 for the mediation model.

(FIGURE 1)

T1 Analysis: Predicting Insomnia Severity

Supporting Hypothesis 6a, a significant effect of family identification T1 on insomnia severity T1 through loneliness T1 and depression T1 was found, *Effect* = -0.74, *Boot SE* = .15, *Boot LLCI* = -1.04, *Boot ULCI* = -.46. Family identification was a negative predictor of loneliness, *Coeff* = -.50, *SE* = .05, *t* = -10.58, *p* < .001, *LLCI* = -.59, *ULCI* = -.41, while loneliness was a positive predictor of depression, *Coeff* = 1.94, *SE* = .17, *t* = 11.68, *p* < .001, *LLCI* = 1.61, *ULCI* = 2.27, which in turn was a positive predictor of insomnia, *Coeff* = .77, *SE* = .10, *t* = 7.92, *p* < .001, *LLCI* = .58, *ULCI* = .96. The total effect of family identification on insomnia was significant, *Effect* = -.97, *SE* = .25, *t* = -3.96, *p* = .0001, *LLCI* = -1.46, *ULCI* = -.49, and this became non-significant when loneliness and depression were accounted for (direct effect), indicating full mediation, *Effect* = -.01, *SE* = .25, *t* = -.05, *p* = .96, *LLCI* = -.51, *ULCI* = .48. See Figure 2 for the mediation model.

(FIGURE 2)

Longitudinal Mediation Analyses

Model six in version 3.0 of Hayes' (2017) PROCESS macro was again used to test Hypothesis 6b: that loneliness and depression at T2 will mediate the relationship between family identification at T1 and sleep quality and insomnia at T2, so that stronger family identification T1 will predict lower loneliness T2, which in turn will predict lower depression T2, which in turn will predict better sleep quality and insomnia T2. The analyses involved the same control variables as the cross-sectional analyses, but also included the T1 versions of the mediator and predictor variables (i.e., loneliness T1, depression T1, sleep quality T1 and insomnia T1).[2] For completeness, both analyses were re-run with the positions of the mediators reversed (i.e., depression T2 placed before loneliness T2). The resultant models were both non-significant.

Longitudinal T1/T2 Analysis: Predicting Sleep Quality

Supporting Hypothesis 6b, a significant effect of family identification T1 on poor sleep quality T2 though loneliness T2 and depression T2 was found, *Effect* = -0.16, *Boot SE* = .07, *Boot*

LLCI = -31, *Boot ULCI* = -.04. Family identification T1 was a negative predictor of loneliness T2, *Coeff* = -.19, *SE* = .07, *t* = -2.64, *p* = .009, *LLCI* = -.33, *ULCI* = -.05, while loneliness T2 was a positive predictor of depression T2, *Coeff* = 1.73, *SE* = .24, *t* = 7.35, *p* < .001, *LLCI*= 1.26, *ULCI* = 2.20, which in turn was a positive predictor of poor sleep quality T2, *Coeff* = .49, *SE* = .10, *t* = 5.06, *p* < .001, *LLCI*= .30, *ULCI* = .68. The total effect of family identification T1 on poor sleep quality T2 was non-significant, *Effect* = -.26, *SE* = .20, *t* = -1.33, *p* = .19, *LLCI* = -.65, *ULCI* = .13 (while this finding does not support hypothesis 1b, it is appropriate to test for indirect effects when the total effect is non-significant-this is known as indirect-only mediation: Zhao, Lynch, & Chen, 2010), and this became weaker when loneliness T2 and depression T2 were accounted for (direct effect), *Effect* = -.02, *SE* = .18, *t* = -.08, *p* = .93, *LLCI* = -.38, *ULCI* = .35. See Figure 3 for the mediation model.

(FIGURE 3)

Longitudinal T1/T2 Analysis: Predicting Insomnia Severity

Supporting Hypothesis 6b, a significant effect of family identification T1 on insomnia severity T2 though loneliness T2 and depression T2 was found, *Effect* = -0.24, *Boot SE* = .11, *Boot LLCI* = -.48, *Boot ULCI* = -.06. Family identification T1 was a negative predictor of loneliness T2, *Coeff* = -.19, *SE* = .07, *t* = -2.65, *p* = .009, *LLCI* = -.33, *ULCI* = -.05, while loneliness T2 was a positive predictor of depression T2, *Coeff* = 1.75, *SE* = .23, *t* = 7.48, *p* < .001, *LLCI*= 1.28, *ULCI* = 2.21, which in turn was a positive predictor of insomnia severity T2, *Coeff* = .72, *SE* = .17, *t* = 4.21, *p* = .0001, *LLCI*= .38, *ULCI* = 1.05. The total effect of family identification T1 on insomnia severity T2 was non-significant, *Effect* = -.51, *SE* = .34, *t* = -1.49, *p* = .14, *LLCI* = -1.18, *ULCI* = .17, and this became weaker when loneliness T2 and depression T2 were accounted for (direct effect), *Effect* = -.09, *SE* = .33, *t* = -.26, *p* = .79, *LLCI* = -.73, *ULCI* = 56. See Figure 4 for the mediation model.

(FIGURE 4)

Discussion

Using both cross-sectional and longitudinal analyses, the present study provides a novel and rigorous exploration of the relationship between group identification and sleep quality/insomnia. The

results support the hypotheses: family identification was a negative predictor of poor sleep quality/insomnia severity cross-sectionally, and family identification was also a negative predictor of depression and loneliness. Moreover, depression and loneliness were both positive predictors of poor sleep quality/insomnia severity, and mediated the relationship between family identification and sleep quality/insomnia severity both cross-sectionally and longitudinally (with family identification negatively predicting loneliness, which in turn positively predicted depression, which in turn positively predicted poor sleep quality/insomnia severity). These results were found after controlling for participants' age, gender, relationship status, highest level of education, and whether they were looking after a young child, as well as controlling for the T1 versions of the mediator and outcome variables in the longitudinal mediation models. While it should be noted that the total effect relationship between T1 family identification and T2 sleep quality/insomnia severity was not significant, it was the case that there was a significant mediated relationship between the variables via T2 loneliness and T2 depression (i.e., an indirect-only mediation: Zhao et al., 2010).

These results support empirical evidence relating to both loneliness and to Social Cure theorising. In terms of the loneliness literature, the results are consistent with observations regarding the relationship between loneliness and depression (e.g., Hakulinen et al., 2018; Holt-Lunstad, et al., 2015). Moreover, the finding that the serial mediation models became non-significant when depression was placed before loneliness supports work indicating that loneliness predicts depression regardless of initial depression levels (Cacioppo et al., 2010).

These results also extend those of Matthews at al. (2017), who found that loneliness was negatively associated with sleep quality in young adults. The present results revealed the same relationship in a sample with a wider age range (18-76 years), but also showed that this relationship is part of a larger story, with loneliness and depression being mediating variables in the relationship between family identification and sleep quality or insomnia severity. Nonetheless, the findings are consistent with the Evolutionary Theory of Loneliness (Cacioppo & Cacioppo, 2018) tested by Matthews et al. (2017), which posits a negative relationship between loneliness and sleep quality due

to loneliness promoting social threat. This in turn triggers physiological responses such as cortisol production, thereby undermining sleep attempts.

The results are also consistent with the Social Cure perspective (Jetten et al., 2012). The present study replicates the previously-established negative relationship between group identification and loneliness (e.g., Haslam et al., 2016; Jose & Lim, 2014) and between group identification and depression (e.g., Cruwys et al., 2014), but extends this by showing evidence of serial mediation between these variables: family identification is a negative predictor of loneliness, which is in turn a positive predictor of depression. Moreover, the model presented in the present study further extends the Social Cure literature by showing that, in turn, depression is a positive predictor of poor sleep quality/insomnia severity. Indeed, the present study is the first to show a mediated relationship between family identification and sleep quality/insomnia severity. This is an important contribution to the Social Cure literature: although the present study features self-report sleep measures, the fact that the study involves the measurement of a biologically-driven behaviour known to be linked intimately to both physical and mental health allows the present research to move beyond the more subjective outcome measures that are commonly utilised in Social Cure research, such as life satisfaction (Wakefield et al., 2017). Moreover, the fact that the results support the study's predictions both cross-sectionally and longitudinally is particularly noteworthy, as the longitudinal aspect of this study allows for stronger claims to be made regarding causal processes than solely cross-sectional data would allow.

These results also extend research exploring the important relationship between family identification and well-being (Sani et al., 2012, 2017; Wakefield et al., 2016, 2017; Naughton et al., 2015). Specifically, the findings from the present study show how experiencing identification with one's family can be a buffer to loneliness and its detrimental mental health effects, which in turn supports better sleep. Since a negative relationship between family identification and paranoid ideation has already been established (Sani et al., 2017), and the relationship between paranoia and loneliness is well-known (Riggio & Kwong, 2011), future research could explore the role played by

paranoid ideation in affecting the relationship between group identification and sleep quality/insomnia severity.

The results also provide an initial suggestion of a link between the Social Cure perspective and the Evolutionary Theory of Loneliness (EVL). While evidence indicates that loneliness has the potential to prime feelings of social threat (Cacioppo & Cacioppo, 2018), it could also be the case that a lack of group identification (which we have shown to predict loneliness) may be the initial catalyst for these feelings. This possibility seems consistent with the EVL: while people who identify strongly with a group are likely to experience altruistic behaviour from fellow group members (Jetten et al., 2012; Levine, Prosser, Evans, & Reicher, 2005) those lacking these meaningful connections are less likely to receive such support, and might therefore be likely to see the world as selfish and threatening. Future research should explore this possibility in more depth.

In their test of the EVL, Matthews et al. (2017) also noted that the association between loneliness and poor sleep quality in the young adults they recruited was especially pronounced for those who had experienced trauma, which they explained as being due to the increase in hypervigilance towards social threats caused by such experiences. A number of researchers working within the Social Cure perspective have explored the idea of trauma, and have differentiated between individual trauma and shared trauma (Kellezi & Reicher, 2012). While individual trauma is experienced exclusively by the individual, shared trauma is experienced by a whole social group (e.g., discrimination, war, or natural disaster), and can lead members to identify more strongly with the group in question. In turn, this identification (and the subsequent support received from fellow group members and increased collective efficacy to deal with the events) can buffer the negative impact of trauma on group members' health and well-being (Kellezi, Reicher & Cassidy, 2009; Muldoon et al., 2017). It would thus be fruitful for future research to explore the relationship between prior/current trauma, group identification, loneliness, depression, and sleep quality/insomnia severity.

Practically, this work supports the idea that interventions designed to help lonely and depressed people create new group identifications and to reconnect with old groups (such as Groups4Health; Haslam et al., 2016) have the potential to benefit sleep quality. Since high-quality

sleep is known to strengthen mental health (Kaneita et al., 2007), and enhanced mental health furnishes people with more of the social resources required for making connections with others (e.g., Hirschfeld et al., 2000), such interventions have the potential to create a 'virtuous cycle' which is likely to enhance health and well-being over time (Miller et al., 2017). Health professionals such as GPs and Health Psychologists should consider the possibility of encouraging patients with sleep problems to engage with an intervention like Groups4Health, or consider offering these patients a referral to one of the increasingly common 'social prescribing' initiatives aimed to help connect patients with groups within their local communities (Halder et al., 2018). Health Psychologists should also explore how such knowledge might prevent sleep problems through public education regarding the importance of social connection.

This work is not without limitations, however. For instance, it should be noted that this research involved a self-report survey, which may be prone to participants giving rushed or incorrect responses. The issue of relatively high levels of participant attrition at T1 and T2 is also important. However, the fact that the T2 responders and non-responders did not differ significantly on the key variable suggests this may not be too problematic. Additionally, a more objective measure of sleep quality/insomnia severity would be preferable, although both scales have been validated and utilised frequently (e.g., Mollayeva et al., 2016; Morin, Belleville, Bélanger, & Ivers, 2011). Moreover, the Pittsburgh Sleep Quality Inventory does include a question about whether the participant uses sleeping medication, which could be seen as an objective measure of sleep-related issues. Nonetheless, future work could benefit from more objective sleep quality and insomnia measures (e.g. actigraphy or polysomnography).

In conclusion, the present study highlights the importance of experiencing subjective feelings of belonging within our social worlds (rather than just having contact with others), and the crucial role that this sense of belongingness plays in affecting our social connectedness, and, ultimately, our well-being. Being part of a meaningful social group not only plays a critical role in predicting our mental health, but also in predicting the quality of a fundamentally important health-related behaviour: sleep.

Endnotes

[1] While this paper is based on the assumption that loneliness, sleep quality, family identification, and depression are distinct aspects of human functioning, it could be argued that the former two variables are merely symptoms of major depressive disorder: an argument strengthened by the fact that some measures of depression include items about sleep problems and loneliness. While we have presented evidence in the Introduction supporting the idea that these variables are unique (albeit related) constructs (e.g., Cacioppo et al., 2010), we also entered the family identification items, loneliness items, depression items, insomnia items, and sleep quality item into a factor analysis (using oblique rotation, as we expected the factors to correlate, and only including items in a factor that loaded at .40 or above). The pattern matrix revealed four factors with eigenvalues above 1.00: the seven depression items loaded onto the first factor (eigenvalue = 9.51), the seven insomnia items and the sleep quality item loaded onto the second factor (as did one of the loneliness items, but the loading was low: -.46) (eigenvalue = 3.45), the four family identification items loaded onto the third factor (eigenvalue = 1.83), and the six loneliness items loaded onto the fourth factor (eigenvalue = 1.91). These results support our belief that these are separate constructs.

[2] We confirmed the appropriateness of using the PROCESS macro to conduct longitudinal analyses in this manner by contacting Amanda Kat Montoya, who has worked on mediation/moderation macros with Andrew Hayes.

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Table 1

Means and Standard Deviations for Variables, and Intercorrelations

Variable	1	2	3	4	5	6	7	8	9
1. Family Id T1 (1-7) ($M = 1.75$; $SD = 0.87$)	-								
2. Loneliness T1 (1-7) ($M = 3.63$; $SD = 1.43$)	-.53***	-							
3. Depression T1 (0-21) ($M = 6.19$; $SD = 4.41$)	-.38***	.64***	-						
4. Poor Sleep Quality T1 (0-21) ($M = 8.41$; $SD = 3.82$)	-.25***	.38***	.59***	-					
5 Insomnia T1 (0-28) ($M = 11.33$; $SD = 6.43$)	-.21***	-.37***	.55***	.79***	-				
6. Family Id T2 (1-7) ($M = 5.32$, $SD = 1.37$)	.83***	-.52***	-.43***	-.30**	-.23*	-			
7. Loneliness T2 (1-7) ($M = 3.66$, $SD = 1.57$)	-.59***	.80***	.59***	.37***	.35***	-.59***	-		
8. Depression T2 (0-21) ($M = 2.97$; $SD = 0.99$)	-.57***	.58***	.74***	.53***	.47***	-.59***	.74***	-	
9. Poor Sleep Quality T2 (0-21) ($M = 8.14$, $SD = 3.83$)	-.35***	.33***	.55***	.69***	.55***	-.41***	.39***	.63***	-
10. Insomnia T2 (0-28) ($M = 10.42$; $SD = 6.27$)	-.39***	.41***	.59***	.61***	.68***	-.34***	.46***	.66***	.78***

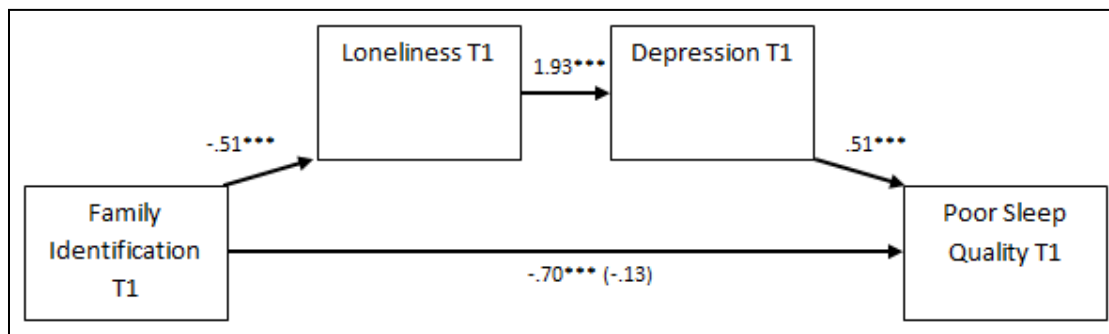Note: ***$p < .001$, **$p < .01$

Figure 1. Model depicting the significant indirect effect of family identification T1 on poor sleep quality T1 via loneliness T1 and depression T1. Gender, age, relationship status, education, and whether participants are caring for a young child were controlled for in the analysis. Bracketed coefficient is the direct effect. Note: ***$p < .001$.
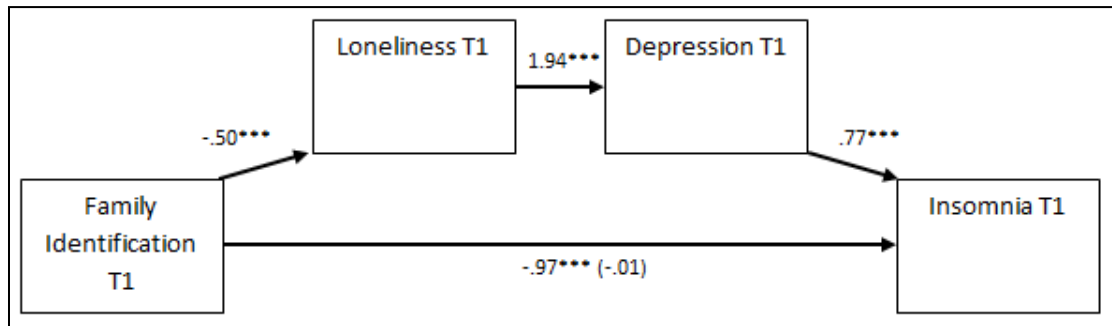
Figure 2. Model depicting the significant indirect effect of family identification T1 on insomnia T1 via loneliness T1 and depression T1. Gender, age, relationship status, education, and whether participants are caring for a young child were controlled for in the analysis. Bracketed coefficient is the direct effect. Note: ***$p < .001$.
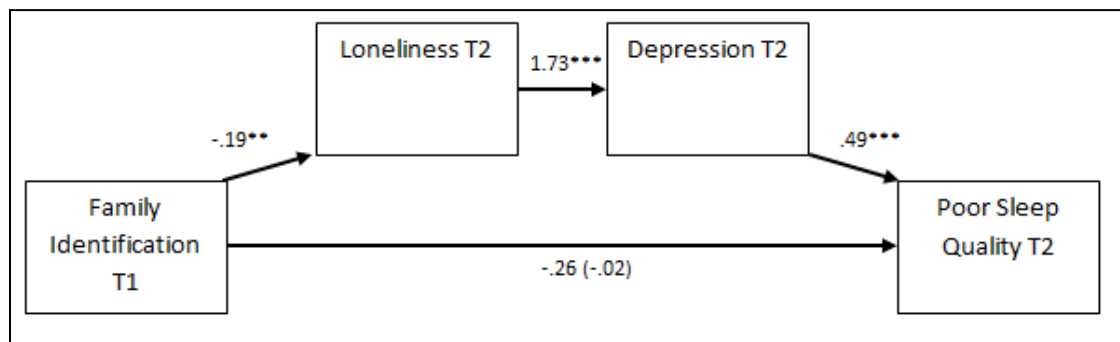

Figure 3. Model depicting the significant indirect effect of Family Identification T1 on poor sleep quality T2 via loneliness T2 and depression T2. Loneliness T1, depression T1, poor sleep quality T1, gender, age, relationship status, education, and whether participants are caring for a young child at T2 were controlled for in the analysis. Bracketed coefficient is the direct effect. Note: ***$p < .001$, **$p < .01$.
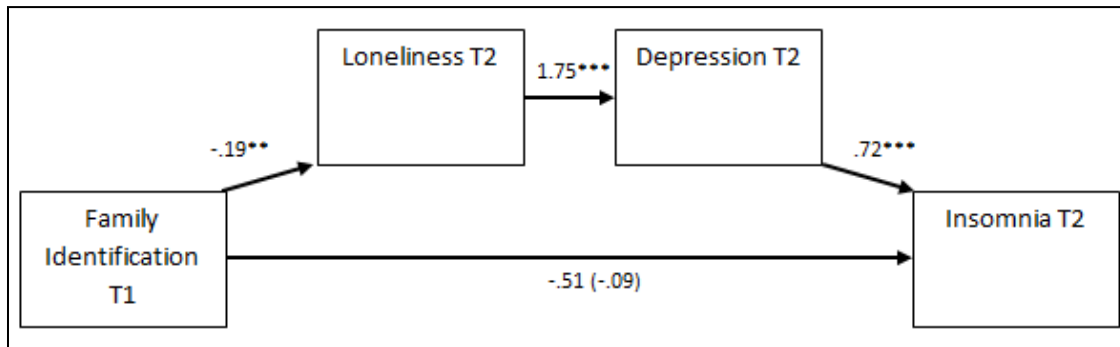
Figure 4. Model depicting the significant indirect effect of family identification T1 on insomnia T2 via loneliness T2 and depression T2. Loneliness T1, depression T1, insomnia T1, gender, age, relationship status, education, and whether participants are caring for a young child at T2 were controlled for in the analysis. Bracketed coefficient is the direct effect. Note: ***$p < .001$, **$p < .01$.