Targeting the link between loneliness and paranoia via an interventionist-causal model framework

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ABSTRACT

Targeting the antecedents of paranoia may be one potential method to reduce or prevent paranoia. For instance, targeting a potential antecedent of paranoia – loneliness – may reduce paranoia. Our first research question was whether loneliness heightens subclinical paranoia and whether negative affect may mediate this effect. Second, we wondered whether this potential effect could be targeted via two interventionist pathways in line with an interventionist-causal model approach: (1) decreasing loneliness, and (2) intervening on the potential mediator – negative affect. In Study 1 ($N = 222$), recollecting an experience of companionship reduced paranoia in participants high in pre-manipulation paranoia but not in participants low in pre-manipulation paranoia. Participants recollecting an experience of loneliness, on the other hand, exhibited increased paranoia, and this effect was mediated by negative affect. In Study 2 ($N = 196$), participants who utilized an emotion-regulation strategy, cognitive reappraisal, to regulate the negative affect associated with loneliness successfully attenuated the effect of loneliness on paranoia. Targeting the effect of loneliness on paranoia by identifying interventionist pathways may be one promising route for reducing and preventing subclinical paranoia.

1. Introduction

Loneliness is a distressing feeling that arises from individuals perceiving their social needs as failing to align with the quantity and quality of their actual social relationships (e.g., Hawkley and Cacioppo, 2010; Peplau, 1982; Pinquart and Sorensen, 2001). Loneliness is not merely social isolation, individuals who are socially isolated do not necessarily perceive themselves as lonely, and individuals who are surrounded by others still experience loneliness. Importantly, experiencing loneliness is associated with a variety of psychopathologies, including personality disorders, psychosis, and depression (for overview see Hawkley and Cacioppo, 2010).

Recent research has focused on a potential association between loneliness and paranoia – persecutory thinking (e.g., Badcock et al., 2015; Gayer-Anderson and Morgan, 2013; Jaya et al., 2016; Lamster et al., 2017). For example, an experience sampling study showed that individuals who are alone tend to more frequently experience paranoia (Myin-Germeys et al., 2001). Further, a relationship between loneliness and paranoia has been observed cross-sectionally in healthy individuals (Jaya et al., 2016) and in individuals with first-episode psychosis (Sündermann et al., 2014). Aside from these correlational findings, a

causal relationship between loneliness and subclinical paranoia has also been observed. An experimental study demonstrated that increasing or decreasing loneliness using a false-feedback experimental paradigm leads to increased and decreased subclinical paranoia (Lamster et al., 2017).

Building on this research, in the current article we investigate the complexity of the previously observed link between loneliness and paranoia and consider this link from an interventionist perspective. Specifically, we focus on two interventionist pathways in line with an interventionist-causal model approach. An interventionist-causal approach entails examining causation in terms of "what would happen under interventions" (Kendler and Campbell, 2009; p. 881). Researchers studying delusional thinking have recently called for this approach as a promising method to discover ways to prevent and attenuate delusions (e.g., Freeman, 2011). Specifically, Freeman (2011) notes that an interventionist-causal model approach regarding delusions entails a focus on "one putative causal factor at a time, show that an intervention can change it, and examine the subsequent effects on the delusional beliefs" (p. 135). Here we focus on loneliness as a putative causal factor of subclinical paranoia.

We considered two interventionist causal pathways regarding the

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relationship between loneliness and paranoia. First, does decreasing loneliness decrease paranoia? In Study 1 we examined whether we can conceptually replicate the findings of Lamster et al. (2017) that reducing loneliness reduces subclinical paranoia. The second interventionist pathway we considered was whether paranoia can be reduced by targeting the mechanisms underlying the link between loneliness and paranoia (see Reininghaus et al., 2015). In Study 2 we examined whether the effects of loneliness on paranoia can be attenuated if a potential mediator is hampered or eliminated.

To potentially intervene on a mediator underlying the link between loneliness and paranoia, we must first identify such a mediator. In line with numerous theoretical models positing an affective pathway to paranoia (e.g. Fowler et al., 2011; Freeman and Garety, 2014; Jaya et al., 2017; Kesting and Lincoln, 2013; Kramer et al., 2014), we propose that negative affect (e.g., feeling sad, irritated, embarrassed) may qualify as such a mediator. Aligning with this possibility, researchers have suggested that the relationship between loneliness and paranoia is mediated by depression (Jaya et al., 2016) and anxiety (Sündermann et al., 2014) – both depression and anxiety are related to negative affect (see Watson and Clark, 1984; see Tellegen, 1985). Further, past research has found negative affect to relate to loneliness (e.g., low pleasure, Mehrabian and Stefi, 1995; seeing the world through a negative lens, Duck et al., 1994) as well as paranoia (Freeman, 2007). Of course, however, numerous other possible mechanisms may also underlie a potential effect of loneliness on paranoia (e.g., negative beliefs about the self; Freeman and Garety, 2014), though we do not examine these here.

If negative affect indeed mediates the effect of loneliness on paranoia, how could one target this mediating process? One well-known intervention technique that can change negative affect is an emotional regulation technique refereed to as cognitive reappraisal – cognitive reappraisal has been found to reduce negative affect with regard to both cognitive measures (self-report) and psychophysiological measures (Gross, 2002). Cognitive reappraisal involves changing the direction of an affective response (e.g., sadness) by reframing or reinterpreting the original emotional stimulus (e.g., Gross, 1998; Ochsner et al., 2002; Ray et al., 2010). For example, cognitively reappraising a past lonely experience could entail reframing the experience as a learning experience in order to reduce resulting negative affect. Such reframing could, by decreasing the negative emotional impact of recalling a lonely experience, reduce the effect of loneliness on paranoia.

We wish to note that in the present studies we solely examine the link between loneliness and *subclinical* paranoia – paranoia in the non-clinical population. Researchers have noted that "paranoid thoughts in non-clinical populations are phenomena of interest in their own right and may inform our understanding of delusions" (Freeman et al., 2005; p. 427). Furthermore, a spectrum of paranoia exists in the general population, that is, paranoid thoughts exist in non-clinical individuals and importantly, paranoia has been associated with important theoretical variables across this spectrum (e.g., Freeman et al., 2010).

1.1. The present research

In Study 1 we examined whether we could conceptually replicate the effects of Lamster et al. (2017). First, does increasing loneliness – induced in the current studies by having participants recall and elaborate on a past lonely experience via mental imagery – lead to increased subclinical paranoia? Second, considering intervention possibilities, does decreasing loneliness – induced by having participants recall and elaborate on a past experience of companionship via mental imagery – lead to decreased subclinical paranoia? Study 1 also examined whether the link between loneliness and subclinical paranoia is mediated by negative mood (e.g., sadness, embarrassment). Study 2 then built on the findings of Study 1. Study 2 examined whether intervening on the link between loneliness and paranoia at the level of the potential mediator – negative affect – can attenuate the effect of

loneliness on paranoia. Specifically, participants cognitively reappraised the lonely experience they were asked to recall in an effort to reduce its associated negative affect.

2. Study 1

2.1. Design and procedure

Study 1 examined whether increasing and decreasing loneliness influences people's level of subclinical paranoia and whether this potential effect is mediated by negative affect. The study was a mixed-design with a 3-level between-subjects factor, loneliness (high loneliness, low loneliness, no-treatment control) and a 2-level within-subjects factor, paranoia (pre-manipulation paranoia, post-manipulation paranoia). After participants' pre-manipulation state level of paranoia was assessed, participants recalled a past lonely experience (high loneliness condition), a past experience of companionship (low loneliness condition), or did not complete a manipulation (no-treatment control). Thereafter, we measured participants' negative affect and then again, participants' state paranoia. All variables were assessed in a state manner (e.g., negative affect) given the experimental nature of the presented studies.

2.2. Sample selection and recruitment

Study 1 was conducted with a non-clinical online sample. Participants were recruited on Mechanical Turk (MTurk; see Buhrmester et al., 2011), an online data collection service. Inclusion criteria were being 18 years or older and residing in the United States. Exclusion criteria included having been approved for less than 95% of past studies on Mturk or having completed less than 100 studies on MTurk. Potential participants were told they would complete a 5-min survey that would ask them about their feelings, attitudes, and how they see the world.

2.3. Participants

We conducted a sample-size estimation based on the observed effect-size of the effect of loneliness on paranoia observed by Lamster et al. (2017), $d = 0.685$. Our sample-size estimation revealed that we needed to recruit approximately 60 participants per condition (180 participants in total) to achieve 95% $(1 − \beta)$ power at a 0.05 alpha level $(\alpha = 0.05)$. To account for potential study dropouts, we recruited 228 (117 female) adults. Six participants were excluded for failing an attention check item (see Supplemental material), resulting in a final sample size of 222 participants (mean age of 36 years, $M = 36.39$, $SD = 13.12$)

2.4. Materials

2.4.1. Pre-manipulation paranoia

We assessed paranoia using an adapted version of the brief state Paranoia Checklist developed by Schlier et al. (2016); e.g., "I currently feel that people are laughing at me" 1 = *Do not believe*, 2 = *Believe a little*, 3 = *Believe it somewhat*, 4 = *Believe it a lot*, 5 = *Absolutely believe it*). The brief state Paranoia Checklist includes 5 items from the original Paranoia Checklist (Freeman et al., 2005) that are sensitive to fluctuations in state paranoia.

2.4.2. Loneliness manipulation

Participants were randomly assigned to one of three conditions (high loneliness, low loneliness, no-treatment control). In the high loneliness condition, participants were first given a definition of loneliness (in line with Hawkley and Cacioppo (2010)). Participants were then asked to recall and record a specific situation in which they felt lonely (within the last 2 years), and were thereafter asked to mentally

visualize this lonely situation (see Supplemental material for comparison of the lonely manipulation used by Lamster et al. (2017)). Specifically, participants were told: "Loneliness is an aversive, painful condition that is made up of different feelings and beliefs. It arises as a reaction to perceived absence of intimacy and social needs and is the result of subjectively seeing a difference between one's desired and actual level of social relationships. Name a concrete situation in which you felt lonely (the moment in which the feeling of loneliness was particularly strong). Describe the situation and your feelings and thoughts during that moment. This experience of loneliness should not have occurred longer than 24 months ago. If you have not had a lonely experience in the last 24 months, then you may select an experience that occurred further in the past. (Max four sentences, bullet points are acceptable)."

Participants in the low loneliness condition (Study 1) followed the same procedure except recollected a situation in which they experienced companionship (i.e., low loneliness). Specifically they were given the following prompt: "Please read carefully: Companionship and feeling socially happy is a healthy and joyful condition that is made up of different feelings and beliefs. It arises as a reaction to perceived satisfaction of intimacy and social needs and is the result of subjectively seeing one's desired and actual level of social relationships as matching. Name a concrete situation in which you felt companionship (the moment in which the feeling of companionship was particularly strong). Describe the situation and your feelings and thoughts during that moment. This experience of companionship should not have occurred longer than 24 months ago. If you have not had an experience of close companionship in the last 24 months, then you may select an experience that occurred further in the past (Max four sentences, bullet points are acceptable)." Participants in the no-treatment control condition completed a word-search task (see Supplemental material).

2.4.3. Negative affect

After the manipulation, we assessed participants' negative affect using an intensity of emotion scale (Stemmler et al., 2001; e.g., "I currently feel: gloomy, depressed, sad, dejected" 1 = *Does not at all apply* to 10 = *Strongly applies*).

2.4.4. Manipulation check

The study included a manipulation check item: "I currently feel: lonely, alone, isolated, excluded" 1 = *Does not at all apply* to 10 = *Strongly applies*.

2.4.5. Post-manipulation paranoia

Participants finally completed the same paranoia measure they completed pre-manipulation to assess post-manipulation paranoia.

2.5. Results

2.5.1. Paranoia scores

Paranoia was calculated by averaging the five paranoia items (separately for pre- and post-manipulation paranoia) and then conducting log-transformations on the resulting averages (to reduce skewness). Pre- and post-manipulation paranoia in Study 1: $M = 0.17$, $SD = 0.18$, $\alpha = 0.88$, and $M = 0.15$, $SD = 0.18$, $\alpha = 0.89$, respectively. For the non-transformed descriptive statistics see Table 1.

2.5.2. Manipulation checks

We conducted a General Linear Model (GLM) with loneliness (high loneliness, low loneliness, no-treatment control) as a between-subjects factor and the manipulation check item (i.e., "I currently feel: lonely, alone, isolated, excluded") as the dependent variable. Participants in the lonely condition, $M = 5.08$, $SD = 3.23$, reported feeling more lonely than those in the low loneliness, $M = 1.91$, $SD = 1.86$, t (142) = 7.35, $p < 0.001$, $d = 1.23$, and no-treatment control conditions, $M = 2.40$, $SD = 2.32$, $t(155) = 6.53$, $p < 0.001$, $d = 1.04$.

Participants in the low loneliness condition, however, did not report feeling less lonely than participants in the no-treatment control condition, $t(141) = 1.13$, $p = 0.258$, $d = 0.23$.

2.5.3. Loneliness influences paranoia

GLMs with loneliness condition (high loneliness, low loneliness, no-treatment control) as a between-subjects factor, pre-manipulation paranoia as a covariate, and post-manipulation paranoia as the dependent variable observed a difference in post-manipulation paranoia depending on loneliness condition, $F(3218) = 9.52$, $p < 0.001$, $d = 0.590$. Pairwise comparisons revealed that participants in the high loneliness condition, $M = 0.19$, $SD = 0.11$, exhibited higher levels of paranoia than those in the no-treatment control, $M = 0.14$, $SD = 0.11$, t (155) = 3.06, $p = 0.002$, $d = 0.488$, and than those in the low loneliness condition, $M = 0.12$, $SD = 0.10$, $M = 0.19$, $SD = 0.11$, t (142) = 4.29, $p < 0.001$, $d = 0.718$. Participants in the low loneliness condition did not exhibit decreased paranoia compared to participants in the no-treatment control condition, $M = 0.14$, $SD = 0.11$, $t(141)$ = 1.11, $p = 0.245$, $d = 0.186$, though the effect was in the predicted direction.[1]

2.5.4. Loneliness leads to paranoia via negative affect

To calculate negative affect, we averaged each participant's responses on the intensity of emotion scale (e.g., "I currently feel: gloomy, depressed, sad, dejected"). We then investigated whether negative affect mediated the relationship between loneliness and paranoia (the high loneliness and control condition) by conducting a mediation analysis using the SPSS PROCESS macro (Hayes, 2012). Using PROCESS model 4, one thousand bootstrap samples were created to calculate 95% bias-corrected and accelerated (BCa) confidence intervals to test the significance of the mediation. We entered loneliness manipulation (high loneliness = 1, no-treatment control = 0) as the independent variable. The low loneliness condition was not included in the analysis because we found no difference in negative affect between the low loneliness, $M = 2.10$, $SD = 1.47$, and no-treatment control conditions, $M = 2.32$, $SD = 1.48$, $t(141) = 0.80$, $p = 0.424$, $d = 0.15$. Negative affect was entered as the mediator, pre-manipulation paranoia as a covariate, and post-manipulation paranoia as the dependent variable.

As predicted, the influence of loneliness on paranoia was mediated by negative affect. While increasing loneliness overall increased paranoia compared to the control condition, $b = 0.28$, $p = 0.001$ (total effect), this effect was no longer significant when accounting for participants' negative affect, $b = 0.01$, $p = 0.880$ (direct effect), indicating a complete mediation (see Table 2)

2.5.5. Exploratory analysis: Loneliness decreases paranoia in individuals high in subclinical paranoia

Unexpectedly, as noted earlier, recollecting an experience of companionship (i.e., the low loneliness condition) did not significantly reduce paranoia (compared to the no-treatment control condition). These results were surprising given research indicating that social support reduces paranoia (Norman et al., 2005) and given the findings of Lamster et al. (2017) that perceiving oneself as less lonely may decrease paranoia. Possibly, this null effect was observed because of a floor effect. Indeed, 75% of participants' pre-manipulation paranoia scores were between 1 and 2 on the 5-point scale. Furthermore, in an exploratory box-and-whiskers plot the median of participants' pre-manipulation paranoia scores (1.20) was shifted to the lower part of the box; Uttl (2005) notes that this is one method to identify floor effects. These values and analysis indicate that there was little room for participants' subclinical paranoia to decrease.

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[1] The same overall results were observed when analyzing the data of Studies 1 and 2 using a repeated-measures approach; that is, calculating an interaction term between loneliness condition and pre and post-manipulation paranoia.

Table 1
Descriptive statistics in Studies 1 and 2.

Condition	Study 1			Study 2		
	High loneliness	Low loneliness	Control	High loneliness	Cognitive reappraisal	Control
Pre-Manipulation Paranoia	$M = 1.67$	$M = 1.63$	$M = 1.62$	$M = 24.55$	$M = 22.43$	$M = 20.25$
	$SD = 0.82$	$SD = 0.80$	$SD = 0.83$	$SD = 22.20$	$SD = 19.73$	$SD = 19.98$
Post-Manipulation Paranoia	$M = 1.73$	$M = 1.42$	$M = 1.49$	$M = 25.43$	$M = 20.31$	$M = 15.42$
	$SD = 0.86$	$SD = 0.72$	$SD = 0.80$	$SD = 22.56$	$SD = 21.02$	$SD = 19.29$
Loneliness	$M = 5.08$	$M = 1.91$	$M = 2.40$	$M = 5.16$	$M = 2.95$	$M = 2.37$
	$SD = 3.23$	$SD = 1.86$	$SD = 2.32$	$SD = 3.03$	$SD = 2.53$	$SD = 2.04$
Negative Affect	$M = 4.34$	$M = 2.15$	$M = 2.47$	$M = 4.48$	$M = 3.12$	$M = 2.79$
	$SD = 2.15$	$SD = 1.53$	$SD = 1.66$	$SD = 2.16$	$SD = 1.74$	$SD = 1.68$

Table 2
Mediation effects in Studies 1 and 2. The effect of loneliness (high loneliness versus no-treatment control) on paranoia was mediated by negative affect.

Total effect	Study 1	$b = 0.283$, $SE = 0.087$, $t(155) = 3.25$, $p = .001$, 95% CI: [0.111, 0.455]
	Study 2	$b = 0.274$, $SE = 0.096$, $t(130) = 2.85$, $p = .005$, 95% CI: [0.084, 0.464]
Direct effect	Study 1	$b = 0.014$, $SE = 0.091$, $t(155) = 0.15$, $p = .881$, 95% CI: [− 0.166, 0.193]
	Study 2	$b = 0.079$, $SE = 0.093$, $t(130) = 0.86$, $p = .394$, 95% CI: [− 0.104, 0.262]
Indirect effect	Study 1	$b = 0.269$, $SE = 0.077$, 95% CI: [0.140, 0.440]
	Study 2	$b = 0.195$, $SE = 0.054$, 95% CI: [0.110, 0.336]

Note. b = unstandardized estimate. SE = Standard Error. CI = Confidence Interval.

Supporting the notion that reducing loneliness did not reduce paranoia because of a floor effect, exploratory, post-hoc analyses indicated that the low loneliness manipulation did decrease paranoia in participants high but not low in pre-manipulation subclinical paranoia. A multiple linear regression revealed that the effect of the low loneliness condition compared to no-treatment control on post-manipulation paranoia was moderated by participants' pre-manipulation paranoia: interaction effect, $t(140) = 2.45$, $p = 0.016$. While participants high in subclinical paranoia ($+ 1 SD$) exhibited a significant decrease in paranoia from pre-manipulation to post-manipulation compared to the no-treatment control condition, $\beta = -0.18$, $p = 0.015$, the same was not true of participants low in subclinical paranoia ($- 1 SD$) at baseline, $\beta = 0.07$, $p = 0.326$.

2.6. Discussion

Study 1 replicated the findings of Lamster et al. (2017) that increasing loneliness heightens individuals' subclinical paranoia. We also found this causal effect to be mediated by negative affect, in line with a number of non-experimental findings (e.g., Jaya et al., 2016; Sündermann et al., 2014). Unexpectedly, however, we failed to replicate the findings of Lamster et al. (2017) that decreasing loneliness decreases paranoia. Exploratory, post-hoc analyses indicated that the low loneliness manipulation did decrease subclinical paranoia for participants high in subclinical paranoia at baseline, but not for those low in subclinical paranoia at baseline.

3. Study 2

3.1. Introduction

The goal of Study 2 was to investigate the second pathway posited by the interventionist-causal model – intervention on the mediator. Therefore, in Study 2, we examined whether individuals can utilize an emotion regulation strategy, cognitive reappraisal (e.g., Gross, 1998; Ochsner et al., 2002; Ray et al., 2010), to regulate the negative affect that loneliness was found to induce in Study 1. In turn, they may be able

to attenuate the effect of loneliness on paranoia.

3.2. Methods

3.2.1. Design and procedure

Study 2 examined whether the effect of loneliness on subclinical paranoia, observed in Study 1, can be reduced by targeting a mediator of the effect – negative affect. The study was a mixed-design with a 3-level between-subjects factor, loneliness (high loneliness, high loneliness with cognitive reappraisal, no-treatment control) and a 2-level within-subjects factor, paranoia (pre-manipulation paranoia, post-manipulation paranoia). Participants' pre-manipulation state paranoia was first assessed, then participants in the high loneliness condition recalled a past lonely experience, participants in the high loneliness with cognitive reappraisal recalled a past lonely experience and then attempted to cognitively reappraise this lonely experience to alter its emotional impact, and participants in the no-treatment control completed a word-search task. Thereafter, we measured participants' negative affect and then again, participants' state paranoia.

3.2.2. Sample selection and recruitment

The sample selection and recruitment was as in Study 1.

3.2.3. Participants

The sample-size estimation was as in Study 1 (i.e., approximately 60 participants per condition). We recruited 197 (100 female) adults. One participant was excluded for failing an attention check item, resulting in a final sample size of 196 participants (mean age of 36 years, $M = 36.24$, $SD = 12.16$).

3.2.4. Materials

The materials of Study 2 were identical to Study 1 except for that (1) we adapted the scale of the paranoia measure to a 0–100 scale to increase the sensitivity of the measure, and (2) the low loneliness condition was replaced by the high loneliness with cognitive reappraisal condition. This second change is elaborated on below.

3.2.5. Loneliness manipulation

The study included three conditions: High loneliness, high loneliness with cognitive reappraisal, and a no-treatment control. The high loneliness and no-treatment control conditions were as in Study 1. Participants in the high loneliness with cognitive reappraisal condition first completed the procedure of the loneliness condition and then cognitively reappraised the recollected lonely experience to lessen its negative emotional impact. Specifically, participants were made aware of their potential negative affect, and then were told to consider the lonely experience in a less 'negative' light: "Please read carefully. You may be feeling shame and embarrassment after re-living your lonely experience. Try to think about the lonely situation in a less negative light in an effort to reduce your feelings of shame and embarrassment. You can achieve this in several different ways. For example, think about

how you learned from the lonely experience and how on the long run it has made you experience less shame in your life (i.e., the experience has made you stronger, more confident, etc.). Please write bullet points about how you overcame the feelings of shame and embarrassment associated with the lonely experience and how you now feel differently."

3.3. Results

3.3.1. Paranoia scores

We calculated pre- and post-manipulation paranoia, $M = 1.07$, $SD = 0.60$, $\alpha = 0.87$, and $M = 0.95$, $SD = 0.67$, $\alpha = 0.90$.

3.3.2. Manipulation check

Participants in the high loneliness condition reported feeling more lonely, $M = 5.16$, $SD = 3.03$, than participants in the no-treatment control, $M = 2.37$, $SD = 2.04$, and the high loneliness with cognitive reappraisal, $M = 2.95$, $SD = 2.53$, conditions, $t(130) = 6.27$, $p < 0.001$, $d = 1.092$ and $t(126) = 4.87$, $p < 0.001$, $d = 0.848$, respectively. The no-treatment control and high loneliness with cognitive reappraisal conditions did not significantly differ, $t(130) = 1.31$, $p = 0.190$, $d = 0.228$.

3.3.3. Loneliness influences paranoia

As in Study 1, we observed a difference in post-manipulation paranoia depending on condition, $F(3,192) = 4.13$, $p = 0.018$, $d = 0.414$. Participants in the high loneliness condition, $M = 1.05$, $SD = 0.38$, exhibited higher levels of paranoia than those in the no-treatment control condition, $M = 0.87$, $SD = 0.37$, $t(130) = 2.85$, $p = 0.005$, $d = 0.496$ (see Fig. 1). Participants in the high loneliness condition exhibited marginally higher levels of paranoia, $M = 1.05$, $SD = 0.38$, than those in the high loneliness with cognitive reappraisal condition, $M = 0.94$, $SD = 0.37$, $t(126) = 1.77$, $p = 0.076$, $d = 0.313$. Importantly, however, no differences in paranoia were observed when comparing participants in the high loneliness with cognitive reappraisal condition and the control condition, $M = 0.87$, $SD = 0.37$, $t(130) = 1.05$, $p = 0.297$, $d = 0.183$ (see Fig. 2).

3.3.4. Loneliness leads to paranoia via negative affect

We investigated whether negative affect mediated the relationship between loneliness and paranoia by conducting the same mediation analysis as in Study 1, except that this analysis included all three conditions (high loneliness, high loneliness with cognitive reappraisal, no-treatment control). We dummy coded these conditions as follows (no-treatment control condition was the reference category): D1: no-treatment control = 0, high loneliness = 1, high loneliness with cognitive reappraisal = 0, D2: no-treatment control = 0, high loneliness = 0, high loneliness with cognitive reappraisal = 1.

The omnibus effect of loneliness on paranoia was fully mediated by negative affect, $M = 3.01$, $SD = 1.80$ (Table 3). Concerning the relative
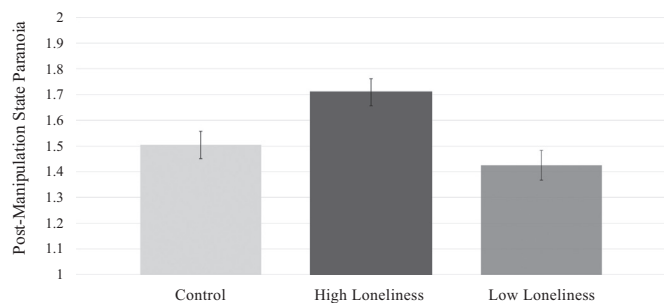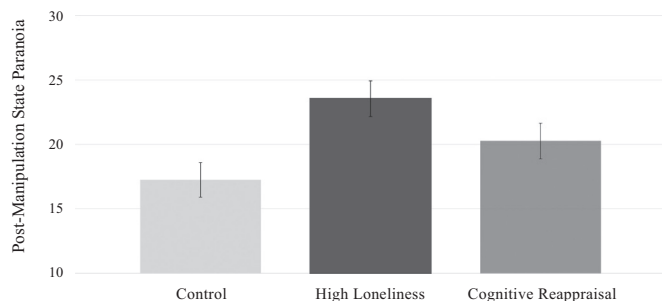

Fig. 1. Post-manipulation paranoia as a function of condition when controlling for pre-manipulation paranoia in Study 1. Error bars represent ± 1 *SE*. Five-point Likert scale: (1 = *Do not believe*, 2 = *Believe a little*, 3 = *Believe it somewhat*, 4 = *Believe it a lot*, 5 = *Absolutely believe it*).

Fig. 2. Post-manipulation paranoia as a function of condition when controlling for pre-manipulation paranoia in Study 2. Error bars represent ± 1 *SE*. One to 100 slider scale with anchors: (10 = *Do not believe*, 30 = *Believe a little*, 50 = *Believe it somewhat*, 70 = *Believe it a lot*, 90 = *Absolutely believe it*).

Table 3
Omnibus mediation effects in Study 2. The effect of loneliness condition (high loneliness, high loneliness with cognitive reappraisal, no-treatment control) on paranoia was mediated by negative affect.

Omnibus Effect – All three conditions	
Total Effect	$R^2 = 0.013$, $F(2,192) = 4.13$, $p = .018$
Direct Effect	$R^2 = 0.001$, $F(2,191) = 0.46$, $p = .630$
Indirect Effect	$b = 0.025$, $SE = 0.011$, 95% CI: [0.008, 0.052]

Note. b = unstandardized estimate. SE = Standard Error. CI = Confidence Interval.

effect of the high loneliness condition versus the no-treatment control condition, we replicated the findings of Study 1. That is, while increasing loneliness overall increased subclinical paranoia compared to the no-treatment control condition (total effect), $b = 0.27$, $p = 0.005$, this effect was no longer significant when accounting for participants' negative affect (direct effect), $b = 0.08$, $p = 0.394$. The indirect effect was significant, $b = 0.19$, $SE = .05$, 95% CI: [0.107, 0.313]. These results indicate a complete mediation (Table 2).

Importantly, in line with a successful intervention at the level of the mediator, a mediation of negative affect was not observed when comparing the high loneliness with cognitive reappraisal condition with the no-treatment control condition, indirect effect: $b = 0.031$, $SE = 0.037$, 95% CI: [− 0.038, 0.106]. Further negating the possibility of a mediation, participants' negative affect in the high loneliness with cognitive reappraisal and the no-treatment control condition did not differ, $b = 0.114$, $SE = 0.15$, $t(126) = 0.77$, $p = 0.445$, 95% CI: [− 0.180, 0.409].

3.4. Discussion

Study 2 found that the effect of loneliness on subclinical paranoia can be effectively intervened upon at the level of the observed mediator – negative affect. Participants who engaged in cognitive reappraisal after recollecting an experience of loneliness did not exhibit increased subclinical paranoia compared to a no-treatment control condition; participants who solely recollected an experience of loneliness (i.e., did not engage in cognitive reappraisal) on the other hand, did exhibit increased subclinical paranoia compared to a no-treatment control condition.

4. General discussion

In two studies we examined a potential causal link between loneliness and paranoia in the non-clinical population. Both studies replicated the past findings of Lamster et al. (2017) that increasing loneliness heightens subclinical paranoia. We extended these past findings by exploring the complexities of this causal relationship (i.e., mechanisms) and by examining potential intervention strategies. Regarding mechanisms, Studies 1 and 2 found the effect of loneliness on

increasing paranoia to be mediated by negative affect (e.g., sad mood). Regarding intervention possibilities, we examined two interventionist pathways identified by the interventionist-causal model (Kendler and Campbell, 2009) – researchers have recently called for applying an interventionist-causal model to specifically paranoia (e.g., Bell and Freeman, 2014; Freeman, 2011). Regarding the first interventionist pathway – decreasing loneliness – Study 1 found that recollecting an experience of companionship did not decrease state subclinical paranoia. These findings are in contrast to Lamster et al. (2017) who found an effect of decreasing loneliness on decreasing subclinical paranoia. We hypothesized that we observed this null finding because of a statistical floor effect – the paranoia of individuals already low in paranoia at baseline cannot greatly decrease. Supporting this possibility, exploratory, post-hoc analyses indicated that decreasing loneliness did decrease paranoia, but only in individuals high in paranoia at baseline. Regarding the second interventionist pathway – intervention at the mediator – Study 2 found that cognitive reappraisal can be utilized by individuals to reduce the effects of loneliness on increasing paranoia by reducing the negative affect associated with loneliness. Participants who recalled an experience of loneliness, but then cognitive reappraised that lonely experience, exhibited no more paranoia than participants in a no-treatment control condition.

The effect of loneliness on paranoia in the presented studies was not trivial; participants in Studies 1 and 2 who recollected a lonely experience (high loneliness condition) exhibited 20% more subclinical paranoia than participants in a no-treatment control condition. To combat these effects, in concordance with the interventionist-causal model, we examined the possibility of decreasing paranoia via a direct causal pathway (by reducing loneliness), and a mediation pathway (intervening on the affective pathway to paranoia). Regarding the direct causal pathway, we were surprised to find that recollecting an experience of companionship reduced paranoia by only around 15%. Regarding the mediation pathway, however, we found that using cognitive reappraisal to reduce the negative affect that loneliness induces attenuated the effect of loneliness on paranoia by around 60%.

Taken together, the current findings provide two avenues for reducing and preventing paranoid ideation – recollecting moments of companionship and utilizing cognitive reappraisal, respectively. We posit that these two interventionist pathways serve different psychological purposes. While companionship, or feeling loved, may *decrease* paranoia, cognitive reappraisal may help *prevent* the effect of social risk factors, such as loneliness, on paranoia. Future research should consider these possibilities to refine current evidence-based interventions for paranoia.

4.1. Theoretical relevance

Our findings in Studies 1 and 2 that loneliness increases subclinical paranoia are in line with past research indicating that social factors related to loneliness also lead to subclinical paranoia. For example, experiences of social exclusion, induced via a Cyberball paradigm (Williams et al., 2000), are shown to increase subclinical paranoia symptoms in healthy individuals (Kesting et al., 2013). Experiencing environmental stressors (e.g., population density, hostility) in a virtual reality paradigm also increase paranoia. This effect was stronger for individuals at high risk for psychosis (Veling et al., 2016). Collectively, such findings have resulted in recent theoretical papers arguing that social risk factors *causally* contribute to psychosis and its symptoms (e.g. Kesting and Lincoln, 2013; Selten et al., 2016).

Our findings also support the existence of an affective pathway to paranoia (e.g. Fowler et al., 2011; Jaya et al., 2017; Kesting and Lincoln, 2013; Kramer et al., 2014) – we found negative affect to mediate the effect of loneliness on paranoia. Further, we demonstrated that an emotion-regulation technique, cognitive reappraisal, can effectively protect against subclinical paranoia by targeting this mediation. This result is in line with findings that emotional regulation

strategies play a role in paranoia. For example, longitudinally, emotion regulation strategies mediate the effect of childhood trauma on paranoia (Lincoln et al., 2017).

4.2. Limitations

We consider a number of limitations of the presented research. First, the use of a subclinical sample restricts the findings' transferability to clinical samples and clinical conditions. For example, whether our findings regarding subclinical paranoia contribute to the understanding of psychosis is questionable. However, we do note that our results emulate the overall observed relationship between loneliness and paranoia found in a clinical sample (Sündermann et al., 2014). Further, a study assessing twins suggested that the genetic and environmental factors influencing paranoia are consistent across clinical severity (Zavos et al., 2014).

A second limitation is that our dependent variables (i.e., negative affect, paranoia) were measured in a state manner. Thus, we cannot conclude that our results carry over to chronic loneliness, depression, and chronic paranoia. However, this seems possible given that our results are in line with the studies presented in Jaya et al. (2016), which measured loneliness, depression and paranoia in a trait manner. Third, the current studies examined an artificially and experimentally induced imagination of loneliness. That is, the utilized loneliness manipulations may not have incited loneliness in the exact form that it is experienced in the 'real world.' One implication of this artificial manipulation is that it may have directly induced frustration or negative affect instead of loneliness. This is unlikely, however, given that our manipulation check analyses indicated that the high loneliness manipulation clearly incited increased state loneliness, and that these effects were larger than the effects of the manipulation on increasing negative affect.

A further limitation of the current research is that the causal mediation via negative affect should be approached with some caution. Specifically, our loneliness manipulation may have directly induced negative affect rather than inducing negative affect via increasing loneliness. Although possible, two reasons counter this claim. Firstly, condition still predicted loneliness (on the lonely manipulation check item) when controlling for negative affect but did not predict negative affect when controlling for loneliness. This finding indicates that participants' increased negative affect in the high loneliness condition (compared to the no-treatment control condition) likely arose as a result of the manipulation inciting loneliness as intended. Second, according to past research examining causal mechanisms in line with the interventionist-causal model approach (Reininghaus et al., 2015), *causal* evidence of a mechanism can be gathered by successfully intervening on the mechanism with an intervention strategy that is known to be able to change said mechanism. In Study 2 we observed just that; cognitive reappraisal targeting the proposed mechanism, negative affect, successfully reduced participants' negative affect and subclinical paranoia. Despite these points, however, the existence of a causal mediation via negative affect should still be approached with caution.

Finally, we note a limitation regarding the utilized cognitive reappraisal instructions in Study 2. Participants were told that they may be feeling shame and embarrassment after re-living the lonely experience, and that they should try to think of the lonely experience in a less negative light. Given that the instructions in our study specifically focused on shame and embarrassment it is unclear whether our findings would generalize to other negative emotions or more broad cognitive reappraisal instructions (e.g., overall bad mood).

5. Conclusion

Our results confirm the causal relationship between loneliness and paranoia, and indicate that this relationship is mediated by negative affect. We intervened on this observed effect by adopting two interventionist pathways in line with an interventionist-causal model

approach. Our results support that targeting loneliness itself (i.e., reducing loneliness) as well as targeting a mediating process (i.e., negative affect) may be effective strategies for decreasing paranoia. An important caveat for the former intervention strategy, however, was that we solely found reducing loneliness to reduce paranoia in individuals high in paranoia at baseline. Overall, though, our findings indicate that intervening on causal social risk factors of paranoia may be a promising route for reducing and preventing subclinical paranoia.

Acknowledgements

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.psychres.2018.02.050.

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