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ORIGINAL PAPER

Struggling with Happiness: A Pathway Leading Depression to Gambling Disorder

Guyonne Rogier[1] · Giancarlo Picci[1] · Patrizia Velotti[1]

Abstract

A number of studies have suggested that depressive mood might lead to the development and/or maintenance of a gambling disorder (GD). The pathways by which such relationships are fostered may involve deficits in emotional regulation capacity and dysfunctional coping styles. This study aims to explore the role played by depressive symptomatology and the regulation of positive emotion in GD. We administered the South Oaks Gambling Inventory (SOGS, Lesieur and Blume in Am J Psychiatry 144(9):1184–1188, 1987), the 21-item Depression Anxiety Stress Scale (DASS-21, Lovibond and Lovibond in Manual for the depression anxiety stress scales. Psychology Foundation, Sydney, 1995) and the Kill-joy Thinking subscale of the Ways of Savouring Checklist (WOSC, Bryant and Veroff in Savoring: a new model of positive experience. Lawrence Erlbaum, Mahwah, 2007) to a sample of pathological gamblers (n = 91) and a sample of community participants (n = 105). The pathological gamblers scored higher on the DASS-21 subscales and obtained higher scores on the Kill-joy Thinking subscale of the WOSC compared to the controls. Moreover, the SOGS scores positively correlate with the DASS-21 subscales, and with the Kill-Joy Thinking measure. Finally, it is evident that Kill-joy Thinking fully mediates the relationship between depressive symptomatology and GD severity. Our results further confirm the roles of depression, anxiety and stress in GD. Moreover, this is the first study to explore the mediating role of dampening processes in the relationship between depression and GD. Future lines of research are also discussed.

Keywords Gambling disorder · Depression · Anxiety · Emotion regulation · Savoring · Dampening

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Introduction

Gambling Disorder and Depression

Gambling disorder (GD) is characterised by a maladaptive and persistent gambling behaviour, which can lead to clinically significant discomfort or impairment (American Psychiatric Association 2013). Moreover, GD is characterized by various psychiatric comorbidities (el-Guebaly et al. 2006; Kim et al. 2006; Lorains et al. 2011; Rogier and Velotti 2018a; Rogier et al. 2017).

In particular, a systematic review of the extant literature (Lorains et al. 2011) indicates that 38% of pathological gamblers (PGs) suffer from a comorbid mood disorder. Since comorbid depression in PGs increases the risk of suicide (Blaszczynski and Farrell 1998; Petry and Kiluk 2002), it is necessary to address this issue in treatment protocols. The causal relationship between depression and GD is still controversial, with some studies indicating that depression symptomatology is a risk factor for GD (Blaszczynski and Farrell 1998; Chou and Afifi 2011; Parhami et al. 2014), while others conclude that GD should be considered a facilitator of depression (Afifi et al. 2016), and still others argue that there are mutual direct links between the two disorders (Dussault et al. 2011).

Laboratory studies have examined the role of depressive mood in gambling behaviour. For instance, Griffiths (1995) has investigated the moods of 60 individuals (habitual gamblers and non-habitual gamblers) while playing with a fruit machine. Habitual and pathological gamblers reported high levels of depressive mood before the gambling task, while non-habitual gamblers did not. A very recent study used a mood induction paradigm to estimate the causal influence of sadness in a slot machine task, providing a monetary reinforcement to recreational gamblers (Devos et al. 2018). In the experimental group (sadness induction), the participants exhibited more persistent gambling behaviour compared to participants assigned to the control condition (no emotional induction).

Despite these preliminary evidences, the way in which depressive symptomatology leads to GD remains partially unexplained. The emotional regulation framework provides a useful tool to approach this issue. Several authors have suggested that unsuccessful emotional regulation processes may be implicated in both the development and maintenance of gambling activity (Blaszczynski and Nower 2002; Lesieur 2001; Rogier and Velotti 2018b; Sharpe 2002). Indeed, all of these models assert that some PGs gamble to "escape" depressive moods. For instance, Lesieur (2001) has labelled this subtype of PGs as "escape-seekers", whereas Blaszczynski and Nower (2002) identify them as the "emotionally-vulnerable" subgroup. In accordance with this, the nosographical definition of the disorder itself encompasses a criterion referring to the use of gambling behaviour as a regulator of dysphoric affect (APA 2013). The hypothesis that depression leads to GD due difficulties with emotion regulation, is supported by studies that indicate high levels of emotion dysregulation in PGs (e.g. Williams et al. 2012; Navas et al. 2018; for a narrative review see Rogier and Velotti 2018b), as well as research that has proven a mediating role of emotion dysregulation in the relationship between psychopathology and GD (Jauregui et al. 2016; Rogier and Velotti 2018a).

However, the concept of emotional dysfunction, in both GD and depression, has not been examined exhaustively. In particular, as already noted in the broadest field of psychopathology (Carl et al. 2013), the scientific community has primarily neglected the role of positive emotion regulation in both GD and depressive symptomatology.

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Hedonic Dysregulation in Depression: the Role of Kill-joy Thinking

A core symptom of depression is anhedonia, which is the inability to experience positive emotional states (APA 2013). Studies have found that depressed individuals exhibit low levels of trait positive affect (i.e. how much people experience positive affect) and that blunted neural responses to gambling rewards (i.e. reward insensitivity) is a factor in depression (Watson et al. 1988; Weinberg et al. 2015). Additionally, a recent meta-analysis has further found that depression is associated with diminished reactivity to positive emotional stimuli (Bylsma et al. 2008).

The healthy regulation of positive emotions is a wide construct that includes the capacity to *savour* (Bryant and Veroff 2007). This concept refers to a range of cognitive and behavioural strategies used to upregulate the emotional states connected to positive experiences. In accordance with the literature on the role played by the regulation of positive emotions in psychopathology (Carl et al. 2013), studies have shown that *savouring* is related to low levels of depression (Smith and Hollinger-Smith 2015) and help-seeking behaviours in depressed individuals (Straszewski and Siegel 2018). More specifically, Bryant and Veroff (2007) have identified a dysfunctional strategy of *savouring* known as Kill-joy Thinking, a dampening cognitive process that down-regulates rather than increases positive emotions. The set of dampening processes elicited by a positive emotional trigger includes reactions as such as feeling guilty, thinking of ways in which the positive events could have been better, or reminding oneself about things one should be doing or responsibilities that one must still face. Preliminary results indicate that dampening processes are related to a negative mood after experiencing a success (Wood et al. 2003) and to depressive symptoms (Feldman et al. 2008; Raes et al. 2012). Despite the fact that Kill-joy Thinking and depression appear closely related, evidence supports the idea that these are two separate constructs. Several studies have revealed that, when controlling for depression levels, dampening processes are associated with panic disorder, social phobia, generalized anxiety disorder and obsessive-compulsive disorder (Carl et al. 2013; Eisner et al. 2009). In accordance with this, experimental evidence suggests that blunted responses to positive stimuli is not an exclusive characteristic of depressed individuals. For instance, using a cue-exposure paradigm, Larson et al. (2007) have observed that individuals with anxious symptomatology do not exhibit blink attenuation during and following the presentation of enjoyable stimuli. Thus, as suggested by Eisner et al. (2009), the role of dampening in psychopathology should not be reduced to a hallmark of depression, but may play other functions, such as the reduction of positive arousal experienced as disturbing in individuals with a panic disorder.

Hedonic Dysregulation in GD

Similarly, in the field of addiction, the role of positive emotions remains on the sidelines of empirical investigations (Carroll asnd Huxley 1994; Rogier and Velotti 2018b). This is rather surprising considering that several theoretical models have argued that the management of positive emotions is involved in the disorder (e.g. Jacobs 1986; McDougall 2004; McConaghy et al. 1988). Promising preliminary data suggests that the difficulty to cope with positive emotional states is central to GD. The most convincing results reveal an association between positive urgency—a personality trait describing the proneness to act rashly under the influence of positive emotional states—and GD

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(Haw 2017; Steward et al. 2017; Blain et al. 2015; Cyders and Smith 2008; Cyders et al. 2007).

An interesting line of research examined the specific nature of pleasurable experiences in individuals suffering from addiction. These contributions theorized the existence of a hedonic dysfunction in addicted individuals that would explain an excessive approach to hedonic stimuli. One of these theories, known as the incentive-sensitization theory of Berridge and Robinson (2008), asserts that addicted individuals, due to the excessive and repeated consumption of highly rewarding stimuli (such as gambling), have developed an unbalanced hedonic state. This would be expressed throughout an asymmetric hedonic sensitivity with elevated responses to addictive rewards and complementary blunted hedonic responses to other sources of pleasure, such as natural rewards. In accordance with this, a recent mindfulness-inspired treatment for addiction has the central objective of restoring hedonic function through training the savouring capacities (Garland 2016). Similarly, the reward deficiency syndrome theory (Volkow et al. 2002; Comings and Blum 2000) postulates the existence of a chronically impaired reward system, likely due to a hypodopaminergic state of subcortical areas. From this perspective, PGs would be driven to compensate for this impairment through involvement in activities providing high hedonic rewards. Unfortunately, the evidence presented by the neuroimaging field are inconclusive, with several studies reporting increased (Joutsa et al. 2012) reactivity of the reward system among PGs, while other report a decreased reactivity (Balodis et al. 2012; Chase and Clark 2010; de Ruiter et al. 2009; Reuter et al. 2005). An interesting and related study was conducted by Sescousse et al. (2013), who observed that PGs exhibit a decreased reactivity (measured throughout the activity of the ventral striatum) to erotic stimuli (i.e. a natural reward). Importantly, it has been recently argued that these two main theories should not necessarily be considered self-exclusive, but that they may be conceptualized as two complementary explanations of the development of GD. As a whole, both converge towards the idea that levels of hedonic dysfunctionalities (e.g. dampening processes, Kill-joy Thinking) should be associated with levels of GD severity.

The Present Study

Despite these interesting premises, to date there remains a lack of research examining the complex interplay between depression, Kill-joy Thinking and GD. As such, we aim to bridge this gap by investigating the topic among a clinical sample of PGs and comparing the results to a group of community participants. Specifically, we formulated the following hypotheses:

H1 We expect to find high levels of both internalizing symptomatology (i.e. depression, anxiety and stress) and down-regulation strategy of positive emotions (i.e. Kill-joy Thinking) among PGs, compared to community participants. This hypothesis was formulated on the basis of previous literature that has suggested high levels of depressive symptomatology among PGs, as well as based on neurobiological and theoretical literature that has discovered abnormalities in the hedonic response of addicted individuals.

H2 In accordance with the findings of previous studies, we expect to observe a significant and positive predictive effect of depressive symptomatology on severity of GD. Moreover, we predict the same pattern of results for Kill-joy Thinking, because the primary

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theoretical models assert that the severity of hedonic dysregulation accounts for the propensity to become addicted to gambling rewards.

H3 In accordance with previous empirical evidence and, in line with the cognitive conceptualization of depression (Beck 1976), we argue that depressive symptomatology orientates cognitive processes. We, thus, expect depressive symptomatology to predict the levels of Kill-joy Thinking.

H4 Finally, as Kill-joy Thinking is strongly associated with depression, and is a hypothesized variable of GD, it is reasonable to predict that the pathway by which depression leads to GD would be partially mediated by an individual's level of Kill-joy Thinking.

Methods

Participants and Procedure

This study was conducted on 196 Italian adults. The clinical group (n = 91) comprised participants (77 males) with a clinician-based current diagnosis of GD, with a mean age of 47.4 years (SD = 13.11), who were recruited from three clinical centres specialized in the treatment of GD. The comparison group (n = 105), with a mean age of 46.88 years (SD = 10.01), included community participants (79 males) who were drawn from the general population using a purposive sampling technique.

Information about the research's objectives and procedure were provided, and the participants' privacy and anonymity were ensured through the signing of a written consent form. The participants filled out self-reported questionnaires in an individual context (private rooms inside clinical centres or at the University of Rome), under the supervision of a clinical psychologist. All procedures complied with the guidelines of the American Psychological Association, and were approved by the Research Ethic Board of the University of Rome.

Measures

A self-report questionnaire was administered to all participants to gather information on the following areas:

Demographic information such as age, gender and nationality.

The severity of the participant's gambling disorder (or lack thereof) was evaluated using the 20-item South Oaks Gambling Screen (SOGS, Lesieur and Blume 1987; Guerreschi and Gander 2002). This instrument also provided cut-off scores to differentiate between participants who were not at risk, those who were and those with pathological gamblers. The reliability of this study was confirmed through the use of a Cronbach's Alpha test (score of 0.94).

Kill-joy Thinking was measured through the use of the Ways of Savouring Checklist (WOSC, Bryant and Veroff 2007; Balzarotti et al. 2018), a self-report questionnaire that assesses capacities to savour positive emotions. This questionnaire asks the participants to describe how they typically react to positive events, using a Likert-type scale ranging from 1 (*Definitely doesn't apply*) to 7 (*Definitely applies*). The participants completed

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all of the items of the WOSC, but only the Kill-Joy Thinking subscale was analysed. Scores for this subscale were obtained by adding the responses given by the participant to 7 of the total 60 items. Examples items include: "I thought about ways in which it could have been better", "I thought about things that made me feel guilty" or "I thought about other things that were hanging over me, problems and worries that I still had to face." This tenth subscale of the WOSC exhibits good internal consistency, with a Cronbach's alpha of 0.84.

Depression, Anxiety and Stress were measured through the use of the Depression Anxiety Stress Scales-21 items (DASS-21, Lovibond and Lovibond 1995; Bottesi et al. 2015), a self-report questionnaire that asks participants to indicate how often the described experience applies to them using a 4-point Likert scale, ranging from 0 (*Never*) to 3 (*Almost ever*). The instrument's excellent psychometric proprieties were confirmed with Cronbach's alphas of 0.88, 0.84 and 0.90 for *Depression*, *Anxiety* and *Stress,* respectively.

Statistical Analyses

Cronbach's alphas were calculated for each instrument to examine the reliability of the measures. A *t* test was performed for each to ensure that the control and clinical groups did not differ in age ($p = 0.14$) or on the basis of gender ($p = .07$). Means and standard deviations were calculated for each variable in the study. The relationships between all of the variables considered in the study were examined by calculating r-Pearson correlations. Subsequently, a *t* test was performed to explore the differences between groups on the DASS-21 and Kill-joy Thinking measures. Finally, the mediating effect of Kill-joy Thinking on the relationship between depression and the severity of GD was examined through a series of regression analyses, in accordance with Baron and Kenny (1986). In particular, we examined whether (1) depression effectively predicts the severity of GD; (2) depression significantly predicted Kill-joy Thinking; (3) Kill-joy Thinking predicts GD severity; (4) depression indirectly predicts the severity of GD through Kill-joy Thinking. Statistical significances were tested using the bootstrap method. All statistical analyses were implemented using SPSS 23.0 software for Windows. AQ1

Table 1 *T* test comparing groups on Kill-joy thinking, depression, anxiety and stress

	Addicted Gamblers (n = 91)		Control group (n = 105)		*t*	*p*
	Mean	SD	Mean	SD		
Kill-joy thinking	23.08	8.81	15.80	6.31	6.15	< .001
DASS-21 depression	5.35	3.84	3.25	2.97	3.99	< .001
DASS-21 anxiety	4.01	3.28	2.74	2.80	2.77	.006
DASS-21 stress	7.12	4.49	5.71	3.46	2.33	.021

SD standard deviation, *DASS* depression anxiety stress scales-21

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Results

Differences Between the Groups

The scores of the two groups were compared with regard to the Kill-joy Thinking and DASS-21 measures through the use of a *t* test. As displayed in Table 1, the results indicate that the means of the two groups differed significantly. In particular, the clinical group scored higher than the control group on the Kill-joy Thinking, depression, anxiety and stress measures.

Relationships Between GD Severity, Kill-joy Thinking, Depression, Anxiety and Stress

The *r*-Pearson correlations between all the study variables are illustrated in Table 2. Thus, it can be seen that GD severity is positively and significantly correlated with Kill-joy Thinking. All subscales of the DASS-21 were positively and significantly correlated with GD severity. Moreover, Kill-joy Thinking was positively and significantly associated with the subscales of the DASS-21.

The Mediating Role of Kill-joy Thinking

The mediating role of Kill-joy Thinking has been explored through the relationship between depression and GD severity. As Table 3 illustrates, depression positively predicts the severity of GD (Step 1), as well as Kill-joy Thinking (Step 2). Moreover, Kill-joy Thinking positively predicts GD severity beyond the role of depression (Step 3), and depression indirectly predicts SOGS scores through Kill-joy Thinking (Step 4). The results indicate that the predictive role of depression in GD severity is entirely mediated by Kill-joy Thinking.

Discussion

This study aimed to explore the role of depressive symptomatology and the regulation of positive emotions in the lives of PGs. Furthermore, we wanted to test the mediating role of Kill-joy Thinking on relationship between depression and GD severity. The results widely support our hypotheses.

Table 2 Correlations between severity of GD, kill-joy thinking, depression, anxiety and stress

	SOGS	Kill-joy Thinking	DASS depression	DASS anxiety	DASS stress
SOGS	–				
Kill-joy Thinking	.41**	–			
DASS-21 depression	.25**	.51**	–		
DASS-21 anxiety	.19*	.51**	.72**	–	
DASS-21 stress	.18*	.37**	.75**	.69**	–

SOGS south oaks gambling screen, *DASS* depression anxiety stress scales-21; * $p < .05$; ** $p < .001$

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Table 3 Direct and indirect effects of Depression on GD severity through Kill-joy Thinking

	B	SE	Bootstrap confidence interval [95%]
Step 1 DASS-21 depression → SOGS $R^2 = .062$; $p = .001$			
Constant	3.29	.65	2.1091 to 4.5361
DASS-21 depression	.41	.121	.2051 to .6190
Step 2 DASS-21 Depression → Kill-joy thinking $R^2 = .260$; $p = < .001$			
Constant	13.95	.84	12.3614 to 15.7373
DASS-21 Depression	1.22	.16	.8654 to 1.5029
Step 3 Kill-joy Thinking → SOGS $R^2 = .172$; $p = < .001$			
Constant	− .52	1.02	− 2.2110 to 1.4224
WOSC10	.30	.05	.2043 to .3754
Step 4 DASS-21 Depression + Kill-joy Thinking → SOGS $R^2 = .174$; $p = < .001$			
Constant	− .54	1.03	− 2.5816 to 1.4947
Depression → SOGS	.12	.14	− .1581 to .3872
Depression → Kill-joy Thinking → SOGS	.32	.09	.1650 to .5172

First, PGs exhibited higher levels of depression, anxiety and stress than the non-clinical group. These levels are also positively correlated to GD severity, in accordance with previous literature which has found that GD is associated with depression (Chou and Afifi 2011; Lorains et al. 2011; Parhami et al. 2014), anxiety disorders (Giddens et al. 2012; Kessler et al. 2008) and life stressors (Roberts et al. 2017).

Moreover, our data illuminates the association between Kill-joy Thinking and depressive symptomatology. This extends the current literature (Wood et al. 2003; Feldman et al. 2008; Raes et al. 2012) on the role of dysfunctional responses to positive events in depression, suggesting that people with depressive symptoms may tend to minimize or eliminate (Feldman et al. 2008) self-relevant positive emotions (i.e. through dampening). Specifically, Gruber et al. (2011) have asserted that depressed individuals struggle to regulate positive emotions related to self-relevant cues.

Subsequently, in our study, the PGs exhibited higher levels of Kill-joy Thinking than the community group, and these levels were found to be positively correlated with the severity of GD. These results extend the literature on the difficulties of managing positive emotions for PGs, and further indicate a new path of research towards the role of savouring in addictions. Indeed, theories that argue in support of a deficit of hedonic regulation in traditional addictions seem to be successfully extended to the topic of GD. Our psychological evidence converges on the neurobiological data of Sescousse et al. (2015), indicating a deficit in hedonic responses to erotic stimuli among PGs. The reward deficiency syndrome theory asserts that individuals with impaired hedonic capacities are more prone to becoming

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addicted to highly rewarding activities. Thus, and in accordance with our results, the levels of hedonic impairments appear to be a relevant predictor of GD severity.

Beyond this interpretation of our results, it can be speculated that Kill-joy Thinking fosters GD severity due to an increased persistence in gambling behaviour. For instance, Kill-joy Thinking refers to a propensity to think of ways in which positive events could have been better. In this regard, a dampening response to a reward may enhance persistent gambling behaviours, even after a win, which sustains the craving for the next success. Moreover, Kill-joy Thinking refers to the tendency to remind oneself about the things one should be doing, such as one's responsibilities. In a gambling context, this may result in increased levels of charges-related concerns, which can interfere with the capacity to savour the current experience, favouring instead a focus on the next gamble to escape from uncomfortable emotional states. It is worth noting that these are only speculations, and should be considered as hypotheses to test in future research that investigates the relationship between Kill-joy Thinking and gambling involvement.

Finally, our results support the hypothesis that depression leads to GD through the mediating role of Kill-joy Thinking. The relationship between comorbid depression and GD has been traditionally understood from the perspective of the "gambling-as-an-escape" hypothesis. In other words, current literature mainly recommends that clinicians treat depressed PGs' inability to manage negative emotions to reduce the risk of gambling behaviour elicited by negative emotional triggers. Our study sheds light on another, likely complementary, mechanism that links depressive symptomatology and GD: difficulty enjoying positive experiences. The restoration of the hedonic capacity is a central aspect in traditional treatments for mood disorders (Beck 1976), and may be proficiently addressed in the treatment of comorbid GD and depression.

Importantly, it can be argued that Kill-joy Thinking is a stable trait that acts as a risk factor for depression which, in turn, predicts GD severity. While the predictive role of dampening processes on depression makes sense—and has been tested in other studies—the mediational model seems less convincing. Indeed, conceptually, dampening processes are considered a hallmark of cognitive depression (Beck 1976): maladaptive schemas are thought to orientate cognitive processes in a maladaptive way, which consequently perpetuates depressive symptomatology. However, our results indicated that depressive symptomatology, after controlling for Kill-joy Thinking levels, is not a significant predictor of GD severity.

The present study is insightful on both empirical and clinical levels. Indeed, this research provides preliminary results on the role of regulation of positive emotions in GD. Moreover, it stimulates future studies aiming to deepen the role of Kill-joy Thinking as a risk factor for the development of GD, as well as a maintenance mechanism of pathological gambling behaviour. Innovative treatments for GD, especially those that focus on comorbidity with depression, will benefit from our findings. For instance, techniques inspired by the treatment options proposed by Garland (2016), which aim to train addicted individuals to enjoy natural positive experiences, may be usefully translated within the context GD treatment.

Limitations

Although our study provides innovative insight, several limitations should be considered. The cross-sectional nature of our study does not allow us to draw irreversible conclusion on the causal relationship between depression, Kill-joy Thinking and GD. Therefore, a

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future longitudinal examination of this topic is needed to support our results. Subsequently, our sample was unbalanced in regard to gender, as there was a much higher proportion of male participants. As women have been shown to be especially prone to suffering from depressive symptoms (Salk et al. 2017), the role of gender should be considered. Finally, the cross-cultural research suggests that the use of dampening processes may vary across cultures (Miyamoto and Ma 2011). Thus, cultural factors may have affected our study, limiting its generalizability.

Future Directions

This study introduces a promising line of research, profiling future directions for empirical investigations into the role of positive emotions in addiction. First, future research should explore whether the observed relationships between GD severity, depression and Kill-joy Thinking vary across subtypes of gamblers. Moreover, future research should be examined the nature of positive emotions by distinguishing their processes of regulation and the complex interplay between the regulation processes of negative and positive emotions. For instance, the role of guilt—elicited by Kill-joy Thinking—can foster in reaction to specific self-relevant positive cues (e.g. positive triggers eliciting pride). Finally, the role of Kill-joy Thinking in GD should be investigated by exploring the relationships between the dysregulation of positive and negative emotional states. AQ2

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

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