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Are Trait Emotional Competencies and Heart Rate Variability Linked to Mental Health of Coronary Heart Disease Patients?

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Abstract

Purpose: Depression and anxiety have been extensively associated with adverse outcomes in coronary heart disease patients. However, psychological and physiological processes underlying the persistence of these troubles in coronary heart disease patients attending cardiac rehabilitation are poorly investigated. Trait emotional competencies and heart rate variability could be some of these processes. Thus, the aim of this study was to assess the predictive value of trait emotional competencies and heart rate variability on depression and anxiety symptoms persistence in coronary heart disease patients.

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Methods: Eighty-four patients who recently presented a myocardial infarction were evaluated at the beginning of cardiac rehabilitation. Forty-two patients continued their rehabilitation program and were then assessed three months later. They completed the Profile of Emotional Competence as well as the Hospital Anxiety and Depression Scale and underwent a 5-minute resting heart rate variability measure.
Results: Low trait emotional competencies score predicted depression symptoms persistence, but unexpectedly, high trait emotional competencies score was also associated with withdrawal from cardiac rehabilitation. Contrary to our expectations, heart rate variability did not predict depression or anxiety symptoms persistence and was not associated with trait emotional competencies.
Conclusions: This study is the first to report an association between trait emotional competencies and depression symptoms persistence in coronary heart disease patients. However, heart rate variability was not associated with either depression or anxiety supporting the idea of mixed literature and highlighting the need of future research.

Keywords
Heart rate variability, emotional competencies, depression, anxiety, coronary heart disease

Introduction

Depression and anxiety have been extensively associated with adverse outcomes in coronary heart disease (CHD) (Barth, Schumacher, & Herrmann-Lingen, 2004; Roest, Martens, Denollet, & De Jonge, 2010). Despite the importance of these associations, physiological and psychological processes implicated in the persistence of depression and anxiety are poorly investigated in this specific population. One of the psychological processes that is known for influencing depression and anxiety persistence is emotional competencies (EC) (Martins, Ramalho, & Morin, 2010). Indeed, it has been shown that depression and anxiety are closely related to EC (Mikolajczak, Luminet, & Menil, 2006; Petrides, Pérez-González, & Furnham, 2007), a global concept of emotional processes including identification, comprehension, expression, regulation, and utilization of one's and others emotions (Mayer & Salovey, 1997; Petrides & Furnham, 2003). We will focus specifically on trait EC deficit as it is more related to affective disorders than ability EC deficit (Goldenberg, Matheson, & Mantler, 2006) and also to more health issues, greater drugs consumption, and specifically those who target the cardiovascular system (Mikolajczak et al., 2015). There are not many studies focusing on the relationships between EC and CHD, but one of them has shown that deficits in emotion identification and expression, two

components of EC, have been linked to an increased risk of all-cause mortality (Kauhanen, Kaplan, Cohen, Julkunen, & Salonen, 1996).

Another process that could explain the persistence of depression and anxiety in CHD patients is heart rate variability (HRV), defined as the time intervals between each heartbeat (Thayer & Lane, 2007). HRV has first been extensively studied in cardiac populations because it is an important and independent prognostic factor in CHD (Buccelletti et al., 2009). Furthermore, a decreased HRV has been often observed in both depression and anxiety disorders (Chalmers, Quintana, Abbott, & Kemp, 2014; Kemp et al., 2010) suggesting that HRV could be a biomarker of psychopathology (Beauchaine & Thayer, 2015). This hypothesis is mainly based on Thayer and Lane's (2000) neurovisceral integration model that explains how HRV reflects the autonomic nervous system function and the balance between its two components: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). Decreased HRV is thought to reflect depressed vagal modulation from the PNS associated with an increased SNS activation. HRV is indeed influenced by the central autonomic network (CAN), the stellate ganglia, and the vagus nerve. The CAN receives information from internal and sensory external environment (input) and sends output that directly influences heart rate. Thus, HRV is considered to be a reflection of the CAN output and more specifically, the index of the physiological responses when emotions arise (Thayer & Lane, 2000). A meta-analysis has recently confirmed that HRV is associated with top-down self-regulatory processes including executive functioning, emotion regulation, working memory, and attentional regulation (Holzman & Bridgett, 2017). All these processes are implicated in depression and anxiety (Ajilchi & Nejati, 2017) suggesting that HRV could partially explain the development and persistence of these disorders.

Emotion regulation thus appears to be only one subcomponent of the processes that are involved in HRV (Holzman & Bridgett, 2017). This is the reason why it has been suggested that trait EC, a wider and global concept of emotional management that encompasses emotion regulation, could be associated with HRV (Batselé, Stefaniak, & Fantini-Hauwel, 2019; Craig et al., 2009). If the mediation hypothesis of HRV on the relationship between emotional processes and health has been known for some years (Appelhans & Luecken, 2006), HRV has been recently proposed as a moderator of this relationship (Batselé et al., 2019; Ode, Hilmert, Zielke, & Robinson, 2010). These studies suggest that having high HRV could be a protective factor against deleterious effects of some personality traits on mental health (Batselé et al., 2019; Ode et al., 2010). This is not surprising considering that high HRV reflects a good balance between PNS and SNS allowing individuals to rapidly engage and disengage themselves in behaviors to adapt adequately to their environment (Thayer & Lane, 2000). One of the studies that only considered emotional processes and HRV together to predict prognostic in CHD patients has shown that low

emotional sensitivity and low HRV were predictive of cardiac death in a eight-year follow-up, although emotional sensitivity and HRV were not associated (Carpeggiani et al., 2005).

Thus, the purpose of this study is to investigate a possible link between trait EC and HRV in CHD patients, with respect to depression and anxiety symptoms persistence three months after the beginning of their cardiac rehabilitation. In accordance to the literature (Appelhans & Luecken, 2006; Batselé et al., 2019; Ode et al., 2010), two different models are tested. The first one suggests that low level of trait EC at baseline will predict persistence of depression and anxiety symptoms at follow-up, but that this relationship is moderated by HRV's level at baseline. The second one hypothesizes that low level of trait EC at baseline will predict depression and anxiety persistence at follow-up, but that this relationship is mediated by HRV's level at baseline.

Methods

Population

Eighty-four patients suffering from a myocardial infarction were recruited in a Belgian hospital (mean age = 61.94 ± 8.90, 17 females). On this initial pool of patients, 50% stopped their cardiac rehabilitation which is a major problem in Europe as only 33% to 56% adhere to the cardiac rehabilitation until the end of the program (Conraads et al., 2012). Thus, we have follow-up data for 42 patients (mean age = 61.38 ± 8.86, 8 females). Regarding education level, 37.8% of the sample reached university, while 45.1% completed high school, and 17.1% stopped their studies in primary school. Patients were included based on their medical diagnosis and drugs prescription. Only patients with medically diagnosed myocardial infarction and consuming beta-blockers were recruited. Exclusion criteria were consuming tricyclic antidepressant drugs as it impacts HRV (Kemp et al., 2010), presenting a severe psychiatric disorder (such as psychotic troubles), having difficulties in understanding and speaking French language, and demonstrating an apparent cognitive deficit that could impact questionnaires completion. Written informed consent was obtained from all patients. The hospital's ethic committee approved this study.

Procedure

Patients were recruited at the beginning of their rehabilitation program (between the first and third session = Time 1). Patients were systematically tested between 9:00 and 12:00 a.m. in a medical consultation local of the cardiac rehabilitation unit. The total duration of the study was approximately 45 minutes. Patients first fulfilled socio-demographic and psychological questionnaires and then

underwent a resting HRV measure. Three months after the inclusion, patients were tested again using the exact same procedure (= Time 2).

Instruments

Trait emotional competencies. The Profile of Emotional Competence (PEC) was used to assess trait EC (Brasseur, Grégoire, Bourdu, & Mikolajczak, 2013). This questionnaire comprises 50 items scored on a five-point Likert scale (from *strongly disagree* to *strongly agree*). It measures the five facets of trait EC: identification, understanding, expression, regulation, and the use of emotions, both one's own emotions (intrapersonal) and those of others (interpersonal). This leads to 10 subscores that are then computed into 3 main factor scores: intrapersonal EC, interpersonal EC, and the total EC factor score. The higher are the scores on this scale, the better are trait EC. Cronbach's alphas are displayed in Table 1.

Depression and anxiety. We used the French validated version of the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983), a 14-item scale (7 for anxiety and 7 for depression) scored on a four-point Likert scale ranging from 0 to 3. Cronbach's alphas are displayed in Table 1.

Heart rate variability. HRV was measured by using a Polar® V800 heart rate monitor allowing to extract HRV parameters (Giles, Draper, & Neil, 2016). Measurements were conducted during 6 minutes (spontaneous breathing and resting state) in order to extract 5 valid minutes, according to the recommendations of the Task Force (1996). The variability between successive R-spikes

Table 1. Descriptive statistics and Cronbach's alphas.

		Time 1	Time 2	t
Age	Mean (SE)	61.38 (8.86)	–	–
HRV	Mean (SE)	2.93 (0.80)	3.12 (0.91)	−1.58
Depression	Mean (SE)	4.48 (3.37)	4.19 (3.23)	0.80
	α	0.71	0.70	
Anxiety	Mean (SE)	7.50 (3.18)	7.45 (3.04)	0.13
	α	0.70	0.60	
EC intra	Mean (SE)	3.16 (0.47)	3.31 (0.42)	−2.47*
	α	0.75	0.73	
EC inter	Mean (SE)	3.19 (0.41)	3.21 (0.46)	−0.32
	α	0.70	0.71	
EC total	Mean (SE)	3.17 (0.39)	3.26 (0.38)	−1.55
	α	0.83	0.85	

EC: emotional competencies; HRV: heart rate variability; SE: standard error.
*$p \leq .05$.

was then analyzed using Kubios 3.0.2 premium version (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014). Among the HRV indicators, we chose to use the root mean square of the successive differences (RMSSD; ms^2), a time domain measure of HRV reflecting the PNS modulation of heart rate (Task Force, 1996). RMSSD is thought to be relatively free of respiratory influences and thus appear as a more reliable measure of HRV than others indices (Hill, Siebenbrock, Sollers, & Thayer, 2009; Task Force, 1996). RMSSD[1] has been natural log-transformed (ln) to fit assumptions of linear analyses (Ellis, Sollers, Edelstein, & Thayer, 2008).

Statistical analyses

All statistical analyses were computed using the software package SPSS version 25 (IBM Corp., 2017). To identify potential covariates to include in subsequent analyses, Pearson correlations between age and the dependent variables (depression and anxiety scores at Time 2) were computed. One-way analyses of variance were also used to test differences between genders on the dependent variables. Relationships between trait EC, depression and anxiety symptoms, and HRV overtime were examined using Pearson correlations. Logistic regression analyze was used to examine group differences in cardiac rehabilitation dropout. Cardiac rehabilitation dropout was coded as follows: 1 = patients who stopped the program and 0 = patients who continued the program. Five predictors were entered into the model: age, depression, anxiety, intrapersonal EC, and HRV. Robust moderation and mediation analyses were performed using the PROCESS macro (Hayes, 2017) with 10,000 bootstrap samples as follows: depression symptoms/anxiety symptoms at Time 2 as outcome variable, intrapersonal EC/interpersonal EC/total EC score at Time 1 as independent variable, HRV at Time 1 as mediator or moderator, and depression symptoms at Time 1 as covariable.

Results

Covariates analyses

Age did not correlate with either depression or anxiety at Time 2 ($r = -.07$, ns and $r = -.19$, ns, respectively). One-way analyses of variance did not reveal any significant differences on depression or anxiety at Time 2 depending on gender ($F(1, 41) = .44$, ns; $F(1, 41) = .48$, ns, respectively). Education level did not influence depression at Time 2 ($F(4,35) = .98$, ns) or with anxiety at Time 2 ($F(4,35) = 4.59$, ns). Therefore, these factors were not entered as covariates in subsequent analyses. The descriptive statistics and time course of changes in HRV levels, depression and anxiety symptoms, as well as EC scores are summarized in Table 1. Only intrapersonal EC significantly changed between Time 1 ($M = 3.16 \pm .47$) and Time 2 ($M = 3.31 \pm .42$) ($t = -2.47$, $p \leq .05$).

Logistic regressions analyze. The model was significant ($\chi^2(5) = 9.71$, $p \leq .05$) and explained 18% of the variance in cardiac rehabilitation dropout. It correctly classified 60.7% of cases. On the five predictors, only intrapersonal EC was associated with dropout (odds ratio (OR) = 5.86, $p \leq .01$). In other words, patients with high intrapersonal EC were more at risk to stop cardiac rehabilitation. A mean difference test for independent samples revealed that patients who stopped cardiac rehabilitation had a mean intrapersonal EC score of $3.46 \pm .43$ while those who continued had a lower mean score $3.16 \pm .47$ ($t = -3.13$, $p \leq .01$). Age was not linked to dropout (OR = 1.02, ns), nor depression (OR = 1.14, ns), anxiety (OR = .94, ns), or HRV (OR = .88, ns).

Correlation analyses. Table 2 shows the correlation matrix between HRV, depression and anxiety symptoms, and EC scores at all time points. At Time 1, depression and anxiety correlated with intrapersonal EC ($r = -.35$, $p \leq .01$; $r = -.39$, $p \leq .01$, respectively), interpersonal EC ($r = -.29$, $p \leq .01$; $r = -.38$, $p \leq .01$, respectively) and the total EC score ($r = -.35$, $p \leq .01$; $r = -.43$, $p \leq .01$, respectively). At Time 2, depression and anxiety also correlated with intrapersonal and the total EC score (but not with interpersonal EC). HRV was not associated significantly with either EC scores or depression and anxiety (see Table 2).

Moderation analyses. A main effect of intrapersonal EC on depression was found ($\beta = -.26$, $t = -2.40$, $p \leq .05$) but no interaction effect HRV × IntraEC was observed ($\beta = -.02$, $t = -.20$, ns) controlling for depression at Time 1 ($\beta = .64$, $t = 5.97$, $p \leq .01$). The overall model explained 63% of depression variance ($F(4, 37) = 15.62$, $p \leq .01$). Intrapersonal EC failed to predict anxiety ($\beta = -1.45$, $t = -1.84$, ns) and no moderation effect HRV × IntraEC was observed ($\beta = .44$, $t = .48$, ns) controlling for anxiety at Time 1 ($\beta = .60$, $t = 4.94$, $p \leq .01$). The overall model explained 57% of anxiety variance ($F(4, 37) = 12.45$, $p \leq .01$). No moderation effect HRV × InterEC or HRV × TotalEC was found to predict depression or anxiety (for detailed results, see Table 3).

Mediation analyses. A main effect of intrapersonal EC was observed to predict depression ($\beta = -.27$, $t = -2.45$, $p = \leq .05$), but no indirect effect was found for HRV ($\beta = .00$; 95% CI = [−.03 to .09]) controlling for depression at Time 1 ($\beta = .64$, $t = 6.08$ $p \leq .01$). The overall model explained 63% of depression variance ($F(3, 38) = 17.45$, $p \leq .01$). Intrapersonal EC failed to predict anxiety ($\beta = -.22$, $t = -1.81$, ns) and no mediation effect of HRV was found ($\beta = .01$, 95% CI = [−.02 to .14]) controlling for anxiety at Time 1 ($\beta = .73$, $t = 5.45$, $p \leq .01$). The overall model explained 57% of anxiety variance ($F(3, 38) = 18.25$, $p \leq .01$). No mediation effect of HRV was found for the relationships between depression or anxiety and interpersonal EC or the total EC score (data not shown).

Table 2. Correlation matrix.

Variables	1. HRV T1	2. DEP T1	3. ANX T1	4. Intra EC T1	5. Inter EC T1	6. Total EC T1	7. HRV T2	8. DEP T2	9. ANX T2	10. Intra EC T2	11. Inter EC T2	12. Total EC T2
2.	.04	–	–	–	–	–	–	–	–	–	–	–
3.	.05	.59**	–	–	–	–	–	–	–	–	–	–
4.	.04	−.35**	−.39**	–	–	–	–	–	–	–	–	–
5.	.00	−.29**	−.38**	.63**	–	–	–	–	–	–	–	–
6.	.02	−.35**	−.43**	.92**	.89**	–	–	–	–	–	–	–
7.	.58**	.01	.21	.04	−.15	−.06	–	–	–	–	–	–
8.	.11	.75**	.53**	−.53**	−.35*	−.50**	.08	–	–	–	–	–
9.	−.01	.46**	.73**	−.48**	−.19	−.39*	.03	.51**	–	–	–	–
10.	.06	−.34*	−.27	.59**	.26	.49**	.10	−.44**	−.44**	–	–	–
11.	.20	−.15	−.18	.27	.56**	.46**	.03	−.19	−.16	.52**	–	–
12.	.15	−.28	−.26	.49**	.47**	.55**	.08	−.35*	−.33*	.86**	.88**	–

EC: emotional competencies; HRV: heart rate variability; ANX: anxiety; DEP: depression.
*$p \leq .05$; **$p \leq .01$.

Table 3. Moderation analyses.

	Depression T2		Anxiety T2
	β (SE)		β (SE)
Depression T1	.64 (.11)**	Anxiety T1	.71 (.14)**
Intra EC T1	−.26 (.11)*	Intra EC T1	−.22 (.12)
HRV T1	−.04	HRV T1	−.10 (.11)
HRV T1 × Intra EC T1	−.02	HRV T1 × Intra EC T1	.06 (.13)
R^2	.63**	R^2	.57**
F	15.62**	*F*	12.45**
Depression T1	.68 (.11)**	Anxiety T1	.84 (.13)**
Inter EC T1	−.18 (.12)	Inter EC T1	.10 (.12)
HRV T1	−.03 (.11)	HRV T1	−.01 (.11)
HRV T1 × Inter EC T1	−.05 (.17)	HRV T1 × Inter EC T1	.38 (.17)
R^2	.59**	R^2	.59**
F	13.57**	*F*	13.39**
Depression T1	.64 (.11)**	Anxiety T1	.75 (.14)**
Total EC T1	−.26 (.11)*	Total EC T1	−.11 (.12)
HRV T1	−.04 (.11)	HRV T1	−.06 (.12)
HRV T1 × Total EC T1	−.07 (.14)	HRV T1 Total EC T1	.17 (.16)
R^2	.62**	R^2	.56**
F	15.40**	*F*	11.73**

β: standardized beta coefficient; SE: standard error; EC: emotional competencies; HRV: heart rate variability.
*$p < .05$; **$p \leq .01$.

Discussion

The aim of this study was to investigate a possible link between trait EC and HRV in CHD patients, with respect to depression and anxiety symptoms persistence three months after the beginning of their cardiac rehabilitation.

Firstly, intrapersonal trait EC predicted cardiac rehabilitation dropout, while age, depression, anxiety, and HRV did not. Indeed, higher intrapersonal trait EC was associated with more risks to stop cardiac rehabilitation. It may not seem logical as patients are informed that cardiac rehabilitation can improve their prognostic and higher trait EC is usually linked to more functional health behaviors (Fernández-Abascal & Martín-Díaz, 2015). But it is also possible that patients presenting high EC practice physical activities outside the cardiac rehabilitation, as it has been shown that people with better EC tend to do more physical activity (Saklofske, Austin, Rohr, & Andrews, 2007). However, a recent review has suggested that high EC may not always have adaptative consequences (Davis & Nichols, 2016). For example, higher EC has been found to amplify the effects of chronic stressors on depression, hopelessness, and suicidal ideation (Ciarrochi, Deane, & Anderson, 2002).

Secondly, no changes overtime were observed for HRV levels. After a myocardial infarction, patients usually exhibit a reduced HRV that is linked to adverse outcomes (Stein, Domitrovich, Huikuri, Kleiger, & Cast Investigators, 2005). Exercise improves vagal activity and reduces SNS overactivity (Delaney, Kelley, Sandercock, & Brodie, 2005). However, previous findings regarding the positive impact of cardiac rehabilitation on HRV are mixed. Indeed, some studies have demonstrated an improvement in HRV after three to six months of cardiac rehabilitation (Delaney et al., 2005; Malfatto et al., 1998) but others did not (Duru et al., 2000; Leitch et al., 1997). Our results show that depression and anxiety scores also did not change between Time 1 and Time 2. An improvement in depression and anxiety symptoms after cardiac rehabilitation has not always been observed (Sharif, Shoul, Janati, Kojuri, & Zare, 2012) even if some studies report such an improvement (Milani & Lavie, 1998; Yohannes, Doherty, Bundy, & Yalfani, 2010). One possible explanation is that the patients included in our study were not specifically treated for depression or anxiety (Ramamurthy, Trejo, & Faraone, 2013). Surprisingly, intrapersonal EC significantly improved over time. This is contradictory with the theoretical conceptualization of trait EC that is supposed to be a stable personality disposition (Mayer & Salovey, 1997; Petrides & Furnham, 2003). To the extent of our knowledge, patients involved in this study were not attending any therapeutic program that could have impacted intrapersonal EC. Our hypothesis is that patients who endured a myocardial infarction are maybe learning to be more attentive and aware about their emotional life. In this idea, it has been suggested that adaptation to myocardial infarction impact and modify patients coping in the early stages following the event (Lowe, Norman, & Bennett, 2000).

Thirdly, two different models (moderation and mediation models) deriving from the literature were tested. Results show that low level of intrapersonal EC was predictive of depression symptoms persistence even after controlling for depression symptoms at baseline. We did not observe the same significant results for anxiety symptoms persistence, although intrapersonal EC exhibited a trend toward significance. It is more than plausible that trait EC also influences anxiety symptoms persistence as suggested by the literature (Martins et al., 2010), but the study is probably underpowered. Regarding the role of HRV, we failed to show any association with depression and anxiety symptoms. This lack of significant result is contradictory with some previous studies showing that reduced HRV is associated with depression and anxiety (Chalmers et al., 2014; Kemp et al., 2010). Indeed, the neurovisceral integration model (Thayer & Lane, 2000) posits that low HRV reflects prefrontal cortex dysfunction, a physiological condition that characterizes psychopathology (Beauchaine & Thayer, 2015). However, others researchers also failed to show a significant relation between HRV and depression or anxiety (Agelink et al., 2001; Frasure-Smith, Lespérance, Irwin, Talajic, & Pollock, 2009; Hammel et al., 2011). These inconsistencies could be partially due to the patients' level of physical activity and psychological stress during the

period of HRV evaluation (Roach, Wilson, Ritchie, & Sheldon, 2004). This is also surprising that we did not find any interaction between HRV and trait EC, as previous studies have observed a buffer effect of HRV on the relationships between trait personalities and distress (Batselé et al., 2019; Ode et al., 2010). One possible explanation is that our population was composed of CHD patients, while the one we just cited was conducted on healthy participants (Batselé et al., 2019; Ode et al., 2010). These different results highlight the need for better understanding of HRV's role in mental health of CHD patients.

Some limitations have to be acknowledged. First, cardiac rehabilitation dropout of 50% was observed from Time 1 to Time 2 leading to a reduced sample. Statistical analyses revealed that people with higher intrapersonal trait EC were more prone to stop their cardiac rehabilitation. However, this finding has to be considered carefully as high EC is usually linked to adaptative health behaviors (Fernández-Abascal & Martín-Díaz, 2015). It is also possible that patients who stopped their cardiac rehabilitation exhibited others characteristics that would explain this dropout (i.e., better physical condition, other physical activity outside the cardiac rehabilitation). The resulting limited sample affects the statistical power of the study and can explain the lack of results regarding the predictive value of intrapersonal trait EC on anxiety. Second, our sample was composed of both males and few females. Although we did not observe significant differences on dependent variables between genders, it could impact the generalizability of our results (Martins et al., 2010).

Conclusion

As depression negatively impacts the prognostic of CHD patients (Barth et al., 2004), it is a major concern to examine the factors that explain the persistence of this trouble. Our study is the first that highlights the predictive value of trait EC on the persistence of depression symptoms in CHD patients at the end of cardiac rehabilitation. Future studies should further evaluate if EC training (Kotsou, Nelis, Grégoire, & Mikolajczak, 2011) could help decreasing the persistence of depression symptoms in this specific population.

Declaration of Conflicting Interests

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Note

1. For clarity reasons, we will refer to HRV in the rest of the manuscript in order to designate lnRMSSD.

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