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ARTICLE



5-HTTLPR polymorphism is associated with nostalgia proneness: The role of neuroticism

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ABSTRACT

Nostalgia, a sentimental longing for the past, is a self-relevant and social emotion. Nostalgia proneness is associated with alleviation of distress or instability (e.g., neuroticism). Although nostalgia proneness is heritable, the specific molecular contributors to this heritability are unknown. We focused on a polymorphism in the promoter of the serotonin transporter gene (5-HTTLPR) as a possible biological basis of nostalgia proneness, because the serotonin system has been associated with sensitivity to negative experience. Participants ($N = 397$ adults) who had reported levels of nostalgia proneness were genotyped. A subsample also completed a measure of neuroticism. Participants with the 5-HTTLPR short allele were higher on nostalgia proneness than those without this allele. Neuroticism mediated the relation between 5-HTTLPR and nostalgia proneness. These findings enrich our understanding of the genetic and personality underpinnings of nostalgia.

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Introduction

Nostalgia is defined as “a sentimental longing or wistful affection for the past” (Pearsall, 1998, p. 1266). The emotion is evoked when one reflects fondly and tenderly upon a momentous occasion from the past (e.g., in reference to one’s childhood, close relationships, or keepsakes). The emotion is bittersweet, albeit more sweet (e.g., pleasant) than bitter (e.g., sad). Nostalgia is self-relevant (because the self is the protagonist in nostalgic accounts), and social (because the self is almost invariably surrounded by close others). Nostalgia is observed across ages and cultures (for review, see: Sedikides et al., 2015).

Although nostalgia is likely a universal emotion, some individuals experience it more frequently than others. Indeed, prior research has established nostalgia proneness as a personality trait (Juhl, Routledge, Arndt, Sedikides, & Wildschut, 2010; Routledge, Arndt, Sedikides, & Wildschut, 2008). Elevated nostalgia proneness is associated with indicators of negative affectivity, such as loneliness (Zhou, Sedikides, Wildschut, & Gao, 2008) and, crucially, neuroticism (Seehusen et al., 2013), which entails negative affect and emotional instability

(Widiger, 2009). However, this association does not necessarily imply that nostalgia proneness is maladaptive. Instead, extensive empirical evidence suggests that nostalgia is an adaptive response to negative affectivity. As a self-relevant and social emotion, nostalgia could nurture various psychological resources, including (but not limited to) self-esteem, social connectedness (a sense of being accepted and loved), and meaning in life, thereby assuaging worries and negative affect (Sedikides et al., 2015).

Both environmental and genetic factors are thought to contribute to individual differences in nostalgia proneness (Johnson, Penke, & Spinath, 2011). A twin study has demonstrated that nostalgia proneness is partly shaped by hereditary influences (Luo, Liu, Cai, Wildschut, & Sedikides, 2016), but the biological basis for this heritability is unclear. One neurochemical system that may be involved in nostalgia proneness is serotonin. This hypothesis is based on two converging bodies of evidence. First, the serotonin system has been implicated in multiple component processes of nostalgia, including autobiographical memory (Sumner et al., 2014), reward (Li et al., 2016), affiliation (Kamilar-Britt & Bedi, 2015), neuroticism (Quilty, Meusel, & Bagby, 2008),

and buffering of social pain (Preller et al., 2016). Second, functional neuroimaging studies suggest that nostalgia is associated with activation of a set of limbic and paralimbic networks for memory, reward, and the self-networks that include the ventral striatum, hippocampus, and medial orbitofrontal cortex (Barrett & Janata, 2016; Oba, Noriuchi, Atomi, Moriguchi, & Kikuchi, 2016). These structures are heavily innervated by the serotonin system (Hensler, 2006; Way, Laćan, Fairbanks, & Melega, 2007). Therefore, on both functional and anatomical grounds, there is good reason to suspect a role for the serotonin system to be involved in nostalgia.

Related to the serotonin system, much of candidate gene research has focused on variation in the serotonin transporter gene. The most studied polymorphism is in the promoter region (5-HTTLPR), which has two primary alleles – short and long. A good deal of this research was sparked by the finding that individuals with the short allele, particularly two copies of the short allele, were more likely to become depressed when exposed to negative life events than individuals with two copies of the long allele exposed to similar events (Caspi et al., 2003). Although follow-up meta-analyses have led to conflicting conclusions regarding this finding (Karg, Burmeister, Shedden, & Sen, 2011; Sharpley, Palanisamy, Glyde, Dillingham, & Agnew, 2014; see also Culverhouse et al., 2017), laboratory stress studies have more reliably shown an association of the short allele with greater cortisol response (Alexander et al., 2014). One interpretation of this line of research suggests that the short allele heightens reactivity to negative events (Drabant et al., 2012; Osinsky et al., 2008).

In order to restore psychological homeostasis, people with short allele may engage in processes such as nostalgia. This inference is supported by evidence. The 5-HTTLPR short allele has been associated with higher levels of neuroticism (Schinka, Busch, & Robichaux-Keene, 2004). Meanwhile, nostalgia proneness is also related to neuroticism (Seehusen et al., 2013). Taken together, short allele carriers may be inclined to neuroticism, and consequently to nostalgia as a coping mechanism for negative events (Sedikides et al., 2015).

To summarize, we propose that serotonin is relevant to the heritability of nostalgia proneness. We focus, in particular, on the serotonin-transporter-linked polymorphic region (5-HTTLPR), a region in the gene (SLC6A4) that codes for the serotonin transporter. We hypothesize that individuals with the short allele in the 5-HTTLPR will manifest relatively higher levels of nostalgia proneness. Furthermore, we hypothesize that the

relation between 5-HTTLPR and nostalgia will be mediated by neuroticism.

Method

Participants

Participants were 397 Americans adults (180 [45.34%] females, 171 [43.07%] males, 46 [11.59%] unspecified; 258 [64.99%] community members, 137 [34.51%] college students, 2 [0.50%] unspecified) recruited by researchers at two large Midwestern US universities as part of a study on romantic relationships. Participants were recruited as dyads, resulting in 129 community couples, 66 student couples, 3 singlets, and 4 unspecified. The mean age of the 347 participants who indicated their age was 31.17 years ($SD = 11.85$).

Using a two-tailed alpha ($=.05$) and Cohen's (1988) effect size standards for r , this sample size afforded sufficient statistical power for detecting large ($r = .5$, power $> .99$) and medium ($r = .3$, power $> .99$) effects, but insufficient power for detecting small effects ($r = .1$, power $= .51$).

Participant race was determined by self-categorization, with 279 (70.28%) classified as White or Caucasian, 46 (11.59%) as Black/African American, 44 (11.08%) unspecified, 14 (3.53%) as mixed, 10 (2.52%) as Asian, 3 (0.76%) as Native Hawaiian/Pacific Islander, and 1 (0.25%) as other. Student participants were recruited through the psychology participant pool and received course credit, whereas community participants were recruited through advertisements and received \$50.¹ All participants provided written informed consent prior to testing.

Materials

Participants completed a packet of questionnaires, two of which pertained to nostalgia proneness and neuroticism. We assessed nostalgia proneness with the Southampton Nostalgia Scale (SNS; Sedikides et al., 2015), which consists of seven items that gauge proclivity to nostalgia (e.g., "How prone are you to feeling nostalgic?"), frequency of nostalgia (e.g., "How often do you experience nostalgia?"), and personal relevance of nostalgia (e.g., "How important is it for you to bring to mind nostalgic experiences?") (1 = *not at all*, 7 = *very much*; Cronbach's $\alpha = .92$ and $.95$ for the student and community samples, respectively). The SNS is a valid and reliable measure of nostalgia, given its (1)

¹Some participants received additional compensation in the form of money or course credit for completing a daily life experience study unrelated to the current research.

convergence with alternative measures of nostalgia (Routledge et al., 2008; Zhou et al., 2008) and (2) correlations with music- and scent-evoked nostalgia (Barrett et al., 2010; Reid, Green, Wildschut, & Sedikides, 2015).

We assessed neuroticism only among student participants with the corresponding subscale of the Big Five Inventory (John & Srivastava, 1999). This subscale consists of eight items (e.g., "I see myself as someone who is depressed, blue") (1 = *disagree strongly*, 5 = *agree strongly*; Cronbach's $\alpha = .82$). This subscale provides a valid and reliable measure of neuroticism (see John, Naumann, & Soto, 2008 for a review).

DNA extraction and genotyping

Participants provided a saliva sample using the Oragene Saliva kit OG-500 (DNA Genotek, ON, Canada). We conducted saliva collection and DNA extraction according to manufacturer (Oragene) recommendations. We genotyped the 5-HTTLPR using primers (Gelernter, Kranzler, & Cubells, 1997) and a protocol modified from Heils et al. (1996) and Anchordoquy, McGeary, Liu, Krauter, and Smolen (2003). The forward primer was 5'-ATGCCAGCACCTAACCCTAATGT-3' (labeled with 6-carboxyfluorescein fluorophore) and the reverse primer was 5'-GGACCGCAAGGTGGGCGGGA-3'. These primers yielded amplicons of 376 bp (short) or 419 bp (long). Polymerase chain reaction (PCR) was performed in a total volume of 20 μ L, containing 100 ng of DNA; 10 nM of each primer; 1x buffer; 2 mM MgCl₂; 10% DMSO (v/v); 2U Amplitaq Gold DNA polymerase (Applied Biosystems, Foster City, CA); 200 μ M of dATP, dCTP, and dTTP; 100 μ M of dGTP; and 7-deaza-2'-dGTP. Cycling conditions consisted of: (1) an initial 12 min denaturation at 94°C; (2) touchdown from 65°C to 55°C decreasing by 0.5°C per cycle with denaturation for 30 sec at 94°C, varied annealing temperatures consisting of 30 sec at 66°C (2 cycles), then 65°C (3 cycles), then 64°C (3 cycles), followed by hybridization for 1 min at 72°C; (3) 35 cycles with an annealing temperature of 63°C and the same denaturation and hybridization parameters; and (4) a final extension for 20 min at 72°C. The PCR products were electrophoresed on an ABI 3730 DNA analyzer (Applied Biosystems) with Genescan LIZ500 size standard (Applied Biosystems). Data collection and analysis used GeneScan and Genemapper software (Applied Biosystems).

We genotyped samples for rs25531 by incubating the reaction product from the 5-HTTLPR amplification with the restriction enzyme MspI (Whisman, Richardson, & Smolen, 2011). We then ran this digest on the ABI3730, according to the procedure described for the 5-HTTLPR. Given that the presence of the rs25531 G allele may render the long allele functionally equivalent to the short allele (Hu et al., 2005), we coded the combination of the rs25531 G allele and the long allele (L_G) as a short allele. Thus, we categorized participants with two short alleles, one short allele and one L_G allele, or two L_G alleles as short/short (SS); we categorized those with one short allele or one L_G allele as short/long (SL); and we categorized those with two long alleles and two rs25531 A alleles as long/long (LL).

For quality control, we reran 16 samples and found perfect concordance. Two samples were heterozygous for an extra-long Long allele (81 bp), and we thus omitted them from analyses due to differential effects on gene expression (Vijayendran, Beach, Plume, Brody, & Philibert, 2012). We could not genotype six samples.

Hierarchical linear modeling

Our data were dyadic and thus may introduce bias in many parametric analyses by violating the common assumption of independence (Bliese & Hanges, 2004). To test for presence of non-independence, we used intraclass correlation coefficient (ICC), which suggested interdependence for couple members' nostalgia (ICC = .151, $p = .005$) and neuroticism (ICC = $-.259$, $p = .012$),² but not the genotype (ICC = .052, $p = .319$). Therefore, we conducted hierarchical linear modeling (HLM) to analyze the dyadic data (Kenny, Kashy, & Cook, 2006),³ using linear mixed effects models. We ran these models based on the MIXED procedure in SPSS 23.0 (REML as estimation method, compound symmetry as covariance structure), with nostalgia as the outcome variable and 5-HTTLPR polymorphism as the level-1 predictor variable. This procedure estimates non-independence within dyads as a covariance. Further, to control for ethnicity, we ran models with ethnicity as another level-1 predictor. We also computed effect sizes in the metric of Pearson's r using the formula $r = \sqrt{t^2/(t^2 + df)}$.

Finally, we conducted HLMs in mediation analysis to estimate the following effects: (1) 5-HTTLPR on neuroticism; (2) neuroticism on nostalgia controlling for 5-HTTLPR; and (3) 5-HTTLPR on nostalgia with

²A negative intraclass correlation indicates that the variability within dyads exceeds the variability across dyads.

³Before HLM analyses, we tested for partner effect (i.e., whether one's 5HTTLPR can predict her/his partner's nostalgia or neuroticism). No partner effect emerged for nostalgia ($B = -0.17$, $SE = 0.17$, $t[324.06] = -0.99$, $p = .324$) or neuroticism ($B = -0.14$, $SE = 0.17$, $t[89.00] = -0.80$, $p = .425$).

Table 1. Genotype distribution across genders, samples, and ethnicities.

	SS	SL	LL
<i>Gender</i>			
Female	39	90	42
Male	49	69	43
Unspecified	8	20	14
<i>Sample</i>			
Community	61	123	64
Student	35	55	34
<i>Ethnicity</i>			
White or Caucasian	61	131	74
Black/African American	10	22	8
Mixed	7	4	3
Asian	8	2	0
Native Hawaiian/Pacific Islander	1	2	0
Other	1	0	0
Unspecified	8	18	14
<i>Total</i>			
	96	179	99

neuroticism controlled. All predictors were at level-1. To gauge the indirect effect from 5-HTTLPR to nostalgia via neuroticism, we used a Monte Carlo macro in SPSS (MCMED; Hayes, 2013).

Results

Genotype

Genotype distribution (Table 1) did not deviate from Hardy-Weinberg equilibrium (Stern, 1943), $\chi^2(2, N = 374) = 0.68, p = .712$. Further, the allelic distribution was comparable across genders ($\chi^2[2, N = 332] = 3.62, p = .163$) and samples ($\chi^2[2, N = 372] = .978, p = .613$). As expected (Gelernter et al., 1997), the allelic variation differed across ethnicities ($\chi^2[12, N = 374] = 28.00, p = .006$). The proportion of the SS category substantially varied from 22.9% in Caucasians to 80.0% in Asians. Consequently, we included ethnicity as a covariate in subsequent analyses.

Genetic effect

In Table 2, we present the mean levels of nostalgia proneness for the three genotypes. Following previous practices (Hu et al., 2005), we compared the mean levels of nostalgia proneness between short allele carriers (participants with short or L_G allele, SS/SL) and those with just the L_A allele (LL). Short allele carriers manifested higher levels of nostalgia proneness ($N = 249, M = 4.22, SD = 1.38$) than LL individuals ($N = 85, M = 3.85, SD = 1.48$), $t(332) = 2.10, p = .037, r = .114$. As participants were nested within couples,⁴ we used a hierarchical linear model (HLM) to re-test the genetic effect on nostalgia proneness (dummy code: LL = 0, SS/SL = 1). Consistent with the mean difference, allelic variants in 5-HTTLPR were significantly predictive of nostalgia, $B = 0.36, SE = 0.18, t(331.31) = 2.06, p = .040, r = .112$.⁵ The effect remained significant when we added ethnicity (Caucasians vs. all others) as a covariate ($B = 0.35, SE = 0.18, t[329.73] = 1.97, p = .049, r = .108$).⁶ We also carried out the HLM separately for the community sample and student sample, and found that the genetic effect did not vary significantly between samples (for more details, see online Supplementary Materials).

Mediation by neuroticism

Prior research has shown that the short allele of the 5-HTTLPR is associated with neuroticism, and neuroticism

Table 2. Means (SD) of nostalgia proneness and neuroticism across genotypes.

	SS	SL	LL
Nostalgia	4.02 (1.37) N = 88	4.32 (1.38) N = 161	3.85 (1.48) N = 85
Neuroticism	2.92 (0.86) N = 27	2.77 (0.75) N = 41	2.49 (0.59) N = 23

Neuroticism was only available for the student sample.

⁴We excluded the three singlets (dyads with data missing from one partner) from HLM analyses since they could not be included in the dyadic data structure.

⁵It is common for researchers to differentiate dyad members on factors like gender and analyze them as distinguishable dyads, particularly in regards to data from heterosexual couples (Kenny & Ledermann, 2010). Given that not all couples in our study were heterosexual and researchers caution against overemphasizing gender differences in dyadic studies (Ackerman, Donnellan, & Kashy, 2011), we treated all couples in HLM as indistinguishable dyads. However, to examine whether it was necessary to treat couples as distinguishable, we excluded homosexual couples ($N = 3$) and tested for distinguishability with only heterosexual couples. Following Kenny et al. (2006), we added gender (1 = male, 2 = female) as a level-1 predictor into the HLM (Model 1, $\chi^2[5] = 1146.21$, Akaike's Information Criterion [AIC; Akaike, 1987] = 1150.21). The gender effect was not significant ($B = -0.25, SE = 0.15, t[163.12] = -1.68, p = .095$), suggesting indistinguishability. The estimate for genetic effect ($B = 0.36, SE = 0.18, t[321.53] = 2.00, p = .046$) from Model 1 was almost the same as the one from the original HLM, which included all couples and treated them as indistinguishable. Furthermore, based on Model 1, we allowed for different variances across genders in the HLM (Model 2, $\chi^2[6] = 1145.37, AIC = 1151.37$). Then, we compared the two models and obtained a non-significant model change, $\Delta\chi^2(1) = -.842, p = .359$. In addition, Model 1 without heterogeneity of variance across genders yielded a lower AIC value, which suggested better fit. Taken together, heterosexual dyad members were indistinguishable, or at least needless to be differentiated.

⁶We also tested the significance of the genetic effect by including demographic variables (sample, age, and gender) as covariates in the HLM. We include the results in online Supplementary Materials.

is prognostic of nostalgia. Building on these findings, we used the student sample to examine (1) the link between 5-HTTLPR and neuroticism and (2) the subsequent link between neuroticism and nostalgia, controlling for 5-HTTLPR (Figure 1; Table 3). We conducted these mediational analyses using HLM, given the participants were nested in couples. The effect of 5-HTTLPR on neuroticism was marginally significant ($p = .069$, $r = .196$). Participants with the short allele ($N = 68$, $M = 2.83$, $SD = 0.79$) were higher on neuroticism than those without short allele ($N = 23$, $M = 2.49$, $SD = 0.59$). (We present, in Table 2, the mean levels of neuroticism for the three genotypes.) Furthermore, neuroticism significantly predicted nostalgia ($p < .001$, $r = .548$), above and beyond 5-HTTLPR. The direct effect of 5-HTTLPR on nostalgia (controlling for neuroticism) was not significant ($p = .437$, $r = .097$). As a final step, we tested the indirect effect of 5-HTTLPR on nostalgia via neuroticism and calculated a 95% confidence interval (CI) by using a Monte Carlo macro (based on 10,000 Monte Carlo samples; Hayes, 2013) in SPSS. The analysis revealed a significant indirect effect as 0.24 (95% CI [0.02, 0.50]).⁷ We also tested the reverse mediation model (i.e., nostalgia mediating the association between 5-HTTLPR and neuroticism) and found that the reverse mediation effect was not significant (see online Supplementary Materials). This further supported neuroticism as a mediator between 5-HTTLPR and nostalgia.

Discussion

Although nostalgia proneness is genetically influenced (Luo et al., 2016), the specific genetic variants contributing to this heritability have not been addressed. In this

Table 3. Hierarchical linear modeling (HLM) coefficients for the mediation model.

	<i>B</i>	<i>SE</i>	<i>t</i>	<i>df</i>	<i>p</i>	<i>r</i>
5-HTTLPR predicting neuroticism	0.33	0.18	1.84	85.07	.069	.196
Neuroticism predicting nostalgia	0.71	0.15	4.87	55.29	.000	.548
5-HTTLPR predicting nostalgia (direct effect)	0.21	0.27	0.78	63.44	.437	.097

sample, the short allele of the 5-HTTLPR was associated with higher levels of nostalgia proneness. Furthermore, in a subsample, neuroticism mediated the relation between 5-HTTLPR and nostalgia proneness.

Implications

Earlier research has linked the 5-HTTLPR polymorphism with heightened reactivity to negative events and emotions. To cope with such distress, people may resort to emotion regulation. Indeed, when exposed to stressful life events, carriers of the susceptible allele of the 5-HTTLPR can soften their emotional reactivity via cognitive reappraisal, an emotion regulation strategy that involves reframing the meaning of an event (Ford, Mauss, Troy, Smolen, & Hankin, 2014; Gross, 1998). Our findings contribute to this literature by showing that individuals with the susceptible allele are more inclined to nostalgia, which fosters psychological resources to counteract the discomfort engendered by adverse experiences (Sedikides et al., 2015; Wildschut, Sedikides, & Cordaro, 2011). For example, the 5-HTTLPR short allele has been associated with greater cortisol response in the Trier Social Stress Test (Alexander et al., 2014). Conversely, an experimental nostalgia induction blunts the subjective distress associated with this same stressor (Routledge et al., 2011; Sedikides & Wildschut, 2017), and such benefits due to momentary

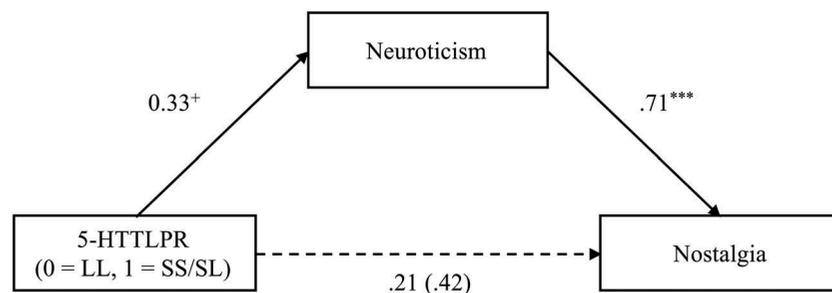


Figure 1. Neuroticism as the mediator of the relation between 5-HTTLPR and nostalgia proneness.

The total effect of 5-HTTLPR ($p = .171$) on nostalgia is in the parentheses, with the direct effect of 5-HTTLPR ($p = .437$) presented outside the parentheses. Significant paths are denoted in solid lines, while non-significant ones are denoted in dashed lines. $^+p < .10$, $***p < .001$.

⁷As nostalgia is a way to counteract life stressors, we wondered whether it moderated the relation between 5HTTLPR and neuroticism. To test this alternative hypothesis, we ran an additional HLM with nostalgia, 5HTTLPR genotypes, and their interaction as level-1 predictors, and with neuroticism as the outcome. The interaction between nostalgia and 5HTTLPR was not significant ($B = -0.05$, $SE = 0.12$, $t[86.14] = -0.46$, $p = .651$). Nostalgia did not moderate the 5HTTLPR-neuroticism association.

nostalgia are more likely to be reaped by people prone to nostalgia (Van Dijke, Wildschut, Leunissen, & Sedikides, 2015; Zou, Wildschut, Cable, & Sedikides, 2017).

Furthermore, we identified neuroticism as a mediator of the association between 5-HTTLPR and nostalgia proneness. Consistent with earlier research, we observed that neuroticism was associated with both 5-HTTLPR (Schinka et al., 2004) and nostalgia (Seehusen et al., 2013). Additionally, we found that neuroticism accounted for part of the link between 5-HTTLPR and nostalgia. This indirect effect provided additional support to our assertion that individuals with the short 5-HTTLPR allele may turn to nostalgia for coping with negative emotionality, including neuroticism (Sedikides et al., 2015; Seehusen et al., 2013).

Limitations and future research

Our study has four primary limitations. First, it was likely underpowered, given the small magnitude of the effect of 5-HTTLPR on nostalgia proneness. Nonetheless, the modest effect sizes were comparable to meta-analytic findings (e.g., $d = 0.18$ for 5-HTTLPR and neuroticism⁸; Munafò et al., 2009), and might be realistic estimates considering many other variables contributing to neuroticism and nostalgia. The power issue was more pertinent to the mediation analyses, where the sample size was substantially reduced. Smaller sample sizes can hinder detection of the direct effect as significant, rendering the distinction between complete and partial mediation highly ambiguous (Rucker, Preacher, Tormala, & Petty, 2011). Because it is inadvisable to make claims of complete (vs. partial) mediation based on the non-significance of the direct effect, we did not adopt this distinction. Replications with larger samples are needed based on power calculations in the 5-HTTLPR literature (Dick et al., 2015). Therefore, our findings should be considered preliminary. Second, as there is evidence showing that the short allele in 5-HTTLPR may entail susceptibility to not only negative but also positive environments (Haase et al., 2015; Schoebi, Way, Karney, & Bradbury, 2012), people with such an allele may function differently and cope with emotional distress differentially under varied environments (Kim et al., 2010; Way & Taylor, 2010). Thus, the influence of 5-HTTLPR on nostalgia proneness might be moderated

by environmental conditions (i.e., a gene-environment interaction). This interaction warrants future investigation. Third, the current study is based on variation of a single gene. It is unlikely that a single gene determines a complex emotion like nostalgia. Future empirical efforts could consider multiple genes and the relations among them in order to locate sets of genes that capture individual differences in nostalgia more fully and reliably. Fourth, although we demonstrated a link between 5-HTTLPR and nostalgia, it is still unknown how they are intertwined. Given that serotonin modulates the neural networks which underlie nostalgia⁹ (Barrett & Janata, 2016; Oba et al., 2016), these brain systems (i.e., memory, reward, and self systems) might constitute pathways by which variation in serotonin transporter function impact nostalgia. This conjecture invites genetic and pharmacological imaging tests.

Conclusion

Nostalgia, a vital and adaptive psychological resource, is embraced by people of all ages and across cultures. However, research on the biological processes underlying nostalgia is in its infancy. The present findings represent a first step toward understanding the biological basis of nostalgia, a step that will hopefully prove generative.

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Disclosure statement

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⁸For the convenience of comparison, $d = 0.18$ is equivalent to $r = 0.09$.

⁹We know that memory, reward, and emotion regulation networks are also modulated by dopamine (Grace, 2016; Nobili et al., 2017). Therefore, it is possible that dopamine is involved in nostalgia. To explore this, we genotyped two polymorphisms in genes related to dopaminergic function: A VNTR in exon 3 of the dopamine receptor D4 gene (DRD4) and a functional SNP in the Catechol-O-methyltransferase gene (COMT, rs4680). Importantly, the DRD4 gene codes for a dopamine receptor (D4) primarily located in the prefrontal cortex (Lahti et al., 1998) and the COMT gene codes for the COMT enzyme, which is the primary means of terminating dopamine signaling in the prefrontal cortex (Käenmäki et al., 2010). However, neither of these genes was associated with nostalgia ($ts < .9$, $ps > .3$, $rs < .05$).

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