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Sex differences in the etiology of disgust sensitivity: A preliminary behavioral genetic analysis

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ABSTRACT

Evidence suggests that Disgust Sensitivity (DS) is a personality trait that may confer risk for the development of some anxiety-related disorders. To examine the origins of this trait we administered the DS subscale of the Disgust Propensity and Sensitivity Scale-Revised to 90 monozygotic and 90 dizygotic twin pairs, of which 55% were women. The DS subscale consists of two dimensions; Somatic Disgust and Ruminative Disgust. Biometrical modeling techniques were used to estimate heritability of the DS dimensions by sex. For women, each dimension was influenced by a combination of genetic and environmental factors. More specifically, 40.1% of the variance in DS was observed to be due to additive genetic factors and the remaining variance due to non-shared environment. Correlations among DS dimensions for women could be explained by genetic and environmental factors influencing the two dimensions. For men, the two dimensions were influenced by environmental but not genetic factors. These findings suggest that the etiology of DS is complex and arises as a function of dimension-specific and non-specific etiologic factors that vary as a function of sex. The implication of these findings for the sex differences in the etiology of some anxiety-related disorders are discussed.

1. Introduction

Emerging research suggests that trait disgust proneness consists of two components (van Overveld, de Jong, Peters, Cavanagh, & Davey, 2006); disgust propensity (DP; the tendency to respond with disgust to various stimuli) and disgust sensitivity (DS; the tendency to experience disgust as aversive). It has been shown that trait disgust is present to a greater or lesser extent in all individuals (Olatunji & Broman-Fulks, 2007) and is relatively stable over time (de Jong, Andrea, & Muris, 1997). A large body of research has also implicated trait disgust in the development of various disorders (Olatunji, Armstrong, & Elwood, 2017; Olatunji, Cisler, McKay, & Phillips, 2010). For example, self-report measures of trait disgust have been found to correlate with measures of spider phobia (de Jong & Merckelbach, 1998; Mulkens, de Jong, & Merckelbach, 1996), even when controlling for trait anxiety (Olatunji, Williams et al., 2007). Research has also found a significant association between trait disgust and symptoms of blood-injection-injury (BII) phobia independent of trait anxiety (Olatunji, Williams, Sawchuk, & Lohr, 2006). Trait disgust may also contribute to post-traumatic stress disorder (PTSD) by increasing the frequency of intrusive memories after exposure to a traumatic event (Bomyea & Amir, 2012) or by enhancing the link between peritraumatic disgust and PTSD-symptom severity (Engelhard, Olatunji, & de Jong, 2011).

Although there is growing evidence suggesting that trait disgust may be transdiagnostic (i.e., relevant to many different disorders), research suggests that it may be most strongly associated with contamination-based obsessive compulsive-disorder (OCD). Indeed, the unique link between trait disgust and symptoms of contamination based OCD has been consistently observed in various samples (Deacon & Olatunji, 2007; Olatunji, Sawchuk, Lohr, & de Jong, 2004; Tolin, Woods, & Abramowitz, 2006). This is perhaps not surprising given the adaptive disease-avoidance function of disgust (Curtis, Aunger, & Rabie, 2004). Indeed, trait disgust predicts estimates regarding the likelihood of catching a disease when confronted with potentially contaminated stimuli, even after controlling for anxiety symptoms (Mitte, 2008). However, a growing body of research suggests that DP and DS components of trait disgust may differ in their association with symptoms of contamination-based OCD. In a regression equation where DP, DS, and negative affect were simultaneously entered as predictor variables, Olatunji, Cisler, Deacon, Connolly, and Lohr (2007) found that only DS significantly predicted fear of contamination and excessive washing behavior. Nicholson and Barnes-Holmes (2012) also found that DS predicted behavioral avoidance of fear-relevant and disgust-relevant stimuli independent of DP and anxiety, while DP did not. However,

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research examining phobic symptoms suggests that DP is a more robust predictor of spider phobia, while both DS and DP appear to play a role in BII phobia (Fergus & Valentiner, 2009).

Research showing that DS and DP yield different patterns of associations with anxiety disorder symptoms suggests different underlying mechanisms. Indeed, it has been proposed that DP may be characterized by avoidant action tendencies to repugnant materials (van Overveld, de Jong, & Peters, 2010), whereas disgust sensitivity is linked with more general emotional sensitivity (Goetz, Lee, Cougle, & Turkel, 2013). Given these differences, a better understanding of how individual differences in DP and DS are acquired may have important implications for the disorders in which the two traits are implicated. Although much remains unknown about the behavioral genetics of DS, some studies have examined the heritability of DP. In an initial study, Rozin and Millman (1987) examined the similarity of DP towards contaminated foods between monozygotic (identical; MZ) and dizygotic (fraternal; DZ) twins. The findings showed that the correlation between identical twins' DP was not significantly different from that of nonidentical twins. Although this initial study suggests that variability in DP does not have a strong genetic component, the exclusive focus on contaminated foods does limit claims that can be made about DP more broadly.

More recent research has attempted to more precisely delineate the genetic contributions to the development of DP. Sherlock, Zietsch, Tybur, and Jern (2016) estimated the proportion of variation due to genetic effects, the shared environment, and other (residual) sources for DP towards pathogens, sex, and moral cues in a sample of female identical and nonidentical twins and their siblings. Twin modeling of these data revealed that approximately half of the variation in DP was due to genetic effects. Although this is the most comprehensive examination of the behavioral genetics of DP to date, it is unclear if the same pattern of findings would be observed for DS. It is also unclear if this pattern of findings observed in females generalizes to males. There is rather convincing evidence that women report more disgust than men (e.g., Haidt, McCauley, & Rozin, 1994) and research has also shown that the sex difference in spider phobia (Connolly, Olatunji, & Lohr, 2008), BII phobia (Olatunji, Arrindell, & Lohr, 2005), contamination-based OCD (Olatunji, Arrindell et al., 2005), and death anxiety (Bassett, 2017) can be accounted for by the sex difference in trait disgust. Accordingly, a better understanding of the sex differences in the behavior genetics of DS may inform current understanding of the sex differences in these disorders.

The present study is the first, to our knowledge, to examine the behavior genetics of DS, the tendency to experience disgust as aversive. DS was assessed by a subscale of the well-validated Disgust Propensity and Sensitivity Scale-Revised (DPSS-R; van Overveld et al., 2006). Psychometric research has shown that the DS subscale of the DPSS-R consists of two components (Goetz, Cougle, & Lee, 2013). The first component reflects emotional sensitivity towards somatic aspects of disgust (Somatic Disgust) and the second component reflects negative appraisals of oneself in response to feeling disgusted (Ruminative Disgust). Goetz and colleagues also present findings suggesting that Somatic and Ruminative DS may differ in their association with anxiety disorder symptoms. Whereas Ruminative Disgust was consistently correlated with measures of obsessional symptoms, Somatic Disgust was more strongly associated with measures of contamination fear and health anxiety. Etiologic factors contributing to the two DS dimensions might include (a) additive genetic influences (i.e., the genes that each make a small but additive contributions to a person's level of severity on a given dimension), or (b) cumulative environmental events (i.e., learning experiences that incrementally strengthen a person's belief about the dangerousness of particular types of disgust-related sensations).

Although no study to date has examined the behavior genetics of DS, an evolutionary-functional level of analysis reveals that women have consistently higher levels of disgust than men (see Al-Shawaf, Lewis, & Buss, 2018 for review). Not only is this sex difference substantial in magnitude, it can be observed across diverse assessment methods and affects a wide array of outcomes. Given this robust sex difference in disgust that is evolutionary prescribed, there may be a stronger genetic component for sensitivity to disgust-related affect for women compared to men. That is, if natural selection favors greater disgust in women, then this will likely shape the selection of genes to be passed on; disgust-related genes will be preferentially passed on, leading to a greater heritability of disgust. Studies have examined the heritability of the related construct of anxiety sensitivity (AS). AS is the fear of anxiety-related sensations, arising from beliefs that the sensations are harmful (Reiss, McNally, & Reiss, 1985). Indeed, research has found strong correlations between the physical, social, and cognitive dimensions of AS and DS (Fergus & Valentiner, 2009). Previous research has shown that AS is heritable (Stein, Jang, & Livesley, 1999) and, more specifically, heritable only in women (Jang, Stein, Taylor, & Livesley, 1999; Taylor, Jang, Stewart, & Stein, 2008).

The present study builds on the existing literature in examining the behavior genetics of DS. Drawing from evolutionary perspectives on disgust and behavior genetics research on the related construct of AS, it was predicted that DS would be heritable in women but not in men. Given that the two dimensions of DS are correlated with one another (Goetz, Cougle et al., 2013), it was predicted that this would be due to shared genetic or environmental factors. In the case of men, we predicted that the two dimensions are correlated because they share a common environmental factor. For women, we predicted that the dimensions would be correlated because the dimensions have a common genetic factor and possibly a common environmental factor. Given the two dimensions are not perfectly correlated, it was also predicted that each dimension would also be influenced by dimension-specific genetic and environmental factors for women, and dimension-specific environmental factors for men.

2. Method

2.1. Participants

The sample consisted of 180 twin pairs (90 MZ and 90 DZ pairs) recruited as part of the Tennessee Twin Study (TTS) cohort (Lahey et al., 2008) for follow-up assessments 11–15 years after initial assessments. The sample consisted of the twin pairs described in Table 1. For this sample, 55% were female and most were either White (73%) or Black (23%).

2.2. Measures

Zygosity was assigned using a highly accurate questionnaire that assessed the physical similarities between pairs of twins (Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). The initial validation of the questionnaire showed that one independent well-trained observer assessed zygosity based on the questionnaire and made the correct diagnosis in 96% of the cases. A weighted index of eight similarity

Table 1
Within-pair Pearson correlations.

Type of twin pair	No. pairs	Disgust Sensitivity Total Score	Somatic Disgust	Ruminative Disgust
MZ-F	49	.47****	.47****	.39***
DZ-F	30	−.14	−.11	−.18
MZ-M	41	.22	.30	−.09
DZ-M	19	.34	.40	.05
DZ-O	41	−.23	−.26	−.19

Note. *p < .05, **p < .01, ***p < .005, ****p < .001. MZ = monozygotic, DZ = dizygotic, M = male, F = female, O = other (i.e., pairs composed of a male and female).

questions also yielded an accuracy of 98%. Ambiguous cases in the present sample were resolved using 12 polymorphic DNA markers obtained from cheek swabs during wave 1, and was confirmed for subjects using DNA from blood or cheek swabs in all cases originally labeled as MZ twins who participated in wave 2.

The Disgust Propensity and Sensitivity Scale-Revised (DPSS-R; van Overveld et al., 2006) is a 16 item measure designed to assess the frequency of disgust experiences (Disgust Propensity) and the emotional impact of disgust experiences (Disgust Sensitivity). Subjects rate their agreement with each item on a scale ranging from 1 ("never") to 5 ("always"). Participants completed the 8 item DS subscale in the present study. Previous research by Goetz, Cougle et al. (2013) suggests that the Disgust Sensitivity subscale consists of two reliable components: Somatic Disgust (Cronbach's alpha = .78) and Ruminative Disgust (Cronbach's alpha = .79). The two components also demonstrated good discriminant validity with Somatic Disgust showing stronger correlations with panic symptoms and Ruminative Disgust showing stronger correlations with measures of obsessional thinking.

The assessment of Somatic and Ruminative Disgust in the present study was informed by the findings of Goetz, Cougle et al. (2013). Specifically, the present study employed 6 items (When I feel disgusted, I worry that I might pass out; It scares me when I feel nauseous; I think disgusting items could cause me illness/infection; When I notice that I feel nauseous, I worry about vomiting; It scares me when I feel faint; I worry that I might swallow a disgusting thing) from the original Disgust Sensitivity Subscale of the DPSS-R to assess Somatic Disgust. The present study also employed the same 2 items (It embarrasses me when I feel disgusted; I think feeling disgust is bad for me) on the Disgust Sensitivity Subscale of the DPSS-R identified by Goetz and colleagues as reflecting Ruminative Disgust.

2.3. Procedure

Twin pairs completed the DS subscale of the DPSS-R online using the Redcap survey function. The final sample for analysis was limited to only those twin pairs for which there were complete data on the DS. That is, 180 pairs. For more details on recruitment, see Lahey et al. (2008).

2.4. Data analytic overview

Heritability estimates were based on similarities within twin pairs. MZ twin pairs share 100% of their segregating genes whereas DZ pairs share 50%. For a given variable (e.g., DS), larger within-pair MZ correlations than within-pair DZ correlations indicate the presence of genetic effects (i.e., effects due to segregating genes) because the greater MZ within-pair similarity is attributed to the twofold greater genetic similarity of MZ than DZ twins. That is, analyses were based on the within-pair similarities of MZ pairs compared to the within-pair similarities of DZ pairs. To compute a within-pair correlation, one member of a twin pair is labeled Twin 1 and the other member of a pair is labeled Twin 2. Thus, for a group of MZ twins, the within-pair correlations for a given variable are the correlations between the scores of Twin 1 and Twin 2. The same approach is used to compute within-pair correlations for DZ twins.

Within-pair twin correlations were initially examined for each sex. For variables in which there was evidence of heritability (i.e., MZ within-pair correlations greater than DZ within-pair correlations), biometric models of genetic and environmental influences were fitted to the covariance matrices of DS scores by means of Maximum Likelihood estimation using Mx software (Neale, Boker, Xie, & Maes, 2006). For each DS scale, MZ and DZ within-pair correlations were decomposed by means of standard biometric structural equation modeling into variance components attributable to the following: additive genetic factors (A), which cumulatively influence a given trait; shared environmental factors (i.e., factors common to both members of a twin pair; C); and nonshared environmental factors (E). Shared environmental factors include family influences common to both members of a twin pair, such as parental instruction or modeling (observational learning). Nonshared environmental factors include events affecting one twin but not the other. The path diagram for modeling A, C, and E can be found in standard texts on twin methodology (e.g., Jang, 2005, p. 34; Neale & Maes, 2004, p. 152).

The best-fitting model was identified by progressively dropping elements of the model and comparing the relative goodness-of-fit (i.e., models ACE, AE, CE, and E were compared with one another, and also compared with ADE and DE). For each observed variable, the least parsimonious model consists of A, C, and E variance components (the ACE model; see Jang, 2005, or Neale & Maes, 2004, for an overview of this basic twin model). The goal of model fitting was to determine whether a more parsimonious model (i.e., AE, CE, or E) had a goodness-of-fit that was equal to or better than the ACE model.

Goodness-of-fit was assessed by the Akaike Information Criterion (AIC) and the Root Mean Square Error of Approximation (RMSEA), where the best-fitting model is that which has the smallest AIC and RMSEA values.

Cholesky's decomposition (Neale et al., 2006) was used to compute genetic and environmental correlations among the observed variables. Genetic correlations are correlations among the genetic components of the variables. Environmental correlations are correlations among the components of the variables due to environmental factors. If phenotypic variables are etiologically related to one another, then they should have significant genetic and/or environmental correlations. The path diagram used for Cholesky's decomposition can be found in Neale and Maes (2004, p. 191) and Jang (2005, p. 35). The genetic correlations between variables were based on what was found to be the best-fitting model (i.e., AE). The goal of the Cholesky decomposition was to compute the correlations between each of the A components for each observed variable, and correlations between each of the E components of the observed variables. If some or all of these correlations are significant, then this suggests that the symptom variables many have genetic and/or environmental factors in common.

3. Result

3.1. Preliminary analysis

For the full sample, the mean total score for the DS subscale of the DPSS-R (i.e., sum of scores on all 8 items) was 14.7 (SD = 5.4), which is consistent with the scores previously reported for unselected samples (Fergus & Valentiner, 2009). In the present sample, there were significant differences between the mean scores for women (M = 15.4, SD = 5.8) and men (M = 13.8, SD = 4.7), $t(358) = 2.92$, $p < .004$. Coefficient alpha values were as follows. Females: DS total = .84, DS somatic = .77, DS ruminative = .79. Males: DS total = .84, DS somatic = .82, DS ruminative = .65. These indicate acceptable values for research purposes. There was no evidence that the pattern of results (e.g., the correlations in Table 1) varied as a function of scale reliability.

3.2. Within-pair correlations

Table 1 shows that within-pair correlations were significant only for female MZ twins. For female twins, the within-pair correlations for MZ twins were greater than those of DZ twins for each of the DS subscales and its somatic and ruminative components ($Zs > 2.45$, $ps < .007$). For male twins, within-pair correlations were not significant, and MZ within-pair correlations were not greater than DZ within-pair correlations for the DS subscale and its components ($Zs < 0.45$, $ps > .300$). Given that MZ correlations were not larger than DZ correlations for males, whereas MZ correlations were larger than DZ correlations for females, more detailed sex-limitation analyses were not warranted because the results clearly showed that disgust sensitivity was heritable

Table 2
Females: Behavioral-genetic modeling fit statistics.

Variable	Model	Goodness-of-Fit Indices	
		AIC	RMSEA
Disgust Sensitivity Total Score			
	ACE	−2.010	0.109
	AE	**−4.010**	**0.082**
	CE	0.070	0.168
	E	2.589	0.141
Somatic Disgust			
	ACE	−2.636	0.093
	AE	**−4.636**	**0.066**
	CE	−0.707	0.151
	E	2.188	0.139
Ruminative Disgust			
	ACE	−2.263	0.102
	AE	**−4.263**	**0.075**
	CE	−1.507	0.131
	E	−0.990	0.108

Note. Bold = best-fitting model for each variable. AIC = Akaike Information Criterion, RMSEA = Root Mean Square Error of Approximation. For the Model column, A = additive genetic effects, C = shared (common) environmental effects, and E = non-shared environmental effects.

only in females. Accordingly, the following behavioral-genetic analyses were limited to females. For males, DS was entirely influenced by environmental factors.

3.3. Behavioral-genetic analyses for females

Table 2 shows the model-fitting results for females. Here it can be seen that for each of the three variables, the best-fitting model consisted of a combination of additive genetic effects (A) and non-shared environment (E). Table 3 shows the parameter estimates (and 95th percentile confidence intervals) for the AE model for each of the disgust variables. Each parameter estimate refers to the proportion of explained phenotypic variance due to either A (additive genetic factors) or E (non-shared environment). The table shows that genetic factors accounted for 31.5–40.4% of the variance in the phenotypic scores on the DS measures, and nonshared environment accounted for the remaining variance.

A Cholesky decomposition was conducted in order to compute the genetic and environmental correlations (and 95th percentile confidence intervals) between somatic disgust and ruminative disgust. The two measures had a very high genetic correlation, whose confidence interval overlapped with 1.000: Genetic r = .863 (.238–.1.000). A parsimonious interpretation of this result is that somatic disgust and ruminative disgust are influenced by a common genetic factor. The two measures had a smaller but significant nonshared environmental correlation: Environmental r = .590 (.392–.738). This suggests that the measures may have environmental etiologic factors in common, but also are likely to be influenced by environmental factors that are specific to each form of DS.

Table 3
Females: Behavioral-genetic modeling: Parameter estimates for the best fitting (AE) model (and 95% confidence intervals).

Variable	A	E
Disgust Sensitivity Total Score	.401 (.138 - .611)	.599 (.390 - .862)
Somatic Disgust	.404 (.143 - .612)	.596 (.389 - .857)
Ruminative Disgust	.315 (.047 - .543)	.685 (.457 - .953)

4. Discussion

A growing body of research has implicated DS, the tendency to experience disgust as aversive, in the development of some anxiety related disorders (Olatunji et al., 2017). However, nature and etiology of DS remains largely unclear. To fill this important gap in the literature, biometrical modeling techniques were used to estimate heritability of the DS and its two dimensions: Somatic Disgust and Ruminative Disgust. The present study is the first, to our knowledge, to examine the behavior genetics of DS. Another major aim of the present study was to also examine sex differences in the behavior genetics of DS. For female twins, the within-pair correlations for MZ twins were greater than those of DZ twins for DS and its two dimensions. For male twins, however, within-pair correlations were not significant, and MZ within-pair correlations were not greater than DZ within-pair correlations for DS and its two dimensions. These findings suggest that the causes of DS differ as a function of sex. In women, DS arises from a combination of additive genetic and non-shared environmental factors, whereas in men DS arises entirely from environmental factors.

The stronger genetic effects of DS for women may partially be explained by the sex specific adaptive function of disgust. According to the compensatory behavioral prophylaxis hypothesis (Fessler & Navarrete, 2003; Fessler, 2001), the mother's body must tolerate a half-foreign blastocyst in order for pregnancy to occur, however the immune system is engineered to detect and attack foreign bodies. As an adaptation that protects the blastocyst from the maternal immune system, immune responses are decreased by progesterone in the luteal phase of the menstrual cycle and in pregnancy (Fleischman & Fessler, 2011). The cost of this adaptation, however, is concurrent heightened susceptibility to disease. Accordingly, disgust has evolved as an adaptive psychological mechanism that promotes avoidance of potential contaminants during these periods of reproductive immunomodulation to decrease the likelihood of infection. Consistent with this hypothesis, disgust reactions are highest in the first relative to the second and third trimesters of pregnancy, a period of heightened immunosuppression and vulnerability to infection (Fessler, Eng, & Navarrete, 2005; Zelazniewicz and Pawlowski, 2015). Furthermore, previous research has shown that measures of DP and DS are highly correlated (r = .59; van Overveld et al., 2006), a finding that would be consistent with the shared function of preventing infectious disease. This evolutionary view may partially explain why higher levels of DP and DS are observed in women compared to men. Given the findings from previous research (Sherlock et al., 2016) and those of the present findings, future research is needed to delineate the extent to which the evolutionary account of heightened disgust reactions among women explains the strong genetic influence on DP and DS among women.

The findings regarding the sex differences in the heritability of DS are also consistent with previous research showing that the tendency to acquire various anxiety disorder symptoms are more strongly heritable in women than men (Eley, 2001). This finding also complements research showing that the sex differences in various anxiety disorders can be explained by sex differences in disgust (Bassett, 2017; Connolly et al., 2008; Olatunji, Arrindell et al., 2005). An important interpretation of this work is that the heritability of DS among women, relative to other personality traits, may be a powerful but understudied determinant of the etiology and maintenance of various anxiety related disorders that are more commonly observed among women compared to men. Although the findings showed that genetic factors accounted for 31.5–40.4% of the variance in the phenotypic scores in DS, it is important to note that nonshared environment accounted for the remaining variance. "Nonshared" features of the environment are any aspects of the environment and any experiences that can be different for different children within the same family. With regards to DS, this may consist of differences in learning history. For example, Stevenson, Oaten, Case, Repacholi, and Wagland (2010) found evidence for parent-child transmission of disgust with parents of younger children emoting

more disgust to their offspring and showing greater behavioral avoidance when exposed to a disgust elicitor.

The present findings also suggest that Somatic and Ruminative DS essentially arise from the same genetic influence for women but differ to some extent in their environmental influences. There are clearly multiple components to DS and these components may be expected to originate from different sources. Differences in the environmental influences to Somatic and Ruminative DS may be explained, in part, by differences in learning influences. Somatic DS reflects emotional sensitivity towards somatic aspects of disgust and it likely originates from the same influences as emotional sensitivity towards physical sensations in general. For example, previous research has shown that the learning experiences of those high in emotional sensitivity towards physical sensations consisted of more parental reinforcement of sick-role behavior related to somatic symptoms in general compared to those low in such sensitivity (Watt, Stewart, & Cox, 1998). Furthermore, stressful life events related to health have been found to be longitudinally associated with emotional sensitivity towards physical sensations (McLaughlin & Hatzenbuehler, 2009). These findings suggest that learning associated with physical health may be more strongly associated with the origins of Somatic DS. This view is consistent with research showing that DS is more strongly associated with health anxiety than DP (Brady, Cisler, & Lohr, 2014).

The environmental influences of Ruminative DS compared to those of Somatic DS may also be relatively nonspecific. Ruminative DS is a pattern of repetitive, self-directed thought, focused on symptoms of disgust and potential causes and implications of those symptoms. The environmental influences of Ruminative DS may be expected to overlap with those of rumination in general. Previous research has shown that general rumination is moderately heritable, with the remaining variance due to nonshared environmental influences (Johnson, Whisman, Corley, Hewitt, & Friedman, 2014). The nonshared environmental influences that differentiate Ruminative DS from Somatic DS may have their origins in adverse experiences in childhood, including emotional maltreatment and overcontrolling parenting (Spasojevic & Alloy, 2002).

The present findings suggest that DS and its components are heritable among women. Although this suggests it may be worthwhile to search for genes that contribute to variance in DS among women, evidence from many large genome wide association studies indicate that there are likely many genetic variants, each with a very small effect on individual differences in DS in women, rather than variants with large effects. However, it is important to note that the present study is preliminary in nature with several limitations. Although this, to our knowledge is the first study to examine the heritability of DS, the sample was not large and the design did not shed light on the nature of the genes involved or the important environmental factors that play a role in DS. Although twin studies are an informative source of information about genetic effects on individual differences, they are not without potential limitations. For example, if the rearing environments of MZ and DZ twins are not analogous, then the estimates of heritability generated from the twin study approach may be confounded with differences between MZ and DZ twins in the similarity of their rearing environment (Felson, 2014). This highlights the importance of controlling for environmental similarity in future research on the heritability of DS. With regard to sample size, it is important to note that there are no hard-and-fast rules for determining sample size. It partly depends on the magnitude of the genetic effects. Table 1 shows that for women the MZ correlations were substantially larger than the DZ correlations, with the absolute value in the difference between MZ and DZ correlations ranging from .57 to .61. These are very large effects, in terms of Cohen's (1988) criteria. This means that it would be possible to detect significant genetic effects even with a relatively small sample. Is it possible that the genetic effects for males would have been significant if the study had greater statistical power? This is possible but Table 1 shows that for males, the MZ correlations were actually *smaller* than the DZ correlations, although non-significantly so. This means that there was no evidence of genetic effects for males, regardless of the statistical power of the study.

Another limitation is that only DS was assessed in the present study. Although Sherlock et al. (2016) have examined the estimated the proportion of variation in DP due to genetic and environmental effects, their measure of DP is specific to pathogens, sex, and moral cues. In contrast, the DP subscale of the DPPS-R assesses one's proneness towards experiencing disgust independent of contextual cues. Accordingly, examination of the DP subscale of the DPSS-R may allow for more meaningful comparisons to the present study. Despite the study limitations, the results encourage further research on the role of genetic and environmental factors in DS and other disgust-related constructs, and along with research on how these factors might vary as a function of sex. A further question for future research is whether the roles of genetic and environmental factors vary as a function of other variables, such as age, ancestry, and severity of disgust-related traits. With regard to severity, a question for further research is whether the importance of genetic factors varies with the severity of disgust-related traits. Answers to questions such as these should provide a better understanding of the causes of disgust-related psychopathology.

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