Genetic and environmental influences on disgust proneness,
contamination sensitivity, and their covariance

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Abstract

Dozens of studies indicate that individuals more prone to experiencing disgust have stronger symptoms of anxiety disorders – especially contamination sensitivity. However, no work has informed the degree to which this relationship arises from genetic versus environmental factors. The present study fills this gap by measuring disgust proneness and contamination sensitivity in a sample of 7,199 twins and siblings of twins, including 1,411 complete twin pairs. Disgust proneness was related to contamination sensitivity, $r = .32$. Multivariate twin modeling revealed that genetic factors accounted for 34% and 40% of the variance in disgust proneness and contamination sensitivity, respectively, and that the correlation between the two traits reflected overlapping genetic (54%) and unshared environmental (46%) – but not shared environmental – influences. While consistent with work indicating that disgust proneness relates to contamination sensitivity, results suggest that parental-modeling hypotheses for explaining this relationship be re-evaluated.

People vary in the degree to which they experience disgust toward things like bodily wastes, dead animals, and spoiled foods – that is, they vary in disgust proneness. A recent meta-analysis of 83 studies indicates that this trait covaries with symptoms of anxiety disorders (i.e., dimensional assessments of categorical diagnoses), even after controlling for negative affect (Olatunji et al, 2017). This relationship is stronger for so-called disgust-related disorders (e.g., obsessive-compulsive disorder, blood-injury-injection phobia, post-traumatic stress) than other anxiety disorders (e.g., social anxiety disorder, agoraphobia, generalized-anxiety disorder), and it is especially strong for a specific facet of obsessive-compulsive disorder: contamination sensitivity (i.e., aversions to direct and indirect contact with objects interpreted as dirty or contaminating).

Clinical research on disgust proneness has sparked hypotheses regarding the etiology of such disorders with the goal of tailoring interventions to target characteristics of disgust not shared by general anxiety or fear (e.g., its distinct developmental inputs, mechanistic underpinnings, and resistance to extinction; Amaroso et al., 2019; Knowles et al., 2019; Olatunji et al., 2017). One prominent hypothesis suggests that higher disgust proneness leads to an overestimate of the threat posed by infectious disease, which in turn leads to vigilance toward pathogen cues and, consequently, downstream anxiety (Olatunji et al., 2017; Widen & Olatunji, 2016). Critically, this hypothesis also proposes factors that might lead to the development of both disgust proneness and contamination sensitivity. While acknowledging genetic influences, it emphasizes potential effects of parents' behavior. Candidate parental behaviors include frequent expressions of disgust and a controlling parenting style, with the former putatively leading children to model disgust, and the latter putatively inhibiting the extinction of children's disgust-related anxieties that might follow from exposure. These possibilities seemingly resonate with

multiple lines of evidence, including findings that children's disgust proneness and fears of disgust-eliciting animals (e.g., spiders) covary with their parents' disgust proneness (Davey et al., 1993), that mothers of spider-phobic girls find spiders to be more disgusting than do mothers of non-phobic girls (De Jong et al., 1997), that parents and offspring score similarly on contamination sensitivity measures (Rozin et al., 1984), that parents' verbal communication of disgust toward animals influences their children's reactions to those animals (Muris et al., 2013), and that parents of younger children avoid contact with disgust-elicitors more than parents of older children, potentially to model such avoidance especially for younger children (Stevenson et al., 2010).

Caution is warranted in interpreting such observations as evidence that adults' disgust proneness and contamination sensitivity stem from such parental dispositions or behaviors. Decades of research finds that similarities between family members are better accounted for by shared genetic factors than by shared environmental ones (Plomin et al., 2016). Further, while some laboratory experiments report that parents' disgust expressions influence offspring behavior, others report similar effects from unrelated individuals (Askew et al., 2014). If disgust proneness, contamination sensitivity, or both are indeed influenced by others' expressions of disgust, then parental effects might be overwhelmed by the influence of the thousands of other people encountered throughout a child's development. Indeed, aspects of the shared environmental (including, potentially, exposure to the same parents) underlie many types of psychopathology in children, but these effects decline over time, to the point that they are undetectable in adults (Bergen et al., 2007).

The rich literature investigating the relationship between disgust proneness and contamination sensitivity is exclusively comprised of phenotypic correlations – that is, bivariate

correlations between observable characteristics. Such correlations alone cannot evaluate the hypotheses that disgust proneness and contamination sensitivity are shaped by parental modeling or parenting styles, as is their covariance. These limitations result from the fact that phenotypic correlations can arise because the same environmental factors influence both traits or because the same genetic factors influence both traits. Twin studies are uniquely positioned to partition such phenotypic correlations into genetic, shared environmental, and non-shared environmental components, because monozygotic (MZ) twins share nearly all of their segregating genes (~100%), while dizygotic (DZ) twins and siblings share, on average, 50% of their segregating genes. At the same time, DZ twins share the same family environments (including being exposed to the same parents), just as MZ twins do. Hence, greater similarities between MZ twins relative to DZ twins indicate genetic influences on a trait. Based on the similarity of MZ twins relative to DZ twins, we can estimate the degree to which DZ twins should be similar based on genetic influences alone. Any similarity that is not genetic in nature is the result of environmental factors shared by twins. If disgust proneness and contamination sensitivity are jointly influenced by exposure to parents who model disgust or adopt a specific parenting style, we should find that shared environmental components influence both traits and their covariance. The current study tested the genetic versus environmental influences on disgust proneness, contamination sensitivity, and their covariance.

Method

Participants

Invitation letters were sent to twins and siblings of twins identified from the Central Population Registry in Finland. Individuals were invited only if their mother tongue listed in the Central Population Registry was Finnish, if they resided in Finland at the time of gathering their

contact addresses, and if they were over 18 years old. A subset of individuals ($N = 7,716$) had participated in similar data collections conducted by the same research group before (for an elaboration on this cohort, see Johansson et al., 2013) and indicated a willingness to participate in future studies (in 2006 or 2012/2013). We also obtained addresses for a cohort of twins and siblings of twins who had not previously participated in studies performed by our research group. These twins were born between March 13, 1988 and October 4, 2000 (their siblings were any biological siblings aged at least 18). In total, addresses of 33,390 individuals were obtained. Of these, 179 had addresses abroad, leaving us with 33,211 eligible addresses. Recipients were invited to respond to an online survey with an individual 8-character code that was used to identify twin pairs and individuals from the same family, and they were offered entry into a raffle that contained 40 gift vouchers to a Finnish network of companies operating in the retail and service (e.g., restaurants and petrol stations) sectors worth €100 each as prizes. The first invitation letters were sent to potential participants in November, 2018. Those who did not respond were sent a maximum of two reminder letters spaced 2-3 weeks apart. We used a postal service that did not include an option to return undeliverable letters; hence, the response rate presented here conservatively estimates that all invitation letters were received. We were informed that 31 individuals were unable to participate (e.g., due to relocation, severe disability, or death). Data collection concluded in the first week of January, 2019.

In total, 9,564 individuals (6,965 twins and 2,592 siblings, 7 unknown) responded, resulting in a total response rate of 29%, with 9,319 (97%) respondents consenting to their data being used for scientific purposes. The current analyses include the 7,199 participants who completed both the disgust proneness and contamination sensitivity instruments and were either twin singletons, members of a twin pair, or siblings of at least one twin who provided data

(66.3% female; $M_{\text{age}} = 29.51$, $SD_{\text{age}} = 7.84$). Of these, 2,822 were members of complete twin pairs, including 274 monozygotic male (MZm), 832 monozygotic female (MZf), 240 dizygotic male (DZm), 740 dizygotic female (DZf) and 736 dizygotic opposite-sex (DOS) twins. A further 3,502 were twin singletons, and 875 were siblings of participating twins. This sample is ~71 times larger than the median sample size included in a recent meta-analysis on this topic (Olatunji et al., 2017) and, to the best of our knowledge, the largest sample to have ever completed disgust proneness and contamination sensitivity instruments. It affords between 80% and 90% power to detect shared environment components of $C = .20$ (depending on additive genetic components ranging between .10 and .50 – see supplement Figure 1 for a power curve). Zygosity was available for some twins based on earlier genotyping, and it was estimated for other twins using instruments with excellent accuracy when validated using DNA polymorphisms (see supplement for further details).

Ethical review

The Ethics Review Board of Åbo Akademi University in Turku, Finland, reviewed and approved the of the research plan describing the data collection procedure. Data collection involved no invasive procedures, and participants were informed that participation was voluntary and that they were free to terminate their participation at any time without giving a reason. All participants provided written, informed consent in accordance with the Declaration of Helsinki prior to responding to the survey.

Measures

All items were presented in Finnish. English versions of the scales were translated by a native Finnish speaker who is fluent in English, and they were back-translated to English by another native Finnish speaker also fluent in English and without access to the original English

items. A native English speaker then compared the back-translated version with the original English versions and flagged discrepant items. The original translator then reviewed and, where appropriate, revised those items. Finally, two other native Finnish speakers checked all items for grammar and comprehensibility.

Contamination sensitivity. To measure contamination sensitivity, we administered the contamination subscale of the Padua Inventory-Revised (Burns et al., 1996). This 10-item instrument asks participants to respond to statements such as “I wash my hands more often and longer than necessary” on a 0 (not at all) to 4 (very much) scale.

Disgust proneness. To measure disgust proneness, we used the seven-item pathogen domain of the Three Domain Disgust Scale (TDDS; Tybur et al., 2009), which asks participants to report how disgusting they find experiences such as “Stepping in dog poop” and “Seeing some mold on old leftovers in your refrigerator” on a 0 (not at all disgusting) to 6 (extremely disgusting) scale. We chose the TDDS because it has less item overlap with contamination sensitivity instruments than do other frequently used disgust instruments. For example, the Disgust Scale (Haidt et al., 1994), which was used most widely in the literature meta-analyzed by Olatunji and colleagues (2017), contains multiple items with content that overlaps with Padua Inventory items. Compare, for example, the Disgust Scale item, “I would never let any part of my body touch the toilet seat in a public restroom,” with the Padua Inventory item, “I avoid using public toilets because I am afraid of disease and contamination.” Given such shared item content, previously observed relationships between disgust proneness instruments and contamination sensitivity might partially reflect method variance. Using the TDDS presumably attenuates this issue, since item content does not similarly overlap with the Padua Inventory.

Before twin modeling, we conducted a principal axis factor analysis on the seven TDDS pathogen items and the 10 Padua Inventory contamination items in SPSS v.25¹. A visual inspection of the scree plot indicated the presence of two factors (the five highest eigenvalues were 5.71, 2.54, 1.01, 0.91, 0.76). After oblimin rotation, the pattern matrix revealed that all seven disgust proneness items ($\alpha = .79$) loaded saliently on one factor (all primary loadings $> .46$, no cross-loadings greater than $.06$), and all 10 contamination sensitivity items ($\alpha = .88$) loaded saliently on the other factor (all primary loadings $> .56$, no cross-loadings greater than $.08$). The relationship between disgust proneness and contamination sensitivity ($r = .32; .38$ when disattenuated for unreliability) was similar to that reported in a recent bias-corrected meta-analysis of 83 published effect sizes estimating the relationship between disgust proneness and anxiety disorder symptoms ($r = .36$; Olatunji et al., 2017).

Statistical analyses

Genetic analyses were conducted with the OpenMx package in R (Boker et al., 2011) using the full information maximum likelihood option, with responses from twin singletons contributing only to estimates of means and variances. We began with a saturated model, which allows means and standard deviations to differ between the sexes and correlations to differ between both zygosity categories and sexes. Differences between the negative log-likelihoods ($-2LL$'s) of more- versus less-constrained models were tested using χ^2 tests ($\alpha < .01$), with degrees of freedom reflecting differences in parameter estimates across models. If equality constraints did not significantly diminish model fit, the more parsimonious model was retained.

¹ We conducted similar analyses using the complete TDDS, which includes 7-item pathogen, sexual, and moral subscales (see Amoroso et al., 2019, for a review of this instrument's clinical utility). A factor analysis indicated four separate factors (contamination sensitivity and pathogen, sexual, and moral disgust). Regressing contamination sensitivity on the three disgust domains revealed that pathogen disgust related to the contamination sensitivity, $r_p = .26$, but relationships with sexual and moral disgust did not exceed $r_p = .06$. See the online supplement for further details.

We first tested for sex differences in means and variances by constraining those values to be equal across the sexes. We then imposed equality constraints on the correlations between male-male and female-female twin pairs to test whether genetic and environmental influences varied across the sexes (quantitative sex differences). Then, we imposed equality constraints on the correlations between same-sex and opposite-sex DZ twins to test for sex differences in the genetic and environmental factors underlying variance in men and women (qualitative sex differences). Finally, we constrained sibling correlations to be equivalent to DZ twin correlations to test for twin-specific environmental effects.

Estimating genetic and environmental effects

Using structural equation modeling, observed phenotypic variance can be partitioned into additive genetic (A), non-additive genetic (D), shared environmental (C), and non-shared environmental (E) components. Since MZ twins share nearly all of their segregating genes (~100%), while DZ twins and siblings share, on average, 50% of their genes, higher MZ compared to DZ and sibling correlations indicate genetic factors (with MZ correlations more than double DZ and sibling correlations indicative of non-additive genetic factors). DZ twin and sibling correlations more than half MZ twin correlations indicate that shared (common) environmental influences underlie trait variance. The remaining variance is attributed to non-shared environmental influences and measurement error. Because C and D have opposing effects on DZ and sibling correlations, they cannot be simultaneously estimated in the classical twin design. In a way similar to that described above, cross-twin-cross-trait correlations are used in a bivariate model to estimate the additive genetic, non-additive genetic, shared environmental, and unique environmental influences on the covariance between disgust proneness and contamination sensitivity.

Results

Contamination sensitivity did not differ between twins and siblings, $\chi^2(2) = 4.28, p = .12$, though disgust proneness did, $\chi^2(2) = 11.74, p = .003$, albeit slightly ($d = .03$). Consistent with past work, both disgust proneness and contamination sensitivity varied across the sexes, with women scoring higher on both traits ($p < .001$; see Supplementary Table 1 for sex-specific means and standard deviations, as well as all MZ, DZ, and sibling correlations). However, twin correlations did not differ between same-sex male and female twin pairs (quantitative sex differences), nor between the same-sex DZ and opposite-sex twins (qualitative sex differences) (all p 's $> .16$). Sibling correlations did not differ from DZ twin correlations (p 's = .77, .85, and .06 for disgust proneness, contamination sensitivity, and their covariance, respectively). Hence, correlations between additive genetic factors were estimated at 1.0 for MZ twins and 0.5 for both siblings and DZ twins (see Supplementary Tables 2 and 3 for all test statistics).

Figure 1. The bivariate genetic model. A1 and A2 are additive genetic factors, with factor loadings a11, a21, and a22, and E1 and E2 are unique environmental factors with factor loadings e11, e21, and e22. For MZ twin pairs, correlation between A in Twins 1 and 2 is 1; for DZ and sibling pairs, this correlation is .5 (Neale & Cardon, 1992). The non-shared environmental effects (E) in Twin 1 are uncorrelated with those in Twin 2. Standardized path coefficient estimates are displayed on the right half of the figure.

Table 1. Standardized genetic and environmental estimates (with 95% confidence intervals) and model fit statistics for the bivariate model. AE models are compared to both ACE and ADE models, and E models are compared to AE models. Results from the best fitting model are depicted in bold.

		Model	A	C	D	E	-2LL	df	Compared to model	χ^2	<i>p</i>
Disgust proneness	1	ACE	34% (27-41%)	0% (0-0%)	-	66% (59-72%)	33741.74	14128	-	-	-
	2	ADE	4% (0-20%)		38% (19-48%)	58% (52-64%)	33722.06	14128	-	-	-
	3	AE	34% (27-41%)		-	66% (59-72%)	33741.74	14129	1	.00	1.00
							33727.60	14129	2	5.40	.02
	4	E	-	-	-	100% (100-100%)	33789.84	14130	3	48.10	<.001
Contamination sensitivity	1	ACE	40% (33-45%)	0% (0-0%)	-	61% (55-67%)	33741.74	14128	-	-	-
	2	ADE	18% (0-36%)	-	26% (4-47%)	56% (51-63%)	33722.06	14128	-	-	-
	3	AE	40% (33-45%)		-	60% (55-67%)	33741.74	14129	1	.00	1.00
							33727.74	14129	2	5.67	.02
	4	E	-	-	-	100% (100-100%)	33915.46	14130	3	173.71	<.001
Covariance	1	ACE	54% (40-68%)	0% (0-0%)	-	46% (31-60%)	33741.74	14128	-	-	-
	2	ADE	27% (0-70%)	-	31% (0-79%)	42% (27-58%)	33722.06	14128	-	-	-
	3	AE	54% (40-68%)	-	-	46% (31-60%)	33741.74	14129	1	.00	1.00
							33723.58	14129	2	1.52	.22
	4	E	-	-	-	100% (100-100%)	33818.93	14130	3	77.18	<.001

MZ correlations for disgust proneness and contamination sensitivity (r 's = .42 and .44, respectively) were more than double DZ correlations (r 's = .11 and .15, respectively), as was the cross-twin-cross-trait correlation (i.e., the correlation between one twin's disgust proneness and the other twin's contamination sensitivity) (r 's = .17 and .07 for MZ and DZ twins, respectively). Consistent with these patterns, constraining C paths to zero in an ACE model did not diminish model fit (p 's = 1.00) – that is, we observed no effect of shared environment on disgust proneness, contamination sensitivity, or their covariance. We thus tested for dominance effects by constraining D paths to zero in an ADE model. None of the three constraints diminished model fit (p 's > .01). Constraining the additive genetic effects (A) to zero did diminish model fit (p 's < .001), so we retained an AE model (see Table 1 for model fit statistics).

Genetic factors accounted for 34% and 40% of the variance in disgust proneness and contamination sensitivity, respectively, with unique environmental factors accounting for the remaining variance (see Table 1 and Figure 1). The overlap between genetic influences on disgust proneness and contamination sensitivity (i.e., the genetic correlation) was .43, and the overlap between (unshared) environmental influences on the two traits (i.e., the nonshared environmental correlation) was .21. Fifty-four percent of the phenotypic correlation between the traits reflects genetic factors, and 46% reflects unshared environmental factors.

Discussion

Over two decades' worth of research has uncovered a relationship between disgust proneness and symptoms of anxiety disorders – especially contamination sensitivity. This is the first genetically-informed study to examine the relationship between the two traits, and it is thus uniquely situated to inform the hypothesis that disgust proneness arises from social transmission from parents to offspring, and that this social transmission leads to higher contamination

sensitivity (Olatunji et al., 2017; Widen & Olatunji, 2016). Twin modeling revealed no evidence that environmental factors shared by twins – which presumably include exposure to the same parents’ fastidious cleaning behavior, expressions of disgust, or controlling parenting style – underlie this covariance. Instead, 54% of the covariance was attributable to genetic factors, with the remainder accounted for by nonshared environmental factors.

At first blush, these results might seem at odds with the observations motivating the parental modeling hypothesis, including young children’s apparent absence of disgust and sense of contamination (Rottman, 2014), parent-offspring resemblance in contamination sensitivity (Rozin et al., 1984), and greater expressions of disgust from parents of younger children than parents of older children (Stevenson et al., 2010). Further reflection suggests that none of these findings strongly supports the parental modeling perspective. Many traits not present before age five develop independently of parental modeling (e.g., psychological changes during puberty). Even if exposure to others’ expressions of disgust influences the development of disgust proneness, then exposure to thousands of non-parents could overwhelm any parental effects (e.g., Askew et al., 2014). Most critically to this study, within-family similarities do not necessarily indicate that a phenotype is socially transmitted by parents – indeed, decades of behavioral genetics work shows that within-family similarities are largely the product of shared genetic rather than shared environmental influences (Plomin et al., 2016).

Naturally, results reported here should not be interpreted as discounting the importance of environmental influences on disgust proneness and contamination sensitivity. Indeed, over half of the variance in each trait was attributable to environmental factors that twins do not share (and measurement error), and some of these factors influenced both traits. These results suggest that, rather than searching for the parental behaviors that lead to high levels of disgust and

contamination sensitivity, researchers can instead focus their efforts on generating and testing hypotheses of environmental factors that create differences between siblings. We discuss two that have been generated via adaptationist perspectives (e.g., Tybur et al., 2013), but that have only been preliminarily tested. First, a history of infectious disease might promote stronger motivations to avoid pathogen cues, either via disgust responses or via discomfort with indirect social contact. Some work supports this hypothesis (Stevenson et al., 2009); other work does not (De Barra et al., 2014) (see Tybur et al., 2018, for a review). Second, having a child might either increase disgust proneness (given a protective effect on offspring) or decrease it (given the need to contact offspring bodily wastes). Preliminary evidence supports the latter hypothesis, with one study reporting that mothers are less disgust prone than childless women (Prokop & Fančovičová, 2016). Similar thinking might inform the genetic correlation between disgust proneness and contamination sensitivity, which could reflect a pleiotropic effect, where the same genes affect both traits. Across taxa, animals engage in multiple strategies to neutralize pathogens, including resistance (e.g., via inflammatory responses) and avoidance (e.g., via motivational states like disgust, or via behavioral avoidance). If individuals compensate for a deficit in the former by increasing the latter, then those genes underlying some aspects of immune function could simultaneously influence both the motivational states specialized for pathogen avoidance (i.e., disgust) and ruminations regarding routes of pathogen transmission. Alternatively, if greater disgust proneness leads to greater contamination sensitivity (Widen & Olatunji, 2016), then those genes underlying disgust proneness will partially also underlie contamination sensitivity. And, of course, the cross-sectional design employed here cannot rule out the possibility that contamination sensitivity – and the genes underlying it – causally

influences disgust proneness. Longitudinal designs would be required to distinguish between these accounts.

The current study also offers some insights into sex differences in the heritability of disgust. Of the two recent twin studies on disgust, one sampled only women (Sherlock et al., 2016), and the other sampled only 180 pairs of twins of both sexes (Olatunji et al., 2019). The former could not test for sex differences, and the latter concluded that trait disgust is heritable for women, but not for men. Importantly, these conclusions were limited by a modest number of male twins (41 MZ and 19 DZ male twin pairs), which afforded low power to detect moderate genetic effects (Verhulst, 2017), and an absence of formal tests of quantitative sex differences. With a much larger sample, we find no evidence for sex-specific heritabilities in disgust proneness, contamination sensitivity, or their covariance (see Supplementary Table 2). Hence, while disgust proneness and contamination sensitivity phenotypes differ across the sexes, the degree to which genes underlie these traits does not.

We note a few caveats to our conclusions. First, the methods employed here cannot speak to the possibility that within-family differences in disgust proneness stem from differences in how often children within the same household observe specific parental behaviors or how they react to those behaviors (Turkheimer & Waldron, 2000). Second, our decision to interpret AE over ADE models was based on a conservative criterion for differences in model fit ($p < .01$) (though further justified by the complexities of interpreting non-additive genetic effects from classical twin designs; e.g., Keller & Coventry, 2005). Third, we did not measure parents' disgust proneness or parenting style. Future work using more complex designs that also assess parental phenotypes and can simultaneously estimate dominant and additive genetic components and shared environment components, as well as correlations and interactions between those (e.g.,

nuclear twin family designs or children-of-twins design), could address these shortcomings. Finally, like most studies investigating the relationship between disgust proneness and contamination sensitivity, we sampled only adults. Hence, we cannot rule out the possibility that aspects of the shared environment influence children's disgust proneness or contamination sensitivity, but that these influences dissipate in adulthood, just as shared environment effects for so many other characteristics do (Bergen et al., 2007). Genetically informed studies of children would be necessary to compare shared environment effects in childhood versus adulthood.

Concluding thoughts

According to a recent meta-analysis (Olatunji, 2017), no study had investigated the relationship between disgust proneness and anxiety disorder symptoms prior to 1996. In just over 20 years since then, at least 83 published studies have done so, and they have shown that disgust-prone individuals have higher anxiety-related symptoms, most notably contamination sensitivity. Generating and testing hypotheses to explain why these traits covary is a necessary step toward leveraging the psychology of disgust to better understand psychopathology (Amaroso et al., 2019). While the current study suggests that aspects of the shared environment do not underlie disgust proneness, contamination sensitivity, or their covariance, it offers directions for multiple other tests of the environmental and genetic underpinnings of disgust and its related disorders.

Author contribution note

J.M.T. developed the study concept. P.J. coordinated all data collection efforts. L.W.W. conducted analyses, and J.M.T. and P.J. provided feedback on these analyses. J.M.T. drafted the manuscript, and L.W.W. and P.J. contributed to revisions. All authors approved the final version of the paper for submission. We are grateful to Annika Karinen and Kimmo Karinen for assistance in translating research instruments.

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Open data and materials note

Finnish and English versions of disgust proneness and contamination sensitivity items are available at osf.io/wmshv/, as are the data and OpenMX scripts used for twin modeling.

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Supplementary Materials

Zygosity estimation

Information on zygosity was available for the 2,371 twins (34.8%) who had participated in earlier surveys (1,342 of these twins were genotyped earlier, and zygosity estimates for the remaining 1029 were based on items inquiring about physical resemblance, see Johansson 2013). For the remaining same-sex twins, we determined zygosity using items based on the Peas in a Pod Questionnaire (PPQ), which inquires about physical resemblance. Several studies have shown that this method yields excellent accuracy when validated using DNA polymorphisms (ranging between 89 to 93%) (Christiansen, 2003; Johansson, 2013; Jarrar, 2018; Sarna, 1978; Rietveld, 2000). Here, participants were first asked whether each of four categories of people (parents, siblings, classmates and strangers; two points per negative response) had difficulties telling them and their twin apart. They then reported how much they and their twin looked alike at school age, with options including “two peas in a pod” (0 points), “like normal siblings” (2 points) and “cannot say” (1 point). Sum scores between 0 and 4 were classed as monozygotic (MZ), but only if the respondent indicated similar hair and eye color to his or her twin during school age. Scores between 8 and 10 were classed as dizygotic (DZ) and scores between 5 and 7 were classed as unknown. In the complete pairs, if one of the twins had an unknown zygosity estimate (8.7%), we selected zygosity based on the co-twin’s responses. Zygosity estimates were inconsistent for 6.9% of twins, and both twins yielded an unknown zygosity estimate in 0.8% of cases. A recent study found that 94.7% of unknown estimates based on the PPQ method were DZ twins (Jarrar, 2018); this result is in line with Johansson (2013), who found that the majority of PPQ misclassifications occurred when genotyped DZ twins were estimated as MZ. We therefore classified twins with contrasting or unknown zygosity estimates as DZ.

Analyses with entire Three-Domain Disgust Scale (TDDS)

A principal-axis factor analysis on the 21 TDDS items (seven each from the pathogen, sexual, and moral domains) and 10 Padua Inventory contamination items (31 items total) yielded a scree plot based on the factor analysis yielded a clear four-factor solution, with the top 10 eigenvalues equal to 7.03, 4.19, 2.17, 1.66, 1.07, .97, .87, .82, .79, and .74. The pattern matrix (based on extracting and obliquely rotating four factors) revealed that all seven pathogen disgust items loaded saliently on one factor (primary loadings between .43 and .69, no cross-loadings greater than .3), all 10 Padua items loaded saliently on one factor (primary loadings between .55 and .78, no cross-loadings greater than .3), all seven moral disgust items loaded saliently on one factor (primary loadings between .47 and .72, no cross-loadings greater than .3) and all seven sexual disgust items loaded saliently on one factor (primary loadings between .45 and .75, one cross-loadings of .3). These analyses suggest that endorsements of disgust toward pathogen cues are distinct from contamination sensitivity, and they are further distinct from endorsements of disgust toward items with sexual and moral content.

The regression analysis indicated that the zero-order relationship between pathogen disgust and contamination sensitivity ($r = .32$) was similar in magnitude ($r_p = .26$) when we controlled for the sexual disgust and moral disgust (along with participant sex and age). Unique relationships between sexual disgust and contamination sensitivity and moral disgust and contamination sensitivity were $r_p = .04$ and $r_p = -.06$, respectively. These results again suggest that the phenotypic correlation reported in the manuscript does not reflect a general relationship between contamination sensitivity and disgust toward disgust elicitors; it instead reflects a relationship between contamination sensitivity and disgust toward pathogen cues.

Supplementary Table 1. Mean scores for male and female twins and siblings (standard deviations), correlations within MZ and DZ twins (95% confidence intervals) and between siblings and twins, and cross-twin-cross-trait and cross-sibling-cross-trait correlations (95% confidence intervals) for disgust proneness (1 to 7 scale) and contamination sensitivity (0 to 4 scale).

	Disgust proneness	Contamination sensitivity
Means		
<i>Male twins</i>	3.75 (1.11)	1.38 (.53)
<i>Female twins</i>	4.27 (1.13)	1.54 (.64)
<i>Male siblings</i>	3.57 (1.12)	1.36 (.48)
<i>Female siblings</i>	4.38 (1.09)	1.60 (.70)
Twin correlations		
<i>MZm</i>	.47 (.33-.58)	.39 (.22-.51)
<i>DZm</i>	.02 (-.17-.21)	.16 (-.14-.39)
<i>MZf</i>	.41 (.32-.48)	.45 (.37-.52)
<i>DZf</i>	.19 (.08-.29)	.17 (.07-.26)
<i>DOS</i>	.05 (-.06-.16)	.12 (-.00-.24)
<i>Total MZ</i>	.42 (.35-.49)	.44 (.37-.50)
<i>Total DZ</i>	.11 (.03-.18)	.15 (.07-.23)
Sibling correlations		
<i>Brothers</i>		
<i>With male twin</i>	.06 (-.15-.25)	-.02 (-.27-.24)
<i>With female twin</i>	.09 (-.04-.22)	.03 (-.09-.14)
<i>Sisters</i>		
<i>With male twin</i>	.13 (.02-.23)	.20 (.10-.30)
<i>With female twin</i>	.15 (.03-.26)	.19 (.08-.29)
<i>Total siblings</i>	.12 (.05-.18)	.14 (.07-.20)
Cross-twin-cross-trait correlations		
<i>MZ</i>	.17 (.12-.23)	
<i>DZ</i>	.07 (.02-.13)	
<i>Siblings</i>	.13 (.09-.18)	

Supplementary Table 2. Test statistics analyzing sex differences in twins. Nested models were compared with saturated ones, which included sex-specific means, standard deviations, and correlations. Means and standard deviations were constrained to be equal across the sexes in Models 2 and 3, respectively. In Model 4, correlations between monozygotic males (MZ_{δ}) and females (MZ_{φ}) and dizygotic males (DZ_{δ}) and females (DZ_{φ}) were constrained to be equal (quantitative sex differences). In Model 5, correlations between same-sex dizygotic (DZ) twins and opposite sex (DOS) twins were constrained to be equal (qualitative sex differences).

		Model	-2LL	df	Compared to model	χ^2 (df)	<i>p</i> -value
Disgust proneness	1	Saturated	18314.6	6055	-	-	-
	2	Mean $_{\varphi}$ = Mean $_{\delta}$	18682.74	6056	1	368.13 (1)	<.001
	3	SD $_{\varphi}$ = SD $_{\delta}$	18314.64	6056	1	.03 (1)	.86
	4	MZ $_{\varphi}$ =MZ $_{\delta}$ & DZ $_{\varphi}$ =DZ $_{\delta}$	18317.86	6057	3	3.26 (2)	.20
	5	DZ=DOS	18319.87	6058	4	2.00 (1)	.16
Contamination sensitivity	1	Saturated	11578.34	6318	-	-	-
	2	Mean $_{\varphi}$ = Mean $_{\delta}$	11717.00	6319	1	138.66 (1)	<.001
	3	SD $_{\varphi}$ = SD $_{\delta}$	11702.38	6319	1	124.04 (1)	<.001
	4	MZ $_{\varphi}$ =MZ $_{\delta}$ & DZ $_{\varphi}$ =DZ $_{\delta}$	11578.95	6320	1	0.62 (2)	.74
	5	DZ=DOS	11579.33	6321	4	0.38 (1)	.54
Covariance	1	Saturated	29362.12	12373	-	-	-
	4	MZ $_{\varphi}$ =MZ $_{\delta}$ & DZ $_{\varphi}$ =DZ $_{\delta}$	29364.87	12375	1	2.75 (2)	.25
	5	DZ=DOS	29365.48	12376	4	.61 (1)	.43

Supplementary Table 3. Test statistics evaluating sex differences in sibling correlations and differences between sibling correlations and DZ correlations. Nested models were compared with saturated ones, which included sex-specific correlations. Model 2 constrained sibling correlations with male twins to be equal to sibling correlations with female twins, Model 3 constrained male sibling correlations with twins to be equal to female sibling correlations with twins, and Model 4 constrained sibling correlations to be equal to DZ twin correlations.

		Model	-2LL	df	Compared to model	χ^2 (df)	<i>p</i> -value
Disgust proneness	1	Saturated twins and siblings	19303.07	6318	-	-	-
	2	Siblings with $\text{twin}_{\text{♀}} = \text{twin}_{\text{♂}}$	19303.20	6320	1	.13 (2)	.94
	3	$\text{Siblings}_{\text{♀}} = \text{Siblings}_{\text{♂}}$	19303.85	6321	2	.65 (1)	.42
	4	Siblings=DZ	19303.93	6322	3	.08 (1)	.77
Contamination sensitivity	1	Saturated twins and siblings	11740.96	6555	-	-	-
	2	Siblings with $\text{twin}_{\text{♀}} = \text{twin}_{\text{♂}}$	11741.14	6557	1	0.17 (2)	.92
	3	$\text{Siblings}_{\text{♀}} = \text{Siblings}_{\text{♂}}$	11748.24	6558	2	7.10 (1)	.01
	4	Siblings=DZ	11748.27	6559	3	0.35 (1)	.85
Covariance	1	Saturated twin and siblings	33782.33	14123	-	-	-
	4	Siblings=DZ	33785.94	14124	1	3.61 (1)	.06

Supplementary Figure 1. Power curve for the common environmental variance component set at 20% as the level of the additive genetic variance component varies (10%, 30% and 50%). These curves underestimate the current studies power to detect common environment effects for two reasons. First, they assume an equal number of MZ and DZ twins. Power to detect an effect of the common environment is higher when there are more DZ than MZ twins (Verhulst, 2017), as in our sample. Second, power to detect common environmental factors is higher when non-twin siblings are added to the bivariate twin model (Posthuma & Boomsma, 2000), as in the current study.

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