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The Quantitative Genetics of Disgust Sensitivity

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Response sensitivity to common disgust elicitors varies considerably among individuals. The sources of these individual differences are largely unknown. In the current study, we use a large sample of female identical and nonidentical twins (N = 1,041 individuals) and their siblings (N = 170) to estimate the proportion of variation due to genetic effects, the shared environment, and other (residual) sources across multiple domains of disgust sensitivity. We also investigate the genetic and environmental influences on the covariation between the different disgust domains. Twin modeling revealed that approximately half of the variation in pathogen, sexual, and moral disgust is due to genetic effects. An independent pathways twin model also revealed that sexual and pathogen disgust sensitivity were influenced by unique sources of genetic variation, while also being significantly affected by a general genetic factor underlying all 3 disgust domains. Moral disgust sensitivity, in contrast, did not exhibit domain-specific genetic variation. These findings are discussed in light of contemporary evolutionary approaches to disgust sensitivity.

Keywords: individual variation, behavioral genetics, evolutionary psychology, pathogen threat, sexual strategies

Contemporary approaches to disgust typically employ an evolutionary perspective to understand the adaptive function and origin of the emotion (Chapman, Kim, Susskind, & Anderson, 2009; Curtis, de Barra, & Aunger, 2011; Kelly, 2011; Oaten, Stevenson, & Case, 2009; Rozin, Haidt, & McCauley, 2008; Tybur, Lieberman, & Griskevicius, 2009; Tybur, Lieberman, Kurzban, & DeScioli, 2013). Such approaches frequently suggest that disgust does not have a single, general adaptive function, but can rather be divided into domains with distinct functions. For example, Tybur et al. (2009) proposed that pathogen, sexual, and moral disgust each constitute functionally specialized disgust domains, meaning that they are elicited by different types of cues, moderated by different types of contextual factors, and specialized for neutralizing different types of adaptive problems that were reliably present in the ancestral environment. Specifically, pathogen disgust is posited to motivate the avoidance of physical contact with infectious microorganisms, sexual disgust is posited to motivate the avoidance of fitness-reducing sexual behaviors, and moral disgust is posited to mitigate the costs imposed by others' violations of social rules (for more detail, see Tybur et al., 2013).

The upsurge in recent evolutionary work on disgust has been paralleled by work investigating individual differences in a trait called *disgust sensitivity*, which refers to the degree to which individuals experience disgust in response to common disgust elicitors. Researchers have become interested in disgust sensitivity partly because it varies with traits ranging from psychopathology (see Davey, 2011 for a review; de Jong & Merckelbach, 1998; Mancini, Gragnani, & D'Olimpio, 2001; Olatunji et al., 2007), to political ideology (Inbar, Pizarro, & Bloom, 2008; Tybur, Merriman, Caldwell Hooper, McDonald, & Navarrete, 2010), to phenomena such as stigmatization (Inbar, Pizarro, Knobe, & Bloom, 2009; Lieberman, Tybur, & Latner, 2012), ethnocentrism (Navarrete & Fessler, 2006), and mate preferences (Jones et al., 2013; Lee et al., 2013).

In addition to investigating how disgust sensitivity relates to these traits, a good deal of this work has been aimed at understanding the dimensionality of disgust sensitivity itself (Haidt, McCauley, & Rozin, 1994; Olatunji et al., 2007; Tybur et al., 2009). Patterns of individual differences in sensitivity to different disgust elicitors have been shown to relate to each other in ways consistent with the adaptationist theory outlined previously. For example, in their initial development of the Disgust Scale, Haidt et al. (1994) found that although self-reports of disgust toward a wide variety of pathogen sources (e.g., corpses, spoiled foods, bodily wastes, interpersonal contact) strongly covaried with each other, they did not covary with disgust toward moral violations. In their

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modification of the Disgust Scale, Olatunji et al. (2007) found that many disgust responses clustered into three highly correlated factors (*r*s = .75, .88, and .65), each of which describe cues to pathogens (e.g., bodily wastes, contact with corpses, interpersonal contact). However, the sexual domain that was included in the original Disgust Scale did not covary strongly with these other factors, and it was eliminated from the revised Disgust Scale. Finally, Tybur et al. (2009) conducted factor analyses on a large number of disgust elicitors that were nominated by participants. A three-factor structure emerged, and these three factors appeared to reflect pathogen, sexual, and moral items. Rather than eliminating the sexual and moral items because they did not covary with the pathogen items, as had been done with previous instruments, Tybur et al. (2009) developed the Three-Domain Disgust Scale (TDDS), a 21-item instrument that measured each of these three factors.

Subsequent work has demonstrated that sex differences and correlations with personality dimensions are consistent with predictions drawn from adaptationist models (e.g., Tybur, Bryan, Lieberman, Caldwell Hooper, & Merriman, 2011; Tybur & de Vries, 2013). As would be expected, openness to experience is negatively related to pathogen and sexual disgust (Tybur et al., 2011). Further, women score much higher on the sexual factor of the TDDS, but they score only slightly higher on the pathogen and moral factors (Tybur et al., 2009, 2011). These differences in sexual disgust are thought to reflect discrepancies in the fitness costs between males' and females' mate choice (Trivers, 1972).

At a fundamental level, investigations into the dimensionality of disgust sensitivity—and the correlation between domains of disgust sensitivity and various other traits of interest—concerns between-individual variation. In contrast, evolutionary approaches to human behavior—including those applied to understanding disgust (e.g., Tybur et al., 2013)—have tended to focus on universals, or evolved mechanisms that calibrate each individual to their specific conditions or environmental circumstances. As such, evolutionarily informed theories of the source of individual differences has been limited and generally oriented toward environmentally induced variation (Zietsch, de Candia, & Keller, 2014). Hence, despite the upsurge in evolutionarily oriented work on disgust sensitivity, little progress has been made in understanding what causes variability between individuals, including the possible role of genetic factors. Exploring the underlying causes of this variation can provide new information regarding the nature of disgust and potentially shed light on processes leading to variation in other related traits.

What Gives Rise to Variability in Disgust Sensitivity?

Some researchers have argued that variability in disgust sensitivity is entirely due to environmental factors, whereas others have proposed that variation may be partly heritable, that is, caused by variation in genes. Researchers favoring a purely environmental account have suggested that differences in disgust sensitivity across individuals result from social transmission during formative years (Kim, Ebesutani, Young, & Olatunji, 2013; Rozin et al., 2008). Similarly, others have argued that culture provides the framework for variation in sensitivity to contaminants (Siegal, Fadda, and Overton (2011). Children from Western countries are more likely to identify germs as a cause for illness transmission (Siegal, Pat, & Eiser, 1990), whereas individuals from non-Western countries avoid contamination because of tradition, familiarity, or social cohesion (Rogers, 1995). Interestingly, children with autism, who have impaired social learning, experience delays in developing disgust, whereas children with other development disorders do not (Kalyva, Pellizzoni, Tavano, Iannello, & Siegal, 2010). This might point to a role of individual differences in socialization in the development of disgust sensitivity.

Supporting environmental perspectives, researchers have found that parents and children score similarly on measures labeled as "food contamination," which has similar item content to disgust sensitivity instruments, for example, "On a 9-point scale how much would you like to eat soup from a thoroughly washed dog bowl?" (Rozin, Fallon, & Mandell, 1984). Specifically, Davey, Forster, and Mayhew (1993) report that correlations between parents and offspring on these items range from .33 to .52. However, influences of genetic and environmental factors are confounded in studies that simply observe phenotypic correlations between parents and offspring. Such correlations can stem from genetic factors, environmental factors, or a combination of the two. Children could score similarly on food contagion sensitivity because they share genes with their parents, or they might simply acquire similar sensitivity through observation of their parent's behavior (or other parentally mediated learning processes; Davey et al., 1993).

Studies of twins can distinguish between genetic and shared environmental effects, as family environment factors are assumed to affect twin pairs equally, whereas genetic effects will vary due to differential genetic similarity between identical and nonidentical twins (100% vs. 50%, respectively). Twin studies of blood-injury phobias, of which disgust responses are a key symptom (Cisler, Olatunji, & Lohr, 2009; Olatunji, Cisler, McKay, & Phillips, 2010), might hint at the presence of heritable basis to pathogen disgust. Neale et al. (1994) found a higher degree of heritability in fear of blood (56% of variance) in a large sample of twins. Similarly, Fear Survey Schedule II data collected from twin samples have shown higher concordance rates for identical twins on items relating to blood, injury, and needles (Rose & Ditto, 1983).

In contrast with these hints at genetic effects, the only study that has used a twin design to test for genetic versus environmental effects on a disgust instrument has supported a pure environmental perspective. Rozin and Millman (1987) investigated the similarity of food contamination disgust between identical and nonidentical twins. Participants in this study indicated how much they would like to eat a contaminated food source on a 9-point scale. The study showed that the correlation between identical twins' food contamination disgust sensitivity ($r = .29$) was not significantly different from nonidentical twins' scores (Rozin & Millman, 1987). The authors interpreted these results as suggesting that variability in food contagion disgust has no genetic component and is, hence, entirely caused by environmental factors (Rozin et al., 2008; Rozin & Millman, 1987). However, Rozin and Millman's (1987) initial study of heritability was conducted with fewer than 40 identical and nonidentical twin pairs. Further, analytical methods available at the time did not yield standard errors and confidence intervals; such statistics would have shown that little could be concluded about the relative magnitude of genetic and environmental effects from a sample of that size.

The Current Investigation

There have been no studies to date that have effectively disentangled environmental and genetic sources of variability in disgust sensitivity. Without basic knowledge of how variability in disgust sensitivity arises, it will be difficult to maximize the knowledge that can be gleaned from the impressive body of research on the topic. In the current study, we aim to provide such basic knowledge using a large sample of identical and nonidentical twins (N = 1,041 individuals) and their siblings (N = 170) to estimate the proportion of variation in pathogen, sexual, and moral disgust sensitivity that is due to genetic effects, the family environment, and other (residual) sources.

In addition, we also investigate the genetic and environmental influences on the covariation between the different disgust domains. This allows us to compare the phenotypic factor structure of disgust sensitivity with the underlying genetic architecture. We use multivariate twin modeling to estimate the extent to which each disgust domain is influenced by specific versus common genetic factors; this can inform the degree to which covariation between pathogen, sexual, and moral disgust sensitivity arises from common genes versus specific genes.

Method

Participants

The statistical analyses in the present study were performed on a sample of 1,903 female twins and siblings of twins (mean age = 33.12, SD = 4.99). This is a subsample of the population-based Genetics of Sexuality and Aggression twin sample in Finland (see Johansson et al., 2013). Data were collected in the fall of 2013, targeting women who had participated in a similar data collection in 2006, and who indicated that they would be interested in participating in survey studies in the future. We were unable to add disgust sensitivity instruments to the twin survey before data were collected on males and, hence, data were only collected on females. All data were collected through a secure online questionnaire. In total, we sent invitations to 5,197 women by postal mail. Individuals who did not respond in any way over the first 2 weeks were sent a reminder letter, followed by another reminder letter another 2 weeks later unless they responded after the first reminder. Twenty-three individuals could not be reached (because the intended recipient had, e.g., moved abroad or passed away after their addresses were obtained from the Central Population Registry of Finland). In total, 2,249 women responded, and of these, 73 individuals did not wish to participate. Thus, the final response rate was 43.5%. An additional 273 women did not complete the necessary parts of the questionnaire, resulting in the final sample of 1,903 women.

The invitation to participate in the study was accompanied by a letter explaining the voluntary nature of the study. Potential participants were informed that they are free to terminate their participation at any stage of the study without providing a reason. Written informed consent was obtained from all participants. The Ethics Committee of the Abo Akademi University (Turku, Finland) approved the research plan in accordance with the Declaration of Helsinki (World Medical Association, 2013).

For the purposes of genetic analyses, pathogen, moral, and sexual disgust sensitivity data were available from 544 identical (mean age = 32.56, SD = 2.83) and 497 nonidentical (mean age = 32.6, SD = 2.84) twins. Data were also obtained from 88 and 82 siblings of monozygotic (MZ; mean age = 33.17, SD = 1.62) and dizygotic (DZ; mean age = 33.17, SD = 1.80) twins, respectively. Zygosity was determined using DNA (Johansson et al., 2013). Only data that were available for both twins were used to estimate genetic effects; however, all available data were used to estimate means, variances, and within-person between-trait covariances via full information maximum likelihood modeling.

Measures

The TDDS (Tybur et al., 2009) is a 21-item measure composed of pathogen, sexual, and moral factors. Each item describes an act, concept, or situation that typically arouses some degree of disgust in individuals. Participants rate the degree to which they find each item disgusting on a 0- to 6-point scale. Items were translated into Finnish and a panel of four individuals with excellent command of both languages subsequently reviewed the translations. Consistent with previous versions in English, Dutch, and Japanese (e.g., Quintelier, Ishii, Weeden, Kurzban, & Braeckman, 2013; Tybur & de Vries, 2013), each of the subscales had acceptable internal consistency ($\alpha > .75$), and the subscales were modestly intercorrelated ($\sim$.30 to .35). Mean scores on TDDS Pathogen, Sexual, and Moral Disgust subscales, respectively, were 3.38 (SD = 1.09), 2.89 (SD = 1.17), and 4.84 (SD = 0.90).

Statistical Analyses

Genetic analyses of the data were conducted in R (R Core Team, 2014) using maximum likelihood modeling procedures contained in the statistical package OpenMx (Boker et al., 2011). We controlled for the mean effects of age by including it as a covariate in all genetic analyses. Maximum-likelihood modeling in OpenMx uses chi square as an indicator of goodness of fit to the data. The change in chi square is compared against change in degrees of freedom when parameters are estimated or constrained within the model (e.g., fixing them at zero, or equating different parameters) to determine the optimal model.

Estimating Genetic and Environmental Effects on Traits

The classical twin design allows variation in a trait to be partitioned into genetic (A and D), shared environmental (C), and residual (E) sources (Neale & Cardon, 1992). Genetic effects themselves may result from additive variation (the sum of allelic effects within and across genes, i.e., A) or nonadditive variation (allelic interactions such as dominance and epistasis, i.e., D). The proportion of variation in a trait due to additive genetic factors is the narrow sense heritability (h^2), and the proportion of variance accounted for by all genetic factors is (additive plus nonadditive) the broad-sense heritability (H^2). Shared environmental influences are those shared between twins; these effects will cause both identical and nonidentical twins to become more similar to each other. Residual effects may be due to unique and idiosyncratic experiences not shared between the twins, measurement error, or stochastic (chance) biological effects (e.g., mutations, neoplastic transformations and cancer). The ability to partition variance in

phenotypes into these components is possible because identical (MZ) twins are genetically identical, whereas nonidentical (DZ) twins share only half of their segregating genes. For example, if additive genetic influences were the only cause of variation in a trait, one would expect a correlation of 1.0 between MZ twin pairs and .5 for DZ twins. Further, if nonadditive genetic sources were exclusively underlying trait variation, MZ correlations would be expected to be 1.0, whereas DZ pairs would correlate at a maximum of .25 (Posthuma et al., 2003). Nonadditive genetic and shared environmental effects are confounded in the classical twin model and are unable to be estimated at the same time.

The classic twin model can be extended to a multivariate model, allowing a decomposition of variance sources over multiple traits. The multivariate model uses cross-twin and cross-trait correlations in order to partition trait covariance in the same way in which variance is partitioned in a univariate twin model. In addition, a multivariate model allows for the partitioning of an observed correlation between two variables in genetic and environmental components. Nontwin siblings can also be included in the model, which enhances statistical power (Dolan, Boomsma, & Neale, 1999; Posthuma & Boomsma, 2000).

Results

Preliminary Analyses

As expected, there were no significant differences in mean disgust sensitivity scores between MZ twins, DZ twins, and nontwin siblings. Moreover, no significant differences were observed between the correlations of nonidentical twin pairs and sibling pairs, except in the case of moral disgust sensitivity, for which the correlation between nonidentical twin pairs was weaker than the correlation between sibling pairs. Given that there is no plausible reason for a real effect in this direction, and given the numerous statistical tests that were conducted in the preliminary testing, this was presumed to be due to sampling error and these correlations were equated in subsequent analyses. Identical twin pairs were more similar than nonidentical twin pairs (see Table 1) across the disgust domains (pathogen, $p = <.001$; sexual, $p = .058$; and moral, $p = <.001$), indicating genetic effects on all three domains of disgust sensitivity. Indeed, for each domain, identical twin pair correlations were more than double the nonidentical twin pair correlations, indicating no *C* variance. This means that there is no evidence that any shared environmental factors influence disgust sensitivity. As per standard practice in such circumstances, we fitted *ADE* models instead of *ACE* models.

Estimating Genetic and Environmental Effects on Traits

Variance components for each trait (see Table 2) were estimated from univariate genetic models. All three disgust domains were then fitted to a trivarate Cholesky ADE model. Although the estimates of *D* were nonzero for sexual and moral disgust domains, dropping *D* from the model did not have a significant effect on model fit ($\chi^2 = 4.36$, $p = .63$). As such, and for the sake of simplicity, we interpret the *AE* model in the knowledge that any nonadditive genetic effects *D* are absorbed into the *A* estimate, which will therefore represent the broad sense heritability of each trait (Keller, Medland, & Duncan, 2010).

The multivariate analysis revealed that genetic effects influenced the observed (phenotypic) correlation between the three disgust domains (see Table 3). As can be seen, the three domains correlated positively and moderately together. The proportion of correlations between the disgust domain phenotypes due to genetic correlation can also be seen in Table 3. A genetic correlation indicates the extent of overlap in the genetic variation of any pair of traits, directly analogous to phenotypic correlation, which indicates the extent of overlap in observed variation of any pair of traits. Genetic correlations can be high even if the heritability of a trait is low, because correlations only indicate the overlap in genetic effects and not their magnitude. The same principles apply to residual correlations.

To further assess common and specific sources of variance in the three disgust domains, we fitted an independent pathways model to the data (see Figure 1). This model parameterizes variation in all three disgust domains as stemming from both common and specific sources of additive genetic and residual variance. As this model is not nested within the Cholesky trivariate model, the fit to the data could not be directly compared. Instead, we compared the models' Akaike information criterion (AIC), which allows for comparisons of non-nested models by weighing goodness-of-fit and parsimony. The AIC was equivalent between the two models, indicating equal suitability for the data. To test whether the data could be modeled even more parsimoniously, we fitted a common pathways model. This model predicts that genetic and environmental variances influence covariation between the disgust domains via a latent factor. As the common pathways model is nested within the independent pathways model (see Gillespie & Martin, 2005), we compared model fit using likelihood ratio chi-square statistics. This common pathways model fit the data significantly worse than the independent pathways model further; this indicates that genetic and environmental factors have

Table 1
Twin-Pair and Twin-Sibling Correlations for Disgust Sensitivity Domains

Zygosity	*r*[95% CI]		
	Pathogen	Sexual	Moral
Identical twin pairs ($n = 131$)	.49 [.36, .59]	.41 [.28, .52]	.50 [.37, .60]
Nonidentical twin pairs ($n = 100$)	.23 [.07, .36]	.20 [.02, .35]	−.12 [−.32, .11]
Sibling pairs ($n = 73$)	.19 [−.04, .40]	.31 [.10, .48]	.39 [.17, .55]
Nonidentical twin and sibling pairs equated ($n = 173$)	.22 [.09, .34]	24 [.11, .36]	.11 [−.04, .26]

Note. CI = confidence interval.

Table 2
Estimates (and 95% Confidence Intervals) of the Proportion of Variance in Disgust Sensitivity Accounted for by Additive Genetic (A), Nonadditive Genetic (D), and Residual (E) Sources

	Pathogen	Sexual	Moral
A	.50 [.00, .60]	.44 [.00, .56]	.00 [.00, .51]
D	.00 [.00, .55]	.02 [.00, .54]	.55 [.01, .65]
A + D	.50 [.37, .61]	.46 [.34, .57]	.55 [.42, .65]
E	.50 [.39, .63]	.54 [.43, .66]	.45 [.35, .58]

Note. The sum of A and D indicates broad-sense heritability estimates for each domain.

different effects on covariance between each disgust domain, $\chi^2(\Delta df = 2) = 6.6$, $p = .04$. As such, we interpret the better-fitting independent pathways model (see Figure 1).

Parameter estimates of the independent pathway model showed that genetic variation in moral disgust sensitivity was primarily common (i.e., shared by all three disgust domains), whereas genetic variation in both pathogen and sexual disgust sensitivity was primarily specific to each domain. Equating moral disgust's specific genetic path to that of pathogen and sexual disgust resulted in significantly worse model fit, $\chi^2 = 4.74$, $p = .03$, and $\chi^2 = 5.88$, $p = .02$; this suggests that the amount of genetic variance accounted for by specific and common genetic factors indeed was different for the moral domain and the other two domains.

Discussion

Using a large sample of female twins and their siblings, we observed that individual differences in disgust sensitivity are substantially heritable. We detected no significant effect of the shared environment of the twins. Genetic effects accounted for approximately 50% of the variation between individuals across pathogen, sexual, and moral disgust domains. All domains share a common genetic influence, which accounted for approximately 18%, 11%, and 41% percent of the variance in pathogen, sexual, and moral disgust sensitivity, respectively. Sensitivity to sexual and pathogen—but not moral—disgust was also subject to specific genetic influences. We note that any measurement error contributes to estimates of residual variance; this suggests that, if anything, our model likely underestimates the proportion of variance due to genetic factors. Notably, these findings stand in direct contrast to the only previous investigation of twin similarity in disgust measures (Rozin & Millman, 1987). This discrepancy might be explained by the low power to detect genetic effects in Rozin and Millman's (1987) study. In sum, we show, for the first time, substantial genetic effects on individual differences in disgust sensitivity.

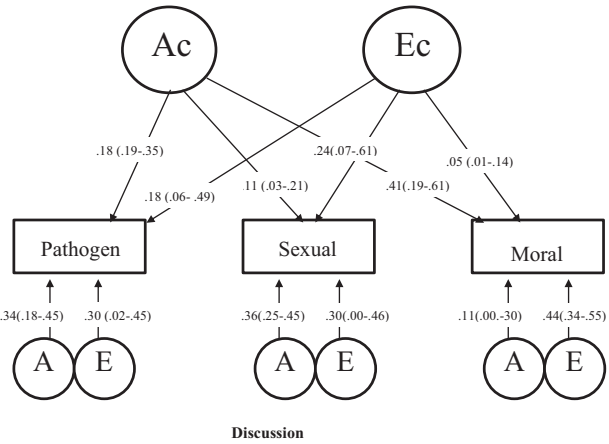

Figure 1. Path diagram of a trivariate *AE* independent pathway model of moral, pathogen and sexual disgust, with squared path coefficients and 95% confidence intervals. Squared path coefficients represent the proportion of variance in an observed trait accounted for by the latent factor from which the path originates. Ac and Ec represent common sources of genetic (*A*) and residual variance (*E*). *A* and *E* are sources of variance specific to each trait.

The finding of heritable variation in disgust sensitivity runs against a weight of opinion supporting an entirely environmentally mediated development of disgust (Kim et al., 2013; Rozin et al., 2008; Rozin & Millman, 1987). Indeed, the environment shared by twins was estimated to account for almost none of the variation in any disgust phenotype. This would include various sources of parental transmission, suggesting that food contagion correlations between parents and offspring found by Davey et al. (1993) were likely due to shared genes between parents and offspring. The influence of parental style, socioeconomic status, schooling and neighborhood type (i.e., urban or rural), household cleanliness, family pet-keeping, and so on, would also be captured by the shared environment of the twins, so these effects likely do not strongly influence disgust sensitivity.

As with pathogen disgust, the environment shared by twins had little effect on variation in sexual or moral disgust. Religiosity and political affiliations tend to be similar within all members of the family and show substantial variation due to the shared environment of twins (Hatemi, Alford, Hibbing, Martin, & Eaves, 2008;

Table 3
Phenotypic, Genetic, and Residual Correlations and Proportion of Phenotypic Correlation Between Disgust Domains Due To Genetic Correlations

	Pathogen-Sexual	Pathogen-Moral	Sexual-Moral
Phenotypic correlation	.35**	.33**	.30**
Genetic correlation	.28*	.53*	43*
Residual correlation	.41*	.20*	.22*
Proportion of phenotypic correlation due to genetic correlation	.40*	.74*	.65*

* $p < .05$. ** $p < .01$.

Kendler & Myers, 2009). Given the relationship between disgust sensitivity and political ideology (and, specifically, sensitivity to sexual disgust; see Inbar, Pizarro, Iyer, & Haidt, 2012; Tybur et al., 2010), it might have been expected that these influences would inform sensitivity to sexual and moral disgust. However, recent arguments have suggested that sentiments related to sexual behaviors and how resources are divided between individuals cause political and religious stances, rather than vice versa (Weeden & Kurzban, 2014). Variation in political ideology might stem from variables with no shared environment influence (e.g., disgust sensitivity) as well as factors influenced by shared environment (e.g., coalitional membership).

Up to this point, there had been no direct evidence that variation in disgust sensitivity might be caused by genes. However, sensitivity to pathogen disgust has been proposed to link with immune system function (Fessler & Navarrete, 2003; Fleischman & Fessler, 2011), which is largely heritable (for example, 53% to 86% across various cytokines; de Craen et al., 2005). Natural and sexual selection in the ancestral environment were frequently driven by the recurring threat of infectious microbes (Maynard Smith, 1978; Tooby, 1982), and direct evidence adaptation to these pervasive threats has been observed in the human genome (Fumagalli et al., 2011). Individuals who are more susceptible to infectious disease (e.g., through compromised immune function) should invest more effort in avoiding cues to pathogens, perhaps by being more disgusted by them. As such, genetic variation in sensitivity to pathogen disgust may to some extent reflect "reactive heritability" (Tooby & Cosmides, 1990), that is, indirect heritability due to calibration to a directly heritable trait (such as immune function).

The causes of variation in sexual disgust sensitivity had been similarly opaque to those underlying pathogen disgust. Tybur et al. (2013) posit sexual disgust as a coopted form of pathogen disgust adapted to avoid detrimental sexual partners. Variation in sociosexuality (orientation toward uncommitted sexual relationships) and number of sexual partners have both been shown to have substantial heritable components (~50% to 60%; Bailey, Kirk, Zhu, Dunne, & Martin, 2000; Zietsch et al., 2008). These behaviors also strongly correlate with variation in sensitivity to sexual disgust (Al-Shawaf, Lewis, & Buss, 2015; Tybur, Inbar, Güler, & Molho, 2015). As such, the genetic variation in sensitivity to sexual disgust that we have observed could, like sensitivity to pathogen disgust, reflect reactive heritability, with individuals following more short-term sexual strategies necessarily exhibiting less disgust toward sexual activities (Gangestad & Simpson, 2000). Alternatively, it may be the case that sexual disgust sensitivity drives sexual strategy.

In terms of moral disgust, it might have been expected that variation in individuals' reactions to third parties' breaches of moral standards are largely a product of the environment in which they are raised, perhaps due to the combined influences of their family's education, religion, and political beliefs. However, our finding of substantial heritable variation in sensitivity to moral disgust—and no shared environmental influence—aligns with previous research demonstrating that various moral sentiments are influenced by genetic variation (Brandt & Wetherell, 2012; Eaves, Eysenck, & Martin, 1989; Olson, Vernon, Harris, & Jang, 2001); for example, upward of 40% of the variation in favorable attitudes to euthanasia, capital punishment, and abortion is due to genetic effects (Olson et al., 2001).

Common genes influenced variation in all three domains of disgust sensitivity. When variation in pathogen and sexual disgust was influenced by specific genetic factors, the common genetic factor accounted for almost all of the genetic variance in sensitivity to moral disgust. The common genetic elements underlying sensitivities to pathogen and sexual disgust might stem from the pathogen risks inherent to sexual interactions. Sexual contact exposes people to pathogens—either those transmitted from nonsexual contact (e.g., influenza virus) or those that are typically transmitted during genital–genital contact (i.e., sexually transmitted infections). Individuals who are more invested in avoiding pathogens, then, might also follow sexual strategies that limit partner number and the extent of sexual content (Tybur, Inbar, Güler, & Molho, 2014). Genes that influence investment in avoiding pathogens (perhaps those that influence ability to combat pathogens) might in turn influence both sensitivities to pathogen and sexual disgust. Additionally, there were common genetic elements influencing sensitivity to sexual disgust that did not influence sensitivity to pathogen disgust. This might stem from the fact that sexual strategies are shaped not only by pathogen avoidance but also by numerous other factors that might have genetic sources (e.g., physical attractiveness, physical dominance in men; see Gangestad & Simpson, 2000; Lukaszewski, Larson, Gildersleeve, Roney, & Haselton, 2014).

Our finding that only those genes that also influence sensitivities to pathogen and sexual disgust influence sensitivity to moral disgust aligns with evidence suggesting that many facets of moral condemnation result from emotional intuitions that serve functions outside of the moral domain. For instance, many third-party behaviors that are widely sanctioned across cultures involve acts that observers find disgusting to engage in themselves, that is, elicitors of pathogen or sexual disgust (Tybur et al., 2013). This might reflect a computational architecture in which experiences of pathogen or sexual disgust act as inputs into the psychology of moral condemnation. Consistent with this, some evidence suggests that individuals who are exposed to disgust-eliciting odors (Schnall, Haidt, Clore, & Jordan, 2008) and tastes (Eskine, Kacinik, & Prinz, 2011) rate social and moral transgressions (i.e., consensual sex with a first cousin) as more immoral (though see Landy & Goodwin, in press). At a trait level, individuals who are more sensitive to pathogen disgust also report greater moral condemnation of myriad moral acts, including those described as violating norms of harm, care, and fairness (Chapman & Anderson, 2014). If feeling pathogen or sexual disgust more frequently or intensely increases moral judgment, then those genes that lead to variation in the pathogen and sexual factors of the TDDS might also influence sensitivity to the moral factor of the TDDS.

There were some limitations of our study that are inherent to the classical twin design. One is that shared environmental effects are confounded with nonadditive genetic effects, such that they cannot be both modeled for a single trait, and if both are present to equal degrees, their effects will cancel each other out. As such, we cannot rule out the presence of some shared environmental effects that have been masked by nonadditive genetic effects.

Another limitation of the classical twin design is that it affords very little statistical power to distinguish additive from nonadditive genetic effects, because both effects predict similar patterns of twin correlations. Although maximum likelihood estimates suggested nonadditive genetic influences for sexual and moral disgust,

the estimates were too imprecise to statistically distinguish them from additive genetic effects. Future twin studies of disgust with larger sample sizes, or that include data from parents, may reveal the extent to which nonadditive effects influence variation in disgust sensitivity, which can be informative in inferring past evolutionary selection pressures (Merilä & Sheldon, 1999).

A further limitation is that we only investigated heritability in women. This stands in contrast with Rozin and Millman's (1987) twin study, which used data from both males and females. Although there is no particular reason to expect great differences in heritability across the sexes, women tend to be more disgust sensitive overall (though specially for sexual disgust; Tybur et al., 2011; Tybur et al., 2009), which raises the possibility of different processes involved in disgust sensitivity development. As such, the extent to which the same or different genes influence men's and women's disgust sensitivity could be investigated in the future, as could sex differences in the aforementioned genetic relationships between disgust domains. That said, men's and women's scores on the TDDS are equally correlated with Big Five personality traits (Tybur et al., 2011), which suggests that they might be similarly related to the processes that lead to variation in personality. Further, it is rare to find sex differences in the genetic architecture of other traits (Vink et al., 2012).

Finally, this research was conducted using a sample of Finnish twins (Johansson et al., 2013), which precludes information about sources of variation between populations. It is important to note that the variance components presented here are proportions of variation *within* this particular population, and it is possible that a sample with more widely varying socioenvironmental contexts might yield detectable shared environmental variance in disgust sensitivity.

Conclusions

We investigated sources of variation in pathogen, sexual, and moral disgust sensitivity using a classical twin study design. Approximately half of the variation in each domain is due to genetic factors, with no evidence for shared environmental effects. This study is to demonstrate genetic influences on disgust sensitivity, and it further yielded novel findings about the genetic architecture underlying the three domains. Understanding sources of variation in disgust may be of benefit to the treatment of related clinical disorders such as obsessive–compulsive and sexual disorders (Olatunji & McKay, 2009; Olatunji & Sawchuk, 2005; Penn & Potts, 1999). The findings may also contribute to greater understanding of the many normal behaviors to which disgust is related, including mate preferences (Jones et al., 2013; Lee et al., 2013), political ideologies (Inbar et al., 2008; Tybur et al., 2010), and social avoidance and punishment (Inbar et al., 2009; Lieberman et al., 2012; Navarrete & Fessler, 2006).

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Correction to Sherlock et al. (2015)

In the article "The Quantitative Genetics of Disgust Sensitivity" by James M. Sherlock, Brendan P. Zietsch, Joshua M. Tyber, and Patrick Jern (*Emotion*, Advance online publication. October 5, 2015. http://dx.doi.org/10.1037/emo0000101), the name of author Joshua M. Tybur was misspelled as Joshua M. Tyber. All versions of this article have been corrected.

http://dx.doi.org/10.1037/emo0000141