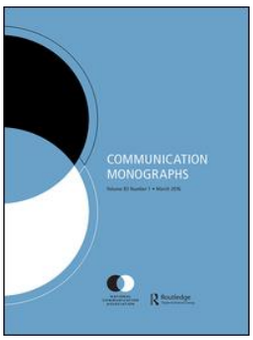

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Heritability of affectionate communication: A twins study

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ABSTRACT

Using a twin study design, we explored the extent to which affectionate communication is a heritable behavioral trait. Participants ($N = 928$) were 464 adult twin pairs (229 monozygotic, 235 dizygotic) who provided data on their affectionate communication behaviors. Through *ACE* modeling, we determined that approximately 45% of the variance in trait expressed affectionate communication is heritable, whereas 21% of the variance in trait received affection was heritable. A bivariate Cholesky decomposition model also revealed that almost 26% of the covariation in expressed and received affection is attributable to additive genetic factors. These estimates were driven primarily by females and those 50 years of age and older. The results suggest the utility of giving greater attention to genetic and biological influences on communicative behaviors by expanding the scope of communication theory beyond consideration of only environmental influences.

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Affectionate communication; genetics; twin study; ACE model; heritability; affection exchange theory

The exchange of affectionate communication is instrumental to the development, maintenance, and satisfaction of most, if not all, close relationships. People vary, however, in their propensities to express and to receive affectionate communication, so a compelling question is what accounts for such variation. Environmental factors, such as which behaviors were reinforced or punished during one's upbringing, are certainly influential (see Floyd, 2019), yet researchers have also considered the probability that one's trait level of affectionate behavior is at least partly heritable. To date, two candidate gene studies have identified specific genes associated with affectionate behavior (Floyd & Denes, 2015; Floyd & York, 2019). Those studies imply that the genetic influence on affectionate communication is nonzero, although the extent to which the affectionate communication tendency is heritable is as yet unknown.

Whereas it would be easy to assume that trait differences in affectionate communication are predominantly learned (or otherwise acquired environmentally), the present study explores the possibility that people vary in their affectionate tendencies at least partly because of their genetic inheritance. This study utilizes a twin design to examine the extent to which individual differences in trait expressed and received affectionate communication are attributable to shared genetic heritage in addition to shared environmental

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influences (aspects of the environment that affect twins similarly, such as growing up in the same community) and nonshared environmental influences (aspects of the environment that affect each twin separately, such as the influences of each twin's unique friends or experiences).

This paper begins with a rationale for viewing affectionate communication as a potentially heritable trait. This is followed by a review of affection exchange theory and a consideration of prior candidate gene studies related to affection. Finally, we discuss previous behavioral genetics studies that have used the twin paradigm to estimate heritability for traits similar to affectionate communication before offering our own research question.

Affectionate communication as a trait

Floyd and Morman (1998) conceptually defined affectionate communication as "an individual's intentional and overt enactment or expression of feelings of closeness, care, and fondness for another" (p. 145), and that definition has undergirded affectionate communication research for more than two decades (see Floyd, 2019). This definition situates affectionate communication as a *behavior,* of course, and many empirical investigations of affectionate communication have explored situational variation in people's affectionate tendencies.

Floyd (2002) argued, however, that people also have relatively stable tendencies to enact a certain level of affectionate behavior – whether high, medium, or low – in most of their relationships. Although most anyone *can* be highly affectionate, in other words, some people routinely are and others are not, so Floyd argued that people's general tendencies could be measured as a trait. Multiple studies have assessed individuals' trait-like levels of how much affection they tend to express to others (regardless of the specific relationship or circumstance) and also of how much affection they tend to receive from others (e.g., Floyd, 2006b).

If people vary in their trait-like tendency to communicate affection, then a valid question is what accounts for variance in that tendency. A social learning orientation (Bandura & Walters, 1977) would primarily implicate environmental influences, on the contention that individuals learn to be more or less affectionate from cultural and parental modeling and reinforcement. Indeed, research finds that both cultural values (Mansson & Sigurðardóttir, 2017) and parental behavior (Floyd & Morman, 2000) covary with people's tendencies to communicate affectionately.

Left unadjudicated in this work is the possibility that variance in trait affection level may be partially accounted for by genetic influences. Associations between parental behavior and the behavioral traits of their (biological) children, as identified by Floyd and Morman (2000), do not definitively establish that children acquired those traits via social learning. Instead, the genetic connection between parents and their biological offspring leaves open the possibility that behavioral traits – like physical traits – are at least partially acquired from parents genetically. Exploring the heritability of the affectionate communication trait adds specificity and clarity to our understanding of how this social tendency develops in humans. The present study's investigation into the heritability of affectionate communication was guided by affection exchange theory, described subsequently.

Affection exchange theory

Affection exchange theory (AET: Floyd, 2019) attempts to explain how and why humans engage in affectionate behavior and with what consequences. AET is described as "neo-Darwinian" insofar as it assumes that survival and procreation are superordinate human goals and claims that affectionate communication is a behavior with adaptive value with respect to those goals. Based on that assertion, multiple studies have demonstrated that affectionate behavior is beneficial for health and well-being, particularly by buffering individuals against deleterious effects of stress (see Floyd, 2019).

To what extent is affectionate behavior innate rather than learned? AET's first postulate is that "the need and capacity for affection are inborn" (Floyd, 2019, p. 29). As Floyd (2019) explains, that assertion proposes that the need to feel affection for conspecifics, as well as the cognitive and emotional capabilities for doing so, evolved in the human species due to the survival implications of affection for newborns. Importantly, this postulate speaks to the innate nature of affectionate emotions rather than affectionate behavior, although AET assumes that the need and capacity for affectionate feelings give rise to the communicative behaviors through which those feelings are made manifest. Thus, AET's central assertion raises the possibility of trait affectionate communication having an etiological basis in genes. From this perspective, person-to-person differences in trait affectionate communication may differ within populations due partly to genetic variation, a prospect we explore subsequently.

A genetic basis for affectionate behavior

When examining the potential genetic basis of a trait, two questions are relevant. First, how much of the variation in the trait is heritable (i.e., attributable to heredity), and second, which particular gene or genes are influential? For social and behavioral traits, the former question is typically examined in behavioral genetics studies and the latter in candidate gene studies. With respect to the affectionate communication trait, research thus far has addressed only two potential candidate genes, so we describe this research first before introducing relevant behavioral genetics research.

Candidate gene studies

Candidate gene studies aim to identify specific genetic loci that account for variation in a particular trait. Empathy, for instance, is associated with variation on several genes, including the dopamine receptor gene (Uzefovsky et al., 2014) and serotonin transporter gene (Gyurak et al., 2013). Similarly, prosocial behavior has been linked to variation on the receptor genes for both dopamine (Sasaki et al., 2013) and oxytocin (Kogan et al., 2011). Thus far, only two studies have examined candidate genes for the affectionate communication trait. Floyd and Denes (2015) hypothesized that genotypic variation on the oxytocin receptor gene (*OXTR*) polymorphism rs53576 would interact with attachment security to predict trait expressed affection. Genotype and attachment style interacted significantly ($\beta^* = -.64$), and as predicted, genotype had a stronger influence on trait affectionate communication for people who were low in attachment security than for those who had high attachment security. This study was the first to connect trait affectionate communication to a specific gene, but it was limited by a small sample ($N = 164$).

In a second study, Floyd and York (2019) examined data from the National Longitudinal Study of Adolescent to Adult Health and focused on the monoamine oxidase A (*MAOA*) gene. Genotypic variation in *MAOA* – specifically, the presence of the low-expressing variant (*MAOA-L*) – predicts aggressive (Ficks & Waldman, 2014) and antisocial behavior (Williams et al., 2009), and some studies have demonstrated this effect primarily or exclusively with men (Alia-Klein et al., 2008). To the extent that *MAOA-L* is positively associated with the tendency to be aggressive and antisocial, it stands to reason that its opposite, the high-expressing variant (*MAOA-H*) is positively associated with the tendency to be affectionate. In line with predictions, Floyd and York found a significant association between *MAOA-H* and trait affectionate communication, although for men only.

Results from both candidate gene studies suggest aspects of affectionate communication are heritable. These two studies demonstrate that the proportion of variance in trait affectionate communication accounted for genetically is nonzero, which naturally raises the question of how much genetic influence on the affectionate communication trait exists in the population. That question is the purview of behavioral genetics studies.

Behavioral genetics studies

Behavioral genetics studies aim to identify how much of the variation in a given trait can be accounted for by genetic inheritance, or heritability, and how much is attributable to shared and nonshared environmental influences. Such studies commonly make use of the twin paradigm, wherein both members of twin pairs are assessed on a trait, and then the within-pair correlations on that trait are computed and compared for identical or monozygotic (MZ) and fraternal or dizygotic (DZ) dyads. For many traits, MZ twins evidence stronger within-pair correlations than do DZ twins, and the larger the difference in the coefficients, the more heritable a trait is seen as being (see, e.g., Falconer, 1989).

The twin paradigm has been used for over half a century to differentiate heritable from environmental influences on human anatomical, physiological, cognitive, and behavioral traits. To date, however, no twin study has examined the heritability of trait expressed affection, although studies have documented heritability estimates for some conceptually similar constructs. For example, Gregory et al. (2009) measured adolescents' prosocial behavior via self-report and parent-report. Self-reported prosocial behavior showed an average heritability of 30.5%, whereas parent-reported prosocial behavior showed a heritability estimate of 61%. Other studies have identified nontrivial heritability estimates for cooperative behavior (Cesarini et al., 2008), interpersonal affiliation (Beatty et al., 2002), and the traits of extraversion, openness, agreeableness, and positive emotionality (Vukasović & Bratko, 2015).

Research questions

In summary, research testing the propositions of AET has provided empirical evidence that affectionate communication is a behavioral trait. Furthermore, two recent candidate gene studies have identified specific genes associated with trait levels of affectionate communication. Consequently, trait affectionate communication should be heritable, although the extent of heritability is unclear. We therefore pose the following research question:

RQ_1: What proportion of the variation in trait affectionate communication behavior is heritable?

Floyd (2002) conceived of trait affectionate communication as having two separable components: trait *expressed* affection, indexing the amount of affection one typically communicates to others, and trait *received* affection, indexing the tendency to have affection communicated to oneself. Floyd argued that measuring each component separately was advantageous, to rule out the possibility that any benefits a study identified as associated with expressed affection were simply the benefits of the affection one received in return, as the two are highly reciprocal ($r \approx .70$; Floyd, 2019). This observation raises the question of why expressed and received affection covary to the extent that they do. Whereas a norm of reciprocity (Gouldner, 1960) explanation would consider affectionate behavior a resource that ought to be reciprocated to maintain relational equity, a behavioral genetics perspective would consider whether the association between expressed and received affection is, in itself, heritable. In other words, do expressed and received affection covary because of common genetic antecedents? To investigate this possibility, we posed a second research question:

RQ_2: What proportion of the covariation in trait expressed affection and trait received affection is heritable?

Method

Participants

Participants ($N = 928$) were both members of 464 adult twin pairs. Participants ranged in age from 19 to 84 years ($M = 49.93$ years, SD = 17.22). There were 467 men and 461 women comprising 229 MZ pairs and 235 DZ pairs. Most (96.1%) did not claim Hispanic ethnicity. A large majority of participants (96.2%) were White, whereas 1.7% were Black/African American, 1.5% were Asian, 1.4% were American Indian or Alaska Natives, 0.2% were native Hawaiian Native or Pacific Islander, and 1.8% claimed another racial background.[1] When asked at what age participants and their twins moved apart/no longer lived in the same home, most (69.5%) reported that it was between the ages of 18 and 21.[2] A power analysis (Visscher, 2004) indicated that a sample size of 450 twin pairs (which we exceeded) would provide in excess of 99% power to detect a heritability estimate of .62, which is the average of heritability estimates for interpersonal affiliation and prosocial behavior, according to meta-analyses (Beatty et al., 2002; Knafo & Israel, 2010).[3]

Procedure

The study's questions and analytical strategy were preregistered with Open Science Framework on 11 June 2019, and the study was approved by the university's institutional review board.[4]

Recruitment

Twin pairs enrolled in the Washington State Twin Registry (WSTR) were considered for participation (Duncan et al., 2019).[5] The WSTR is a database of twin pairs who have

consented to participate in behavioral and health-related research. As of November 2019, the WSTR comprised 9221 adult twin pairs and 550 juvenile twin pairs. Some studies are conducted by the registry itself and others by researchers who contract with the registry.

Because data collection occurred online, the sampling frame was limited to twin pairs in which both members had a valid email address. Using Dillman et al.'s (2014) tailored design method, we issued an initial invitation email message, followed by three reminder emails, all of which varied in the time of day when they were sent. Invitations were issued between 24 April and 26 June 2019. To encourage participation from both members of each prospective twin pair, we made reminder telephone calls to non-responding cotwins beginning in June 2019. Overall response rate for completed twin pairs was 36.9%.

Twins agreeing to participate filled out a questionnaire on Qualtrics and their data were matched to those of their cotwins by a unique study-issued identifier. Participants were asked explicitly not to discuss their answers to any of the survey questions with their cotwin until both had completed and submitted their questionnaires. In exchange for taking part, participants were offered a $5 Tango gift card.

Confirmation of zygosity

In the WSTR, the zygosity of same-sex twin pairs is classified according to a unit-weighted pair zygosity sum (PZS) score rather than by self-identification.[6] The PZS score is calculated from responses to five questions about childhood similarity. The same method has also been used by other twin registries, including the Minnesota Twin Registry (Krueger & Johnson, 2002). In the WSTR, confirmation of zygosity is provided by DNA testing, using both the AmpFLSTR® Identifiler® Plus PCR Amplification Kit (Thermo Fisher Scientific, Warrington, Cheshire, UK) and the PowerPlex® 16 HS System (Promega Corporation, Madison, WI), whose results are virtually identical (Hannelius et al., 2007). Because genotyping the entire twin registry would be cost-prohibitive, WSTR has genotyped only some of the twin pairs in the registry. Of the 464 pairs in the current study, 72 (16%) have been genotyped for confirmation of zygosity. Of those, 68 pairs were correctly classified on the basis of their PZS score, an accuracy rate of 94.4% (95% CI: 0.8638, 0.9847). Two pairs who were determined by genotyping to be MZ had been classified as DZ, and two pairs who were actually DZ had been classified as MZ. On the basis of these results, we reclassified those four pairs in our data file.

Measures

Trait expressed affection was measured with the 10-item Trait Affection Scale-Given (TAS-G; Floyd, 2002). TAS-G asks participants to report how demonstrative they generally are of their affection for others. Level of agreement was assessed on a 9-point scale anchored with 1 (*strongly disagree*) and 9 (*strongly agree*). Cronbach's alpha was .96. *Trait received affection* was measured with the 6-item Trait Affection Scale-Received (TAS-R; Floyd, 2002). TAS-R asks participants how much affection they generally receive from other people. TAS-R was measured on a 9-point scale. Cronbach's alpha was .87. Both TAS-G and TAS-R have been extensively validated and evidence multiple forms of psychometric adequacy (for extended discussion, see Floyd, 2019). Consistent with prior research, TAS-G and TAS-R were strongly correlated, r (926) = .67, p (2-tailed) < 0.001.

Analysis plan

Analysis of twin data uses a special form of latent structural equation modeling called *ACE* modeling. *ACE* modeling departs from common latent approaches that are used in communication research to, for example, examine unobserved dimensions that underlie a composite of observed indicators (see Stephenson & Holbert, 2003). Whereas *ACE* modeling relies on similar principles as these more standard forms of latent modeling, the objectives, data requirements, and assumptions differ to the extent they warrant added description.

The primary goal of *ACE* is to model twins' distinct genetic and environmentally related characteristics as latent factors that explain variance in individuals' observed traits. That is, the goal is to use what is known about these unique dyads to determine why traits differ between individuals. The *ACE* approach allows researchers to estimate the extent to which trait variation in the human population is due to additive genetic traits (modeled as latent factor *A*), common environments (modeled as *C*), or unique environmental experiences (*E*) among the sample.[7]

A researcher must specify an *ACE* model by explicitly accounting for two properties distinct to biological twins. First, it is widely established that MZ twins share 100% of their DNA in common whereas DZ twins share approximately 50%. The researcher must therefore ensure the *ACE* model accounts for these differences in genetic similarity by fixing the covariance between latent factor (*A*) for twin 1 and (*A*) for twin 2 at 1.0 for identical twins and 0.5 for fraternal twins. Second, because it is assumed that all twins, regardless of zygosity, share a common social and physical environment at least early in the life course, the covariance for the latent factor (*C*) is constrained at 1.0 for all twin pairs.[8] The (*E*) factor does not covary across twins in the model. It is freely estimated and captures trait variance not explained by (*A*) and (*C*).

To identify an *ACE* model, the researcher must make several additional specifications. For example, the means for latent factors (*A*), (*C*) and (*E*) must be set at 0 and the variances set at 1.0. Model paths linking latent factors to twin 1 must be set equal to those of twin 2 as there is no statistical reason to expect differences across paths (for an overview, see York, 2020).

ACE model output provides path estimates for each latent factor: (a^2 for *A*), (c^2 for *C*) and (e^2 for *E*). When standardized, these path estimates represent the total proportion of variance in the observed variable explained by each latent factor. The estimate for additive genetic influence (a^2) is a primary vector of interest to the researcher. This value is sometimes called the "estimate of heritability" and represents the influence of genes on differences in the observed variable.

In addition, post-estimation likelihood ratio (LR) tests are used to determine whether a full *ACE* model or a reduced or "nested" model best fits the twin data. If LR tests determine that the *AE* model with the common environmental factor dropped provides a better fit to the data, the researcher should only interpret results from the more parsimonious *AE* model. If genes explain no variance in the observed variable, LR tests should show the *CE* model provides the best fit and should be interpreted rather than the *ACE* model with the superfluous (*A*) parameter.

Finally, we estimated a multivariate Cholesky decomposition model to determine the degree to which the *association* between trait expressed and received affection is explained by factors (*A*), (*C*), and (*E*). Cholesky models, rather than using latent factors to

decompose *variance* in a single trait measured among each twin and cotwin, use latent factors to decompose the *covariance* between two or more observed traits. To do so, they rely not only on within-individual, within- and cross-trait correlations on variables of interest, but cross-twin, within- and cross-trait variable relationships (see Medland & Hatemi, 2009). Another way to state this analytical approach would be that univariate *ACE* modeling attempts to discover the genetic and environmental sources of variation in a trait, whereas multivariate *ACE* modeling is designed to discover the genetic and environmental sources of covariation between traits. Univariate *ACE* modeling is used to trace person-to-person trait differences within populations back to their root genetic and environmental sources. Multivariate modeling is used to trace differences in trait relationships back to their genetic and environmental sources.

To estimate univariate and multivariate *ACE* models we used the *OpenMX* package for R. We adapted R syntax from Maes for univariate models (2016c) and for multivariate models (2016a). These particular models assume that observed traits are measured as continuous variables. We also included tests in these models for biological sex and age covariates. Covariates such as age and sex are not included directly in *ACE* models, but are rather used to check model assumptions, such as homogeneity in estimates across subgroups in the analysis (e.g., equivalent estimates for males/females) (Medland & Hatemi, 2009). As we note below, we did find significant differences in *ACE* estimates across subgroups. We thus report our primary univariate and multivariate *ACE* estimates along with estimates broken out by sex and age.

Results

Descriptive analyses

Observed scores on trait expressed affection ranged from 1.00 to 9.00, with a mean of 5.96 (SD = 1.80). Observed scores on trait received affection also ranged from 1.00 to 9.00, with a mean of 5.65 (SD = 1.64). As multiple other studies have also shown (Floyd, 2019), women scored significantly higher on trait expressed affection ($M = 6.34$, SD = 1.78) than did men ($M = 5.58$, SD = 1.74), Welch's t (924.827) = −6.63, p (two-tailed) < 0.001, $r = .21$. Women also scored significantly higher on trait received affection ($M = 5.93$, SD = 0.80) than did men ($M = 5.37$, SD = 0.07), Welch's t (906.165) = −5.25, p (two-tailed) < 0.001, $r = .44$. Participant age was significantly associated with trait expressed affection, r (926) = .20, p (two-tailed) < 0.001, and with trait received affection, r (926) = .19, p (two-tailed) < 0.001.

Table 1 outlines within-pair correlations on the variables of interest. Importantly, within-pair correlations on trait expressed and received affection showed identical twins were more similar on each measure than were fraternal twins. Notably, within-pair correlations were far stronger for female identical twins than female fraternal twins. The same was not true of male twins. There were also larger gaps in correlations between identical and fraternal twins over 50 years old than twins under 50. These differences in correlations are initial indicators that genetic influence may operate differently within subgroups.

Research questions

To address RQ_1, we estimated *ACE* models that partition the variance in trait expressed affection and trait received affection attributable to each of three sources. Our main

Table 1. Within-pair correlations.

	Trait Affection Expressed (TAS-G)		Trait Affection Received (TAS-R)	
	Identical (MZ) Twins	Fraternal (DZ) Twins	Identical (MZ) Twins	Fraternal (DZ) Twins
Total *r*	0.52†	0.24†	0.40†	0.26†
Male/male	0.46†	0.47†	0.28**	0.42†
Female/female	0.56†	0.13	0.49†	0.21
Male/female	–	−0.05	–	0.07
Age < 50	0.49†	0.30†	0.32†	0.31†
Age ≥ 50	0.48†	0.12	0.41†	0.19*

Note. $N = 928$ individuals comprising 464 pairs (235 DZ; 229 MZ). Cell entries represent within-pair Pearson correlation coefficients (*r*)
*$p < 0.05$
**$p < 0.01$
†$p < 0.001$.

results appear in Table 2. The first column in the table, denoted a^2, indicates the proportion of variance attributable to additive genetic traits (heritability), whereas the c^2 column identifies variance attributable to common environmental factors and the e^2 column indicates the variance attributable to unique environmental factors. Model fit indices appear to the right of these columns. Only the results from the best fit model, depicted in bold type, are interpreted.

Results indicate nontrivial contributions of additive genetic traits for both affectionate communication variables. For trait expressed affection, the *AE* model was preferred over the *ACE* model. In this model, 45% of the variance was heritable and 55% was attributable to unique environmental influences, whereas the estimate for common environmental factors was zero. The full *ACE* model was most parsimonious for trait received affection. Here, 21% of the variance in the trait was explained by the genetic factor, whereas the common environment factor accounted for 14%, and the unique environment factor accounted for 65%.

Tests for differences in *ACE* estimates across covariates were significant in both the TAS-G ($\chi^2 = 28.32$, $p < 0.001$) and TAS-R models ($\chi^2 = 26.45$, $p < 0.001$). Consequently, we conducted a multiple group, sex-limited *ACE* model in Table 3. This model is based on syntax by Maes (2016b) and is used to explore sex differences in the proportion of

Table 2. Univariate ACE estimates for TAS-G and TAS-R.

Variable	Model	a^2	c^2	e^2	AIC	−2LL	χ^2	*p*
TAS-G	ACE	0.45	0.00	0.55	1772.51	3618.51	Base	–
	AE	**0.45**	–	**0.55**	**1770.51**	**3618.51**	**0.00**	**1.00**
	CE	–	0.34	0.66	1779.95	3627.95	9.44	0.00
TAS-R	**ACE**	**0.21**	**0.14**	**0.65**	**1623.70**	**3469.70**	**Base**	–
	AE	0.37	–	0.63	1622.74	3470.74	1.04	0.31
	CE	–	0.29	0.71	1623.43	3471.43	1.73	0.19

Notes: $N = 928$ individuals comprising 464 pairs (235 DZ; 229 MZ). a^2 = additive genetic traits, c^2 = common environment, e^2 = unique environment. The a^2, c^2, and e^2 values are standardized path estimates. They represent the proportion of total variance in each observed variable explained by the additive genetic factor, common environment factor, and unique environment factor. Together, they should sum to 1.0 or 100% of the variance in observed traits. AIC = Akaike Information Criteria. −2LL is −2 times the loglikelihood. The *ACE* model for each variable is followed by a nested *AE* and *CE* model with one parameter dropped. χ^2 indicates change in model fit as a result of dropping (*A*) or (*C*) from the base model. Models shown in bold are the best fitting models based on LR tests of fit (*p* values shown at right).

Table 3. Univariate ACE estimates for TAS-G and TAS-R by biological sex.

Variable	Model	a^2	c^2	e^2	AIC	−2LL	χ^2	p
Women								
TAS-G	**ACE**	**0.48**	**0.00**	**0.52**	**1747.55**	**3583.55**	**Base**	–
	AE	0.48	–	0.52	1750.74	3588.74	5.19	0.07
	CE	–	0.28	0.72	1763.53	3603.53	19.98	0.00
TAS-R	**ACE**	**0.42**	**0.00**	**0.58**	**1607.39**	**3443.39**	**Base**	–
	AE	0.42	–	0.58	1611.13	3449.13	5.75	0.06
	CE	–	0.29	0.71	1613.50	3453.50	10.12	0.02
Men								
TAS-G	**ACE**	**0.00**	**0.39**	**0.61**	**1747.55**	**3583.55**	**Base**	–
	AE	0.42	–	0.58	1750.74	3588.74	5.19	0.07
	CE	–	0.35	0.65	1763.53	3603.53	19.98	0.00
TAS-R	**ACE**	**0.00**	**0.31**	**0.69**	**1607.39**	**3443.39**	**Base**	–
	AE	0.31	–	0.69	1611.13	3449.13	5.75	0.06
	CE	–	0.26	0.74	1613.50	3453.50	10.12	0.02

Note: $N = 928$ individuals comprising 464 pairs (235 DZ; 229 MZ). a^2 = additive genetic traits, c^2 = common environment, e^2 = unique environment. AIC = Akaike Information Criteria. −2LL is −2 times the loglikelihood. The quantitative non-scalar sex difference *ACE* model is followed by nested *AE* and *CE* sex difference models. χ^2 and p values are for LR tests. Best fitting models are bolded. Fit statistics are repeated as female and male twins are modeled simultaneously.

variance explained by latent genetic and environmental factors. Similarly, we show univariate *ACE* models by age in Table 4 to explore how estimates change as a function of age.

The results shown in Tables 3 and 4 suggest genetic effects found in the initial univariate *ACE* models are driven mainly by female and older respondents. Table 3 shows that latent genetic factor accounted for 48% of variance in trait expressed affection and 42% of variance in trait received affection among women, whereas there was a null genetic effect for men. Table 4 shows there is more consistent genetic influence among twins 50 years and older, with the latent genetic factor explaining 39% of the variance in trait expressed affection and 37% in trait received affection. Among respondents under 50 years old, the latent genetic factor accounts for 40% of variance in trait expressed affection but only 2% in trait received affection.

To address RQ_2, we estimated bivariate Cholesky decomposition models, which examine the extent to which covariation in two traits is attributable to additive genetic

Table 4. Univariate ACE estimates for TAS-G and TAS-R by age.

Variable	Model	a^2	c^2	e^2	AIC	−2LL	χ^2	p
Age < 50								
TAS-G	**ACE**	**0.40**	**0.09**	**0.51**	**913.84**	**1873.84**	**Base**	–
	AE	0.50	–	0.50	912.12	1874.12	0.27	0.60
	CE	–	0.39	0.61	915.73	1877.73	3.89	0.05
TAS-R	**ACE**	**0.02**	**0.30**	**0.68**	**807.88**	**1767.88**	**Base**	–
	AE	0.37	–	0.63	808.56	1770.56	2.67	0.10
	CE	–	0.31	0.69	805.89	1767.89	0.01	0.93
Age ≥ 50								
TAS-G	ACE	0.39	0.00	0.61	861.69	1741.69	Base	–
	AE	**0.39**	–	**0.61**	**859.69**	**1741.69**	**0.00**	**1.00**
	CE	–	0.27	0.73	864.80	1746.80	5.11	0.02
TAS-R	ACE	0.37	0.00	0.63	816.58	1696.58	Base	–
	AE	**0.37**	–	**0.63**	**816.58**	**1696.58**	**0.00**	**1.00**
	CE	–	0.27	0.73	817.00	1699.00	2.42	0.12

Notes: $N = 928$ individuals comprising 464 pairs (235 DZ; 229 MZ). a^2 = additive genetic traits, c^2 = common environment, e^2 = unique environment. AIC = Akaike Information Criteria. −2LL is −2 times the loglikelihood. χ^2 and p are associated with LR tests of model fit and indicate change in fit as result of dropping (*A*) or (*C*) from the base model. Models in bold are the best fit models.

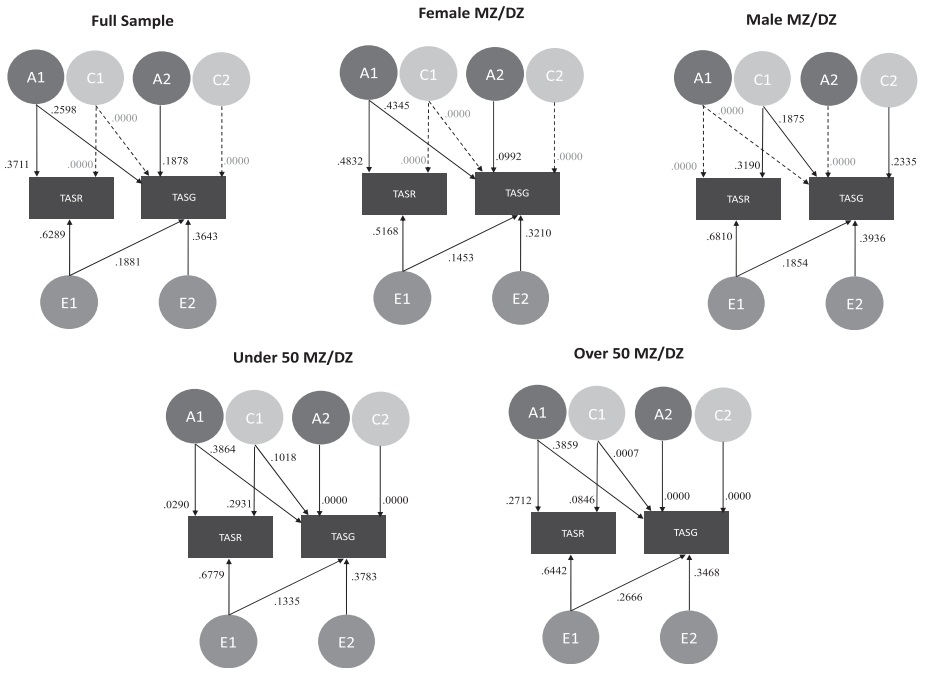

Figure 1. Bivariate Cholesky decomposition models. Note: Full sample $N = 928$ individuals comprising 464 pairs (235 DZ; 229 MZ). TAS-R = trait received affection; TAS-G = trait expressed affection. (*A*), (*C*), and (*E*) denote the latent additive genetic, common environment, and unique environment factors for twin 1 and 2. Standardized estimates are shown. For the full sample, the *AE* model provided the more parsimonious fit to these data (−2LL = 6589.74, $\chi^2 = 2.02$, $p = 0.57$). The *AE* model was preferred for females (−2LL = 2777.54, $\chi^2 = 0.10$, $p = 0.99$) and *CE* for males (2LL = 2709.01, $\chi^2 = 0.10$, $p = 1.00$). *ACE* was preferred for those under 50 (−2LL = 3454.09) and 50 and older (−2LL = 3108.65).

factors, common environmental factors, and unique environmental factors. We show results from a full sample Cholesky model followed by results from separate models by sex and age in Figure 1. The full sample Cholesky model in the upper left corner of Figure 1 indicates that 25.98% of the association between expressed and received affection is heritable, whereas unique environmental factors accounted for 18.81% of the variation in their association, and no variance in the association was attributable to common environmental factors. Put differently, shared genes accounted for more covariance between TAS-G and TAS-R than did the unique environment and common environment (e.g., parenting, culture) combined.

However, consistent with the univariate models, genetic explanations of covariance were more apparent in a Cholesky model that considered females separately from males. When females were considered by themselves, the *AE* model was preferred, and approximately 43.45% of the relationship between trait expressed and trait received affection was explained by latent genetic traits. For males, the *CE* model was preferred, and latent genetic traits explained an estimated zero percent of covariation between traits. The standard *ACE* model was most parsimonious when considering all twins under 50 years old as one group separately from those 50 years and older. In both of

these models, almost 39% of the relationship between trait expressed and received affection was heritable. Environmental influences were relatively small.

Discussion

Affection exchange theory observes that, in addition to varying their levels of affection within individual relationships, people also have a trait level of affectionate behavior, which evolved in the human species due to its contributions to the long-term goals of viability and fertility. This claim raises the probability that individual variation in that trait is not wholly accounted for environmentally. Rather, it suggests that a nontrivial proportion of the variance is accounted for by genetic inheritance. Previous candidate gene studies have shown that variants of the genes *OXTR* and *MAOA* are significantly related to trait affectionate communication, implying at least a partly genetic basis for the trait. However, no previous research has quantified the extent to which variance in the affectionate communication trait is attributable to a genetic factor, as opposed to shared or nonshared environmental influences. We thus undertook the current study with that goal in mind.

Using a sample of MZ and DZ twins, we found that at the univariate level, approximately 45% of the variance in trait expressed affectionate communication is heritable, whereas the remainder is attributable to nonshared environmental influences. Critically, the common environment factor explained zero percent of the variance in trait expressed affection. This means that what are presumably shared influences such as being raised by the same parents in the same culture had no impact on trait expressed affectionate communication. The overall heritability estimate of .45 is strongly in line with heritability estimates of related constructs. In fact, it is similar to the average of estimates for affiliation (.70; Beatty et al., 2002), prosocial behavior (.54; Knafo & Israel, 2010), extraversion (.39; Vukasović & Bratko, 2015), and positive affect (.36; Eid et al., 2003). Given that nontrivial proportions of the variance in these related traits also have a genetic basis, it is unsurprising to discover the same for expressed affectionate communication.

We also documented a heritability estimate of .21 for trait received affection. Given the respective nature of the expressed and received affection traits, it is unsurprising that received affection is less heritable than expressed affection. Specifically, expressed affection indexes one's own tendency to communicate affection to others, a behavior that – like being talkative or friendly – is largely within one's individual control. On the contrary, received affection reflects the extent to which one receives affectionate expressions from others, and although individual characteristics – such as social attractiveness or warmth – may increase the odds of receiving affectionate messages, the extent to which one receives expressions of affection is conceptually under other people's control, as much as or more than under one's own. One may certainly inherit a greater or lesser tendency to be kind and considerate (Gregory et al., 2009), which could conceivably influence the level of affection one receives from others. Nonetheless, trait received affection is conceptually a more socially contingent trait than trait expressed affection, which makes its lower heritability estimate unsurprising.

Importantly, however, significant subgroup effects were observed for both trait expressed affection and trait received affection based on sex, and additionally for trait received affection based on age. Sex effects indicate that the heritability estimates for

both expressed and received affection are limited to women; similarly, the heritability estimate for received affection is substantially higher (.37) for those 50 years of age or older than for those under 50 years of age (.02). As both of these effects were unhypothesized, we can only speculate as to their meaning.

It is instructive to consider, first, that heritability in other psychosocial and behavioral traits also varies by sex and/or by age. Although most human traits do not evidence sex-specific genetic *or* environmental effects (Stringer et al., 2017), a range of traits has demonstrated sex differences (Boardman et al., 2008; Jansson et al., 2004) or age differences (Bergen et al., 2007) in heritability. Why might biological sex influence the heritability of expressed and received affectionate behavior? One potential clue – also suggested by Jansson et al. (2004) in the context of depression – is the finding that, nearly without exception, women both express and receive more affection than do men (for review, see Floyd, 2019).[9] Assuming the sex difference in prevalence does not reflect a biased measurement strategy, it is therefore possible that the higher rates of expressed and received affection observed in women reflect a true sex difference in genetic influences on these tendencies. Such a difference may be evolutionarily adaptive. For instance, Taylor et al.'s (2000) tend-and-befriend theory explains that tending to offspring and befriending allies are advantageous stress reactions for women, compared to fight-or-flight responses, largely because such reactions induce oxytocinergic calm in women more than they do in men.[10] As Floyd (2006a) pointed out, tending and befriending – and especially the latter – embody the exchange of affectionate behavior, making Taylor et al.'s argument relevant for understanding why tendencies toward affectionate communication may have a stronger genetic foundation for women than for men.

In the present study, age demonstrated no effect on the heritability of trait expressed affection; rather, the heritability of trait received affection was substantially stronger for those 50 years of age and older than for those younger than 50 years of age. The explanation for this unhypothesized finding is somewhat more speculative, for two reasons. First, unlike for biological sex, research has documented few age effects on affectionate behavior, and none for trait received affection (see Floyd, 2019), so a consistent difference between older and younger individuals in the behavior offers no clue as to a difference in its heritability. Second, insofar as social and emotional regulation abilities tend to increase with age and experience (e.g., Lawton et al., 1992), one might logically expect that genetic effects on such behaviors become *less* potent over time, rather than more. To ascertain why genes are more strongly related to received affection for older than younger adults, therefore, future research may explore predictors of received affection among older adults to determine whether any major predictors – such as physical attractiveness, intelligence, or modal vocal pitch – might have genetic bases.

Finally, multiple studies have documented strong covariation between expressed and received affection (see Floyd, 2019), raising the question of what accounts for this covariation. A social exchange orientation would explain that, at least in satisfying relationships, "one good turn deserves another," so when relational partners receive expressions of affection, they typically reciprocate them to maintain balance in the exchange of that relational resource. This, in itself, theoretically accounts for the strong reciprocity between trait measures of expressed and received affection – yet the observation that both traits are heritable to a nontrivial degree suggests that their covariation may also be heritable. That is, the same genetic factor that explains individual differences in each trait may

also be partly responsible for why the two traits covary. Indeed, we discovered that one quarter of the covariance between expressed and received affection (25.98%) is attributable to additive genetic factors, which suggests overlap in the genes contributing to each trait. This finding implies expressed and received affectionate communication do not covary entirely as a function of social exchange expectations.

Age had no effect on heritability estimates for the covariance between expressed and received affection, but the estimates once again differed between women and men. For women, a substantial proportion of the covariation between expressed and received affection – more than 43% – was heritable, whereas for men, all of the covariation was attributable to shared or unique environmental effects. Although unhypothesized, this finding is unsurprising in light of the univariate results; insofar as both expressed and received affectionate communication are partly heritable for women but not for men, it is certainly understandable that covariation in these behavioral tendencies would show a similarly sex-differentiated heritability pattern.

Implications

The study's principal implication is that theoretic efforts to understand and account for variance in communicative behavior may benefit by paying greater attention to genetic influences. A pervasive assumption in communication theory has been the primacy of environmental influences such as culture, gender, socioeconomic status, parenting behavior, and media effects, to the near exclusion of potential genetic or otherwise biological factors. The present findings add to a growing empirical literature illuminating the limitations of that assumption. The implication is not that environmental influences are irrelevant in shaping communicative traits; rather, it is that the etiology of some communicative traits is more accurately described as incorporating both biological and environmental effects, as well as their potential interactions, at least for some populations.

Expanding the scope of communication theory to include biological and genetic influences may therefore be useful with respect to accounting for variance in social behavior. Importantly, genetic effects may contribute not only to behavioral dispositions themselves but also to the environmental influences that shape those dispositions. For instance, social media use is implicated in social behavior (e.g., Korda & Itani, 2013), yet social media use is strongly heritable (York, 2017, 2019). Similarly, parenting style influences behavioral tendencies in children (O'Leary & Vidair, 2005), yet parenting style is also strongly heritable (McGuire et al., 2012). In both instances, therefore, what may appear to be a proximal environmental influence on behavior may in fact have functional-ultimate genetic antecedents which remain unadjudicated by environment-centric theoretical models.

As useful as it is to identify what the present findings imply, it is perhaps even more important to underscore what they *do not* imply. First, the univariate finding attributing 45% of the overall variation in trait expressed affection to heritable genetic factors does not imply that 45% of a *specific individual's* affectionate communication has a genetic basis. As Krueger et al. (2008) explain, "the concept of heritability applies not to individuals but, rather, to differences among many individuals. Stated in statistical terms, heritability applies to the variance of a set of observations rather than to a single specific observation" (p. 1487). Heritability estimates gauge the extent to which traits differ between individuals in a population due to genetic variation.

Second, attributing any of the variance in a behavioral tendency to genetic factors does not imply that that percentage of the behavior is preordained and beyond an individual's ability to control. Such a claim invokes both the deterministic fallacy – the false belief that genetic influences on behavior are deterministic rather than probabilistic – and the immutability fallacy – the false belief that genetically influenced behavioral traits are unchangeable (see Dar-Nimrod & Heine, 2011). As an analogy, up to half of the variance in the behavioral trait of aggressiveness is heritable (Tuvblad & Baker, 2011), and specific variants of the *MAOA* and *5HTT* genes have been extensively linked to aggressive behavioral tendencies in children, adolescents, and adults (see Veroude et al., 2016). Notably, however, those observations do not imply that having aggressive biological parents or specific variants on the *MAOA* and *5HTT* genes *determines* one's tendency toward aggression, nor that aggressive behavior is uncontrollable or unchangeable (in fact, cognitive behavioral therapies show high efficacy for treating aggressive behavioral tendencies; Hoogsteder et al., 2014).

Relatedly, the finding that all of the variation in men's expressed and received affectionate tendencies is attributable to environmental factors – particularly *nonshared* environmental factors – supports the theoretic efficacy of interventions intended to increase men's affectionate behavior. As Floyd (2015) reported, complaints about men's less frequent and less overt displays of affection (relative to women) are common in the context of marital and family therapy (see also Doss et al., 2004). Although few behavioral interventions have yet been systematically developed and tested for efficacy with any population (see, e.g., Andrews et al., 2013, for an exception), therapeutic interventions such as L'abate's (2008) 3HC (for hugging, holding, huddling, and cuddling) may have promise for affecting the environmental conditions that encourage the exchange of affection in close relationships.

Finally, although *ACE* models partition variance into heritable, common environmental, and nonshared environmental influences, we cannot rule out the possibility that some variance in trait affectionate behavior is accounted for by gene-environment (GxE) interactions, wherein a given genetic factor is influential only under particular environmental conditions (see Ritz et al., 2017). This possibility could be tested in future work that, like Floyd and Denes (2015), measures both genotypes and environmental factors with which they theoretically interact.

Strengths, limitations, and conclusions

This study benefited from a sample of adult twin dyads that included a subsample of opposite-sex twins, who are sometimes excluded from twin studies (e.g., Hahn et al., 2016). DNA-based confirmation of zygosity, albeit for only a portion of the sample thus far, is a second strength of the current design, insofar as some studies rely heavily on researchers' impressions of twins' similarities in appearance and behavior to confirm zygosity (e.g., Beatty et al., 2002).

As participants in the Washington State Twin Registry, however, the participants comprised a convenience sample rather than a representative sample of the adult twin population. As such, an unknown degree of sampling bias is to be expected, as in all convenience samples. For instance, it is plausible that point estimates for trait expressed affection and trait received affection differ from the true population means – either the means among the U.S. singleton population or twin population or both. In theory, differences in sample and population means would not disrupt variable relationships or the

degree to which shared genetic relative to environmental factors among sample respondents explain variance in a given trait. However, because *ACE* models use variable means for start values, it is possible that under- or over-estimated average scores for TAS-G and TAS-R can inflate or depress *ACE* estimates.

An additional potential limitation is that, in *ACE* modeling, measurement error is reflected in the unique environmental factor (*E*), rather than being estimated separately from the *ACE* components. Consequently, excessive measurement error can inflate estimates of (*E*) and downwardly bias estimates of genetic (*A*) and shared environmental influence (*C*) on a trait, which also warrants caution in interpreting model estimates.

Notes

1. These percentages sum to >100 because some participants reported multiple racial backgrounds.
2. When asked at what age they moved apart from their twin, 2.5% of participants said they still lived with their twin, whereas 0.2% moved apart before age 6; 0.2% between 6 and 10; 0.5% between 11 and 14; 10.3% between 15 and 17; 69.5% between 18 and 21; 13.0% between 22 and 24; and 3.8% at age 25 or older.
3. We selected interpersonal affiliation and prosocial behavior as constructs to use for the power analysis because these seemed closest conceptually to affectionate behavior among the traits for which heritability estimates existed, and also because heritability estimates for these constructs were based on meta-analyses instead of single studies.
4. An anonymized view of the OSF preregistration is available at https://osf.io/hdnmp/?view_only=2417667886be487bafc16b51c403804e.
5. For more information on the WSTR, see https://wstwinregistry.org/.
6. Other-sex pairs are automatically classified as DZ.
7. Estimates of the variance components (*A*), (*C*), and (*E*) always sum to 100% or 1.0 of the variance explained in a given observed variable. By definition, any variance not accounted for by twins' shared genes (*A*) or shared environment (*C*) is accounted for by the unique environment factor (*E*). In this sense, (*E*) represents any experience idiosyncratic to one twin and not the cotwin, while at the same time (*E*) serves as the model's error term.
8. Only 11.2% of our respondents reported no longer living together prior to age 18. The majority (82.5%) reported separating during ages 18 to 24. In addition, prior twin studies confirm that a common upbringing is a safe assumption. That is, it is widely safe to assume a twin and cotwin are born at roughly the same time, raised together in the same household by the same parents, and are socialized within the same culture. One practical objection to this assumption is that, even if twins are reared in the same home, they may have novel experiences (e.g., having a distinct peer). *ACE* models address this objection in part through the (*E*) factor, which is intended to capture environmental experience unique to each twin.
9. Floyd (2019) pointed out that nearly every study that has explored the effect of biological sex has reported that women both express and receive more affectionate communication than do men, and those studies that have not documented such a difference have reported null results. There is no documentation of men, in any culture or age group, scoring significantly higher than women on either TAS-G or TAS-R.
10. Empirical evidence for tend-and-befriend theory's claim of a sex difference in oxytocinergic reactivity to stressors is inconsistent, however (see, e.g., Floyd et al., 2010).

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No potential conflict of interest was reported by the author(s).

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