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Genetic and Environmental Influences on Media Use and Communication Behaviors

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A great deal of scholarly work has explored the motivations behind media consumption and other various communication traits. However, little research has investigated the sources of these motivations and virtually no research considers their potential genetic underpinnings. Drawing on the field of behavior genetics, we use a classical twin design study to examine the genetic and environmental influences on nine communication behaviors. Our findings indicate a substantial portion of the total variance in media habits can be attributed to genes, as much as one-third of the variance in some instances. Mass communication scholars would benefit by paying closer attention to heritability when thinking about the causes as well as the consequences of media traits in contemporary society.

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Media consumption behaviors, comprised of observable acts of individual media exposure (LaRose, 2010), have been heavily studied in mass communication. Mainly described as the product of a conscious selection process, these behaviors have been understood using a multitude of seminal communication theories including the "uses and gratifications" (U&G) paradigm. Developed in the 1940s, U&G sought to address behavior indicated by audiences who seek out content from the media that provide them with specific experiences (Lowery & DeFleur, 1995). This article follows this formative research by examining an area frequently overlooked by media scholars—motivation. Scholars have provided ample evidence of how media use and other communication behaviors gratify a series of different motivations, yet current research is limited in its ability to explain the causes of these motivations.

U&G theory has evolved and been applied to a variety of behaviors including—most recently—Internet use. Throughout this evolution, motivation has remained a basic element of this research, although its sources are largely unexamined. While mass communication researchers develop the paradigm theoretically and broaden its empirical application, we still struggle to explain the origins of motivations underlying communication behaviors. And while scholars have linked media behaviors to specific

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individual needs and personality traits, our understanding of how these motivations form is lacking. More simply, U&G research has difficulty explaining why an individual chooses to engage (or not engage) in various types of communication even as we have learned much about her specific choices given a decision to engage media. This study aims to help address this important gap by examining genetic and environmental influences on media use, news consumption, and interpersonal communication.

Enriching our understanding of the underpinnings of communication behaviors is important for several reasons. First, communication behaviors are important predictors for other characteristics, judgments, and actions. Media consumption serves as a strong antecedent of political knowledge (Delli Carpini & Keeter, 1996), economic evaluations (Hetherington, 1998), and voting (Weaver, 1996). Second, political interest and information seeking are central to effective democratic functioning (Prior, 2010). They work in tandem to affect electoral participation and the likelihood of being mobilized to participate (Brady, Schlozman, & Verba, 1999). As scholars continue to explore variation in political interest, it is also important to understand why some individuals are more motivated than others to seek information. Finally, changes in technology and the media environment have afforded citizens more opportunities to consume—or ignore—information. These transformations to our media ecology have altered researchers' understanding of media effects. Scholars argue that these changes will most affect people motivated to seek information (Bennett & Iyengar, 2008). Consequently, understanding the sources of these motivations is critical.

An expansive literature on behavioral genetics has demonstrated the influence of heredity on social traits, behaviors, and attitudes, as varied as personality characteristics (Eaves, Eysenck, & Martin, 1989; Loehlin, 1992), social network composition (Fowler, Dawes, & Christakis, 2009), cigarette use (Vink, Willemsen, & Boomsma, 2005), political participation (Fowler, Baker, & Dawes, 2008), political partisanship (Hatemi, Alford, Hibbing, Martin, & Eaves, 2008), political ideology (Alford, Funk, & Hibbing, 2005), and attitudes about economic risk (Zhang et al., 2009). We extend this line of inquiry by examining whether the propensity to be informed, use media, and communicate with others is also partially heritable. To this end, we use a genetically informative research design, a classical twin study, and data from the National Longitudinal Study of Adolescent Health (Add Health) and the Midlife in the United States National Study of Health and Well-Being (MIDUS). These large national surveys included subsamples of twin pairs to isolate genetic and environmental influences on nine communication traits.

Uses and gratifications paradigm

Early uses and gratifications research sought to explain how individuals use media to satisfy personal needs, including both social and emotional ones (Cantril, 1942). Branching from traditional media effects literature, the uses and gratifications paradigm (U&G) examines the purpose of media behavior. According to Lowery and

DeFleur (1995), U&G allows researchers to address important theoretical questions surrounding individuals' active reasoning for choosing to engage in a host of media behaviors (p. 400). Bryant and Oliver (2008) describe media use behavior as "goal directed and purposive" (p. 167). Audience activity remains a central tenant of the U&G paradigm with individual communication motives influencing audience behaviors. According to Rubin (1994), the U&G perspective posits that there are key determinants of individual media use including both psychological and social environments.

While U&G research in the 1940s focused on categorizing audience members into types of media users, in the 1960s researchers began using the theory to predict media choices (Gersen, 1966; Mendelsohn, 1964; Katz & Foulkes, 1962). During this time, researchers also began investigating how and why different individuals are motivated to use media differently, a significant shift in the U&G paradigm (Wimmer & Dominick, 1994; Katz, Blumler, & Gurevitch, 1974). As Ruggiero (2000) explains, researchers during the 1950s and 1960s became interested in the precursors of media gratifications. Specifically, research became more concerned with the root causes of media use rather than the effects of media use, thus shifting the focus on the individual from effect to motive.

As forms of mediated communication increased through the mid-20th century allowing more variation in choice (Bennett & Iyengar, 2008), scholars were plagued by questions of why some individuals chose certain types of media over others. This shift in focus led to Rosengren's (1974) model, which still serves as the basis of contemporary U&G research. Rosengren theorized that "individual needs" interact with both personal characteristics as well as social environments to produce "motivations" for communication behaviors. These motivations are gratified by media use but also lead to different patterns of media effects. Rosengren's model focuses on the individual, by drawing attention to the needs that drive motivations and lead to media use. However, the model also uses the individual-level variation to explain why some people are affected by the information they consume and others remain largely unaffected. Because media use is driven by individual motivations, audience members react differently to sources of communication and messages. While scholars may disagree about the logistics of specified models, they largely agree that individual motivations are the underlying cause of all communication behaviors, including media use.

Etiology of media use motivations

A key assumption in U&G research is that audience members are active participants in the process of mediated communication (McQuail, 1994). This theoretical understanding has broad applicability. Recently, scholars have examined individual motivations for a host of communication behaviors from interpersonal interaction (Rubin, Perse, & Barbato, 1988) and cable television subscription (LaRose & Atkin, 1991; Donohew, Palmgreen, & Rayburn, 1987) to VCR (Lin, 1993; Rubin & Rubin, 1989), and Internet use (Flaherty, Pearce, & Rubin, 1998; Papacharissi & Rubin, 2000).

While the technology varies, the main focus of the research deals with individual-level motivations and needs. How are these motivations formed? Contemporary research suggests these motivations are complex and deeply rooted, a function of parental influence and enduring personality traits (Blumler, 1985; Donohew et al., 1987; Lull, 1995; Plomin, Corley, DeFries, & Fulker, 1990).

One of the seminal studies exploring determinants of media use was conducted by Plomin et al. (1990; Ridley, 2003) and examined differences in television-viewing habits among children. Building on a study by Loehlin and Nichols (1976) that found that television viewing was more highly correlated among identical twins than fraternal twins,[1] Plomin and colleagues replicated this study and extend it to identify heritable and environmental influences on television use. Using the Colorado Adoption Project (CAP), they find that adolescent television use is a product of both environmental and genetic influences. While offspring do have similar television-viewing patterns as their parents, environmental influence only accounts for 20% of the variance of children's television use.

Plomin et al. (1990) report that up to 34% of television viewing among children in their sample can be attributed to genetic influences. Importantly, they emphasize that they are not identifying a "television gene." Rather, television viewing is a complex phenotype that is a result of a series of different chemical reactions. Thus, it is "heritable but not inherited" (Plomin et al., 1990, p. 371). While innovative, the Plomin et al.'s study consisted of 3- to 5-year olds, a demographic with arguably less behavioral autonomy. The study also only focuses on one form of media use, television viewing (see Bouchard, 2004; Bouchard & McGue, 2003).

Building on this line of research, Sherry (2001) finds a relationship between "temperament," an enduring and genetically influenced aspect of one's personality, and media use motivations. Sherry's work is consistent with psychological trait theory, which suggests "biologically rooted individual differences in behavior tendencies that are present early in life and are relatively stable across various kinds of situations and over the course of time" (Bates, 1989, p. 4). Communication scholars have argued that there are innate aspects of an individual's personality that are related to individual media consumption and specific communication behaviors (Rosengren, 1974; Rosengren, Wenner, & Palmgreen, 1985; Weaver, 2000).

Significant research indicates that variation across the Big Five personality traits (openness, neuroticism, conscientiousness, agreeableness, and extraversion) are associated with interpersonal communication and media use habits. As expected, individuals with high levels of extraversion, typically characterized with positivity and tendency to seek out others, express more interest in interpersonal communication than their introverted counterparts (Weaver, 2000). These individuals also are more likely to participate in online communication activities (Hamburger & Ben-Artzi, 2000) and social media (Correa, Hinsley, & Zuniga, 2010). While extroverts enjoy watching television, it does not fill the same need as interpersonal communication (Weaver, 2003). Neuroticism, which is largely described as emotional instability, is a strong predictor of television use (Weaver, 2003) and computer instant messaging

(Ehrenberg, Juckes, White, & Walsh, 2008). This is consistent with Sherry's (2001) survey of 285 adults, which found that temperament is related to television use. Specifically, negative mood, low task orientation, and behavioral rigidity—all personality traits associated with neuroticism—predict an individual's desire to watch television.

In addition to explaining certain communication behaviors, personality traits are also significant predictors of political information consumption. As part of a larger research agenda examining the influence of personality characteristics on political behaviors, Gerber, Huber, Doherty, and Dowling (2010) find that the Big Five personality traits predict political knowledge levels (see also Mondak, Hibbing, Canache, Seligson, & Anderson, 2010; Mondak & Halperin, 2008) and political discussion (Hibbing, Ritchie, & Anderson 2010). The personal drive to discover new information is also a strong predictor of news consumption. Delli Carpini and Keeter (1996) extend these findings by developing the Opportunity-Motivation-Ability framework (OMA; Delli Carpini & Keeter, 1996). Opportunity is environment based, referring to the frequency with which information is made available to an individual. Motivation and ability are both individual characteristics and are described as both innate and learnable skills (Prior, 2007). While the OMA framework does not explicitly include the heritability of motivation and ability (Delli Carpini & Keeter, 1996; Luskin, 1990) information seeking is clearly a product of both environmental and individual factors. Political knowledge research has demonstrated that education, prior knowledge, and political involvement are the strongest predictors of knowledge about public affairs (Delli Carpini & Keeter, 1996; Zaller, 1996). In addition, research has demonstrated that these three variables also predict news consumption (Althaus & Tewksbury, 2000). In short, information seeking hinges on the opportunities to learn, as well as intrapersonal characteristics, such as the individual's motivation and ability, both of which may be partially explained by heritability.

This scholarship is useful in our understanding the active audience, but researchers know remarkably less about predicting media use and information seeking among passive audiences. Passivity plays two distinct roles in communication behaviors. First, individuals may use media as part of their routine or habit (Oellette & Wood, 1998). For example, even though weekday television programming significantly differs from weekend programming, individuals tend to watch television regardless of the day of the week (Rosenstein & Grant, 1997). In a recent examination of habitual media use, LaRose (2010) argues that there are inherent thought processes that guide such use. He contends habitual behaviors can form outside of consciousness, thus suggesting an alternate approach to understanding media behaviors relative to the traditional U&G paradigm (LaRose & Eastin, 2004). Secondly, research has shown that television viewers are reluctant to turn off a medium even when the programing/content offered at the time does not specifically meet their preferences (Rosenstein & Grant, 1997). Rather than purposive consumption, individual decisions to consume media are largely independent of the program they are consuming.

While researchers have some understanding of how different motivations relate to various communication behaviors, current research struggles to explain routine

media use seemingly not driven by clear, overt motivations. In addition, work on personality traits and media use indicates that there is relationship between the existence of specific traits and communication behaviors (e.g. temperament and television viewing) yet scholars have not begun to dissect the development of these personalities traits. Behavior genetics research demonstrates that there are both genetic and environment influences on personality traits, and that traits "are genotypically influenced latent characteristics of persons that determine the way in which individuals respond to the social world they encounter" (Brody, 1994, p. 119). This project delves deeper into the examination of these characteristics, motivations, needs, in order to provide a multifaceted understanding of individual communication behaviors.

Sherry (2001) points out that the communication field trails behind other social science researchers of human behavior. While other areas of social science have begun focusing on, and found, the genetic influences on variety of different behaviors (Scarr, 1992), communication scholars have yet to really consider genetic-specific questions. Weber, Sherry, and Mathiak (2008, p. 43) argue for a paradigm shift toward a "neurophysiological perspective" in order to better explain communication behaviors and they urge communication scholars to employ more advanced quantitative skills in order to advance the field. This project heeds their suggestion with the understanding that incorporating a genetic understanding does not render extant communication theories useless, but rather, augments existing theory through a more complex understanding of the motivation underlying media use: "If communication researchers continue to remain enamored of an early twentieth century ontology and ignore the building evidence of biological influence on behavior, our theories risk becoming outmoded" (Sherry, 2004, p. 102).

In their seminal article on political ideology and genetics, Alford et al. (2005) assert social scientists have a responsibility to consider the widespread genetic and environmental antecedents of human behavior. Because the majority of prior research on communication behaviors deals with environmental influences, we are left with an incomplete picture of the underlying causes of media use. We argue that it is necessary to expand research on these behaviors to consider genetic influences as well as environmental ones. While studies have shown that relationships do exist between biologically rooted traits and communication behaviors, the role of both types of influences (genetic and environmental) is unclear. This article builds on previous research by identifying the genetic underpinnings of communication behaviors. We start by describing the logic of the classical twin design (CTD), one of the multiple ways to examine the effects of genes on behavior, and turn to describing how we model the effects of genes in the specific context of media use and other communication activities.

Behavior genetics and the twin paradigm

Previous research on communication behaviors implies inherent personality traits explain why an individual engages in a particular communicative or media activity.

Many traits, either directly or indirectly, are the results of genetic and environmental factors (Caspi et al., 2002). One focus of behavioral genetics is decomposing the total variation of a trait in a behavioral trait into variance that is environmental and genetic. We begin with defining conventional terms used in this type of research and then discuss the statistics required for understanding biometric modeling. We then turn to how these methods are specifically applied to media consumption habits.

At the most basic level, a ''gene'' is the unit of inheritance that influences a ''phenotype,'' or the observable and measurable trait (Fuller & Thompson, 1978). In our case, the phenotypes are media use, interpersonal communication, and other communication variables.[2] To some, research investing genetic influence on physical characteristics (e.g. height, body mass index, hair color) may seem quite plausible, whereas psychiatric conditions (e.g. depression, attention deficit hyperactivity disorder) and social behaviors are less obvious candidates for such research. However, a great deal of work has shown that variation in nonphysical characteristics is to some degree heritable (Eaves et al., 1989).

As Alford et al. (2005) explain, because genes instruct the productions of proteins, they are directly involved in the chemical reactions within the human body: ''Each protein has a chemical sequence that interacts with other chemicals in the body, sometimes reacting directly with these other chemicals but often serving as enzymes that facilitate but are not directly altered by chemical reactions. If a gene coding for a particular enzyme is absent, the chemical reaction it is meant to enhance will occur with less efficiency'' (p. 154). In fact, researchers have shown that genes influence psychological traits (Bouchard, 2004; Eaves et al., 1989) and social behaviors (Freese & Shostak 2009; Freese, 2008).

Extending this work, we expect that communication behaviors will also be partially heritable. As discussed earlier, previous research demonstrates that personality traits can serve as strong predictors of media use and other communicative behaviors. For example, extraversion, neuroticism, and openness to experience have been found to predict the use of social networking sites (Ross et al., 2009; Zywica & Danowski, 2008). Therefore, it is perfectly viable that a series of genes, which contribute to proteins and subsequent chemical reactions, could influence personality traits that affect communication behaviors. As Sherry (2004) points out, mass communication scholars have long ignored the role of biology in understanding individual differences in communication behaviors and only recently have adopted a trait perspective in their studies. While this project focuses on the genetic and environmental variation instead of identifying the specific biological traits that influence communication behaviors, it compliments Sherry's (2004) argument by adding to the nature/nurture discussion. To examine both genetic and environmental influences on communication behaviors, we focus on nine media use and communication items from three different studies and rely on a common method in behavioral genetics, the CTD.

CTD (Boomsma, Busjahn, & Peltonen, 2002) allows researchers to decompose the sources of variation in a trait, and infer the extent to which this variation is attributable to genetic and environmental factors. The CTD does not allow us to tell how many

genes or what genes affect the specific manifestation of a particular trait. While we can generate an estimate of the percentage of the variance explained by environmental and genetic factors, we cannot ascertain whether a particular genetic characteristic leads to an increase or decrease in the mean of a particular trait. This distinction is important. For example, we are not arguing that a particular genetic characteristic leads to media use. We do not purport to identify a "media use gene," rather we explore whether genetic characteristics account for variation in media consumption.

The CTD is informed by the basic genetic structures of the two different types of twins: identical, or monozygotic (MZ) twins, and fraternal, or dizygotic (DZ). MZ twins originate from the same egg, which is fertilized by the same sperm. These twins share 100% of their genetic makeup. Dizygotic (DZ) twins develop from separate eggs, fertilized by different sperm (Hall, 2003; Snieder, Wang, & MacGregor, 2010). As such, DZ twins share 50% of the their genes, which is the same, on average, as any pair of full siblings. The CTD allows researchers to compare MZ twins to DZ twins to obtain an estimate of how much variation in a particular characteristic is genetic. In a CTD, sets of twins occupy similar environments; they are raised in the same households and are exposed to similar early life experiences. As such, if variation in a phenotype has a strong genetic component, the correlation between MZ twins' should be much higher than DZ twins'. However, if variation in a phenotype is dominated by environmental influence, the cotwin correlation would be the same between both MZ and DZ twin pairs (Martin & Eaves, 1977).

Recognizing these differences in correlations for MZ and DZ twins allows investigators to construct empirical models identifying the proportion of the variation in a phenotype that is genetic and environmental. When single phenotypes are examined, observed variation is broken into two sources of variation: genetic and environmental (Posthuma, 2009). With information from MZ and DZ twins, behavior geneticists can estimate the influence of genetic and environmental components.

In this framework, there are two types of genetic variance, both additive genetic variance (A) and dominance genetic variance (D).[3] Many twin studies focus on additive genetic variance (A), which represents the combined influence of one or multiple genes. Likewise, there are two types of environmental influences contemplated by classic twin studies. The variation that remains after accounting for the genetic variance can be decomposed into variance attributable to the common environment (C)—that is, variance shared by each individual in the twin pair—as well as environmental variance unique to each twin (E). Twins, in actuality, experience a common environment (C). Common environmental influences include the rearing style of parents, shared schooling and religious observance the twins experience, and the culture of the place in which the twins reside, assuming they are raised together. Following other CTD studies, we assume the common environment is the same for MZ and DZ twins. This assumption is the *equal environments assumption* (EEA).

By making this assumption, one can then compare the correlation between MZ twins and DZ twins. If MZ twins are more similar, it is assumed that the similarity is due to a greater genetic similarity. However, observers may anticipate that MZ twins

have a sufficiently different upbringing than DZ twins to violate this assumption. For example, MZ twins may be dressed alike while DZ twins are not. Medland and Hatemi (2009) review a multitude of approaches used to test the EEA, such as directly observing families to detect the extent to which MZ are actually treated differently, using more extensive data to decompose the common environment relaxing the assumption that twins who perceive themselves to be DZs experience family life differently than those who perceive themselves to be MZ, and using more extensive data to examine heritability among nonsibling twins. They also outline a useful thought exercise applied to political phenotypes: ''it is difficult to conceive of a population where parents of MZ twins would purposely or unconsciously socialize their children to support the same political party, whereas parents of DZ twins, simply because the twins do not look alike or are not genetically identical, socialize them for opposing political values'' (Medland and Hatemi, 2009, p. 199).

While we also cannot imagine a scenario in which parents would treat MZ and DZ children differently in socializing them to use media, we do account for gender differences because several CTD studies have found that there can be differences in the way parents treat DZ twins of different genders in terms of social behaviors. For this reason, modern CTD studies typically separate analyses of male MZ/DZ twins, female MZ/DZ twins, and DZ twins of different genders. In order to account for these differences in our own study, we only analyze same-sex twin pairs and we test for effects of gender across the phenotypes, the results of which are in our results section. Following these theoretical assumptions, the *A*, *C*, *D*, and *E* variance components can be estimated using structural equation modeling (SEM) (for a review, see Medland & Hatemi, 2009).

Estimation and model assumptions

Behavioral genetics researchers commonly use SEM to estimate ''variance components'' for phenotypes using twin study data (Medland & Hatemi, 2009; Neale & Cardon, 1992; Neale & Maes, 2004). SEM allows us to decompose the variance matrix into variance explained by genes and variance explained by both common and unique environmental factors. The models can be estimated using either maximum likelihood (Martin & Eaves, 1977; Medland & Hatemi, 2009) or Bayesian methods (Eaves et al., 2005; Fowler et al., 2008). SEM is advantageous for many reasons. It allows the researcher to model complex relationships, such as situations where genes interact with the environment. It also allows the researcher to control for assumption violations inherent in certain circumstances, such as when the data are categorical. In the case of continuous variables, the ACE estimates will be the same using the Falconer (1960) equations, which others use (Alford et al., 2005), but the SEM approach is more flexible by allowing the researcher to test parameter restrictions and violations to the assumptions underlying the CTD. Finally, it is more conducive to hypothesis testing as standard errors are estimated and the *A*, *C*, *E*, and *D* components can each be given a confidence interval (Medland & Hatemi, 2009).

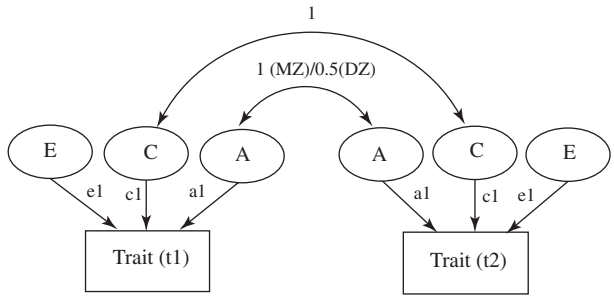

Figure 1 Model specification for the ACE model.

When studying twin pairs using SEM, there are limits to the numbers of parameters that can be empirically identified. For example, twin studies with data for twins raised in the same household forces researchers to choose between estimating models that test for the variation associated with the common environment (C) or dominant genetic influence (D) (Neale & Cardon, 1992). The data we use here focuses on twins raised together. Consequently, for each of the models below, we chose to estimate ACE specifications, characterizing additive genetic influences as well as the common and unique environment. Across our models we do not find a considerable difference in model fit when comparing an ACE to an ADE model.[4]

Figure 1 shows a structural equation modeling for the ACE model.[5] Several model specifications are made prior to estimating the genetic and environment variance components. The ACE model fixes the covariance between the MZ twins' additive genetics at 1, given that MZ twins share identical genes. On the other hand, the covariance between DZ twins' additive genetics is set at 0.5, because DZ twins have about half of the same genes, on average. To account for the common environment, the covariance for both MZ and DZ twins' is 1; this variance component represents the influence of the environment shared by both twins. Finally, the E component is the residual, the environment unique to each individual in the twin pair.

The parameter estimates linking $a1$, $c1$, and $e1$ to the observed trait are specified to be equal across twins, as there is no reason to expect different coefficients for twin 1 and twin 2. Finally, the means for A, C, and E are set at 0 and the variances are constrained at 1, which is necessary to statistically identify the model (Kaplan, 2000). From the model in Figure 1, the total variance in the trait is given by squaring the path coefficients of $a1$, $c1$, and $e1$ and then summing these terms, σ^2 trait $= a^2 + c^2 + e^2$. From this, we calculate the percentage of the total variance attributable to each orthogonal component. For instance, the variance explained by additive genetic factors is $a^2/(a^2 + c^2 + e^2)$; for the common environment, it is $c^2/(a^2 + c^2 + e^2)$; for the unique environment, it is $e^2/(a^2 + c^2 + e^2)$.

Finally, if one of the variance components is not significantly discernable from zero, then a researcher should prefer a more parsimonious model where the nonstatistically significant component is dropped. For instance, if additive genes (a^2) and

the unique environment estimate (e^2) account for much of the variance, and (c^2) accounts for little, one should prefer an AE model over an ACE model, as it is a more parsimonious model. Likewise, if additive genetic influence (a^2) accounts for a trivial portion of the variance, one should prefer CE model, which is tantamount to stating that the common and unique environment account for the total variance. One can compare the ACE model to the AE and CE models by comparing model fit with a likelihood ratio test.[6] If the model fit is not significantly worsened by dropping a parameter, then the model without that parameter should be preferred. As such, we estimate an ACE model for each communication variable, as well as the reduced AE and CE models.

Data

We rely on two datasets to examine the heritability of communication and media consumption behaviors. The first is The National Longitudinal Study of Adolescent Health (Add Health), which is a panel study that begins in youth and extends to adulthood (Harris, 2008). Participants ranged in ages from 18 to 28 in Wave III. During Waves III and IV, several communication and consumption behavior questions were included on the survey. For this reason, these are the only two waves useful in ascertaining the genetic contribution to these behaviors. In the data, we drop twin pairs if one or both twins do not participate in both waves and as previously mentioned, we only analyze same-sex pairs. The total number of same-sex twins who participated in Wave III was $n = 865$ individuals. Of those, $n = 443$ were MZ and $n = 412$ were DZ twins. The total number of same-sex twin pairs who participated in Wave IV was $n = 873$ (441 MZ and 432 DZ twins). The Wave III data was collected between 2001 and 2002, and the Wave IV data was collected from 2007 to 2009.

We also rely on the Midlife in the United States National Study of Health and Well-Being data from 1995 to 1996 (MIDUS). These data include a number of behavioral and psychological questions pertaining to physical and psychological health. Unlike the Add Health data, these data focus on behavioral and psychological changes through adulthood. While the sample is large, consisting of over 7,000 adults, MIDUS includes only a subsample of twin pairs. We identified $n = 1382$ individuals that had same-sex twin pairs (684 MZ and 698 DZ twins).

We focus on nine media behavior and communication items from these studies. Full question wording for all nine items is available in the Appendix. Specifically, we look at computer use, television-viewing habits, one's perceived importance of relying on the news, and interpersonal communication habits. Because we are dealing with secondary data, many of the habits are assessed with single questions, rather than multi-item scales. While this is a limitation of our data, we are able to account for this type of measurement error by focusing on nine different items in two unique datasets. The majority of the consumption questions asked respondents to estimate the number of hours consuming particular media per week, with the exception of the single computer use item. Computer use and days watching television per week

were both coded from 1 to 7 indicating how many days an individual engages in that activity per week.

The interpersonal communication questions gauged individuals' comfort with interacting and communicating with others. The questions were framed in terms of "How much do you agree or disagree with the following statement, with agree indicating high levels of comfort. The responses were coded from one to five, ranging from strongly agree to strongly disagree. The single news reliance item asked respondents how important it was for them to be informed about the news and was coded from one to ten. Because of the nonnormality of the media consumption per week questions, we transformed these variables by first adding 1 and then calculating natural logarithms for each respondent.[7]

Understanding previous literature on media consumption and communication behaviors, as well as the model specifications of classic twin studies, we propose the following research question:

What proportion of the variance in the nine communication traits can be attributed to genetic (A), common environment (C), and unique environment (E) components?

Results

Table 1 presents the descriptive statistics for our variables of interest. The columns of means and the 95% inferential confidence intervals (ICIs) allow us to compare the average scores for DZ and MZ pairs (Levine, Weber, Hullett, Park, & Lindsey, 2008; Levine, Weber, Park, & Hullett, 2008; Tryon, 2001). In every case, the inferential confidence intervals overlap, suggesting no difference between MZ and DZ twins (at the alpha = 0.05) level.[8] Table 1 also presents the correlations between MZ and DZ twins. For these variables, the MZ twin correlation is consistently greater than the correlation between DZ twins, pointing to a genetic component underlying variation on all nine of these traits.

The genetic foundations of communication habits

The additive genetic, common environment, and unique environment variance components for the nine items were estimated using SEM. All models were estimated using maximum likelihood with M*plus* version 6,11 (Muthen & Muthen, 1998–2011). Because M*plus* employs full information maximum likelihood, we include cases where one twin is missing from the data, though our results are identical if we use listwise deletion. As noted in Figure 1, several specifications were made prior to estimation. Following these specifications we report the standardized variance components and 95% confidence intervals (CI) in Table 2. We also report several indicators of model fit: The Akaike Information Criterion (AIC), the Bayesian Information Criterion (BIC), and $-2 \times$ log-likelihood.

Table 2 reports multiple models: ACE, CE, and AE specifications. We compare the fit of the nested CE and AE models to the ACE model. A nonsignificant change in

Table 1 Descriptive Statistics for Add Health and MIDUS Data

Media Variable	Y_{DZ}	Y_{MZ}	r_{DZ}	r_{MZ}
Add Health Wave III				
Computer use	3.84	3.94	0.18	0.33
	(3.63, 4.01)	(3.78, 4.09)		
Video	1.40	1.37	0.08	0.32
	(1.34, 1.46)	(1.31, 1.43)		
Days TV	5.48	5.31	0.09	0.25
	(5.33, 5.63)	(5.17, 5.45)		
Hours TV	2.28	2.29	0.10	0.38
	(2.21, 2.35)	(2.23, 2.35)		
Add Health Wave IV				
Internet	1.39	1.44	0.27	0.33
	(1.32, 1.46)	(1.37, 1.51)		
Hours TV	2.28	2.21	0.12	0.22
	(2.20, 2.34)	(2.15, 2.26)		
Talk to others	3.47	3.43	0.12	0.31
	(3.40, 3.54)	(3.37, 3.48)		
Parties	3.42	3.34	0.14	0.20
	(3.34, 3.49)	(3.27, 3.41)		
MIDUS				
News	7.12	6.96	0.22	0.34
	(6.98, 7.26)	(6.82, 7.10)		

Note: Point estimates and 95% inferential confidence intervals (Tryon, 2001) in brackets. DZ = dizygotic twin pairs; MZ = monozygotic twin pairs. Computer and Days TV were coded as number of days per week and coded from 0 to 7. The interpersonal communication questions are coded from 1 to 5. All other variables were coded in hours of consumption per week, and these variables were then natural log transformed because of positively skewed distributions. Zero-order correlations are for MZ and DZ twins.

model fit suggests a more parsimonious model, indicating it is the preferred model. A large change in fit indicates that dropping the *A* or *C* parameter worsens the model. We compare the CE and AE models to the full ACE model by conducting a likelihood ratio test. The best-fitting model is in bold.[9]

The first column in Table 2 represents the proportion of variance explained by genes. Across the board, the a^2 estimates indicate a strong and pervasive effect of genes on these media consumption behaviors. Consider the proportion of the total variance of computer use explained by genetic factors, relative to common or unique environmental factors: Thirty-four percent of the variance is explained by additive genetic factors (95% CI: 0.23–0.45). Similarly, variation in television-viewing behavior is also partially explained by genes. For example, 35% of the variance in hours spent watching TV (Wave III) is explained by genes (95% CI: 0.25–0.47), compared to 23% of individuals in Wave IV (95% CI: 0.11–0.35). Twenty-four

Table 2 ACE, AE, and CE Model Estimations

Variable	Model	a^2	c^2	e^2	AIC	BIC	−2LL	χ^2	p
Computer Use	ACE	0.34 (0.23, 0.45)	0 (0.00, 0.00)	0.66 (0.55, 0.77)	3,955.42	3,971.91	3,947.42	Base	—
	CE	—	0.26 (0.16, 0.35)	0.74 (0.65, 0.84)	3,957.36	3,969.73	3,951.36	3.94	0.05
	AE	**0.34 (0.23, 0.45)**	**—**	**0.66 (0.55, 0.77)**	**3,953.42**	**3,965.79**	**3,947.42**	**0.00**	**1.00**
Video	ACE	0.30 (0.19, 0.41)	0 (0.00, 0.00)	0.70 (0.59, 0.82)	2,190.67	2,207.15	2,182.68	Base	—
	CE	—	0.21 (0.12, 0.31)	0.79 (0.70, 0.88)	2,194.06	2,206.42	2,188.06	5.38	0.02
	AE	**0.30 (0.19, 0.41)**	**—**	**0.70 (0.59, 0.82)**	**2,188.67**	**2,201.03**	**2,182.67**	**0.01**	**0.92**
Days TV	ACE	0.24 (0.13, 0.36)	0 (0.00, 0.00)	0.76 (0.64, 0.87)	3,777.29	3,793.79	3,769.30	Base	—
	CE	—	0.18 (0.08, 0.27)	0.82 (0.73, 0.92)	3,778.05	3,790.41	3,772.04	2.74	0.10
	AE	**0.24 (0.13, 0.36)**	**—**	**0.76 (0.64, 0.87)**	**3,775.29**	**3,787.66**	**3,769.29**	**0.01**	**.92**
Hours TV	ACE	0.35 (0.25, 0.47)	0 (0.00, 0.00)	0.64 (0.53, 0.75)	2,216.37	2,232.85	2,208.38	Base	—
	CE	—	0.24 (0.15, 0.33)	0.76 (0.67, 0.85)	2,223.58	2,235.94	2,217.58	9.2	0.00
	AE	**0.36 (0.25, 0.47)**	**—**	**0.64 (0.53, 0.75)**	**2,214.37**	**2,226.73**	**2,208.38**	**0.00**	**1.00**
Internet	**ACE**	**0.10 (−0.26, 0.41)**	**0.23 (−0.07, 0.52)**	**0.67 (0.55, 0.80)**	**2,345.29**	**2,361.81**	**2,337.30**	**Base**	—
	CE	—	0.31 (0.21, 0.40)	0.70 (0.60, 0.79)	2,343.60	2,355.99	2,337.60	0.3	0.58
	AE	0.36 (0.25, 0.47)	—	0.64 (0.53, 0.75)	2,345.40	2,357.78	2,339.40	2.1	0.15
Hours TV	**ACE**	**0.23 (0.11, 0.35)**	**0 (0.00, 0.00)**	**0.77 (0.65, 0.89)**	**2,307.36**	**2,323.93**	**2,299.36**	**Base**	—
	CE	—	0.17 (0.07, 0.26)	0.83 (0.74, 0.93)	2,274.57	2,306.81	2,300.81	1.45	0.23
	AE	0.23 (0.11, 0.35)	—	0.77 (0.65, 0.89)	2,305.36	2,317.36	2,299.36	0.00	1.00
Talk	ACE	0.29 (0.18, 0.40)	0 (0.00, 0.00)	0.71 (0.60, 0.82)	2,643.69	2,660.25	2,635.68	Base	—
	CE	—	0.22 (0.12, 0.31)	0.78 (0.69, 0.88)	2,645.02	2,657.44	2,639.02	3.34	0.07
	AE	**0.29 (0.18, 0.40)**	**—**	**0.71 (0.60, 0.82)**	**2,641.69**	**2,654.11**	**2,635.68**	**0.00**	**1.00**
Parties	**ACE**	**0.09 (−0.29, 0.47)**	**0.10 (−0.21, 0.41)**	**0.81 (0.68, 0.94)**	**2,585.24**	**2,601.81**	**2,577.24**	**Base**	—
	CE	—	0.17 (0.08, 0.27)	0.83 (0.74, 0.92)	2,583.48	2,595.90	2,577.47	0.23	0.88
	AE	0.21 (0.10, 0.33)	—	0.79 (0.68, 0.91)	2,583.65	2,596.08	2,577.65	0.41	0.52
News	**ACE**	**0.35 (−0.04, 0.53)**	**0.10 (−0.14, 0.33)**	**0.66 (0.57, 0.76)**	**5,823.28**	**5,841.35**	**5,815.28**	**Base**	—
	CE	—	0.28 (0.21, 0.36)	0.72 (0.64, 0.79)	5,824.04	5,837.59	5,818.04	2.76	0.10
	AE	0.35 (0.27, 0.44)	—	0.65 (0.56, 0.73)	5,821.91	5,835.46	5,815.90	0.62	0.43

Note: Wave III, Wave IV (Add Health), and MIDUS. ACE models followed by reduced form AE and CE models. Best-fitting models, as determined by a likelihood ration test, are in bold. AIC = Akaike Information Criterion, BIC = Bayesian Information Criterion. −2LL is −2 times the log-likelihood. 95% confidence intervals are in parentheses. a^2 = additive genetic factors, c^2 = common environmental factors, and e^2 = unique environmental factors. For Internet use, Hours TV, Parties, and News, the tests indicate that *A* or *C* can be dropped from the model. Simultaneously dropping both from the model significantly worsen the model fit; however, we note that it should be modeled as ACE.

percent of the total variance in days watching television per week is explained by genetic factors (95% CI: 0.13–0.36). Considering the ACE models, across all nine variables, the average proportion of the total variance explained by genetic factors was 23.67%, ranging from 9 to 35%.

What is also striking is the small role of common environment in media consumption behaviors. With the exception of Internet consumption, the c^2 estimates are negligible. The average contribution of the common environment to the total variance is 5.1%. And, for all nine variables, the 95% CIs overlap with 0. Moreover, when comparing the full ACE model to the reduced model, in most cases the AE model is the best-fitting model. This does not suggest the environment plays no role, just that common environmental factors operating on both twins, such as parenting and culture, play a minimal role in explaining the variance in these variables. Unique environmental factors (E), however, account for a sizable portion of the variance in communication behaviors. For instance, e^2 for computer use is 0.66 (95% CI: 0.55–0.77); for news consumption, 69% of the variance is explained by unique environmental factors (95% CI: 0.60–0.78); for number of days of TV consumption, 76% of the variance is explained by the unique environment (95% CI: 0.64–0.87). On average, 71.2% of the variance in these behaviors was explained by the unique environment, ranging from 0.66 to 0.81.

Because a parsimonious model should be preferred over a complex model, for each of the variables we compared models with fewer parameters to the ACE models. Specifically, we estimated reduced form CE and AE models for all nine variables. Note that the CE model is where genes explain no variation, and the AE model is where the common environment accounts for none of the variance. As the reduced models are empirically nested within the ACE model, we conducted a likelihood ration test examining whether dropping A or C worsens the overall fit. For seven of the nine media consumption variables, an AE model is preferred over the full ACE model, indicating that common environment does not account for any of the total variance. For two of the variables, Internet consumption and interpersonal communication at parties, neither an AE nor a CE were better fitting models suggesting that both the additive genetic component (a^2) and common environment component (c^2) significantly contribute to the total variance and should be included in the model.

Overall, we find a striking effect of genes and a minimal effect of the common environment on phenotypic variance. What this suggests is whatever role the environment plays in media use and communication socialization, it is idiosyncratic to the individual and not directly attributable to common socialization.

Discussion

Consistent with previous work on information consumption (Delli Carpini & Keeter, 1996), we find that media consumption and communication behaviors cannot be solely explained by environmental factors external to information users. Using

nine media traits from two unique datasets, we find that a nontrivial portion of variation is explained by genetic factors. We estimate that one fifth to one third of the variance in media consumption and communication behaviors are explained by additive genetic factors. Our findings support previous research suggesting that there are strong individualized components that underlie the motivations behind media use and communication behaviors. The results also extend previous research by not only suggesting that these media behaviors are individualized and driven by specific motivations, which we demonstrate are partially explained by heredity.

This does not suggest that there is a ''media consumption gene,'' on the contrary, a^2 represents the joint contribution of multiple genes, which likely influence the development of personality traits and other intrapersonal characteristics. As previously scholars have demonstrated (Sherry, 2001), different media use behaviors are influenced by personality traits. Our study adds to these findings by suggesting that the development of such personality traits is partially determined by genetics.

Similarly, the findings should not be misconstrued as evidence of genetic determinism—that one's media behaviors are predetermined and entirely contingent upon one's parents. While a substantial amount of variation in media consumption and communication behaviors can be attributed to genes, the unique environment explains a much larger percent of the variation. On average, 23.67% of the total variance can be explained by additive genetic factors, while a significant portion of the remaining variance was explained by unique environmental factors. As such, our study moves us closer to better understanding how important each of these components are in our communication behaviors like media use, news consumption, and interpersonal communication.

Equally important, we find that the common environment accounts for very little of the total variance. For most of the communication behavior variables, an AE, not an ACE, model was the best-fitting model. This demonstrates that the role of common environment is small in explaining the total variance. This is consistent with LaRose (2010) who argues ''habits'' play an important part in shaping media use behaviors. A nonsignificant c^2 does not mean that culture, socialization, or environmental factors operating are not important. Rather, the effect of these factors may be responding differently and subsequently captured in the unique environment (e^2) term.

In other words, if two twins responded to the same event or environmental circumstances different, this would be represented as unique environmental variance. For our models, the nonsignificant c^2 term indicates that environmental variables that are responded to by both twins, plays a very little role in explaining variation in media use, news consumption, and interpersonal communication.

Consequently, our finding that additive genetic and unique environmental factors explain the vast majority variance in media consumption dovetails with previous work demonstrating a genetic component to forms of political participation (Fowler

et al., 2008), political efficacy (Funk, Smith, Alford, Hibbing, Hatemi, & Hibbing, 2010), and voter turnout (Loewen & Dawes, 2010). It is also consistent with Gerber et al.'s (2010) research suggesting that personality traits are significant predictors of information-seeking behaviors.

It is important to underscore the limitations of our findings. Our nine media use and communication variables are single-item questions and rely on the accuracy of self-reporting. There has been significant research demonstrating self-report bias in measures of media exposure. As Prior (2009, 2010) explains, while media exposure is a central variable in mass communication research, the most common tool used to measure this variable—survey research—suffers from issues of validity. While we acknowledge this, we are less interested in the single-item questions and more intrigued with the notion that there is an underlying personality trait of media use, interpersonal communication, and news attentiveness. Therefore, while each trait is measured using a single question, the consistency of our results across the majority of the traits provides the validity in our results that analyzing each individual question cannot.

We are cautious in our interpretation of these results. We estimate heritability coefficients ranging from 0.09 to 0.34, suggesting that given behaviors could be as much as 34% heritable in the populations from which we have data. Our principal inference is that genes influence elements of media use and communication behavior, across a variety of contemporary behaviors, given the present data. Conclusions much beyond this will require much more replication and scientific study.

We are cognizant of long-standing criticism of twin studies, which often revolve around potential violations of the model's assumptions, other empirical issues, as well as the validity and meaning of estimated heritability. We have discussed the EEA that the MZ and DZ twins do not have systematically different experiences inside common environments, perhaps the most controversial assumption of the CTD (Beckwith & Morris, 2008; Horwitz, Videon, Schmitz, & Davis, 2003); however, we agree with Medland and Hatemi (2009) that it is theoretically unlikely parents would intentionally subject different types of twins to systematically different socialization to political and social behaviors. In addition, we have attempted to ease concerns about violations of the EEA by using inferential confidence intervals in Table 1 (Tryon, 2001; Levine, Weber, Hullett, et al., 2008).

There are criticisms of twin studies beyond concerns about the EEA. For example, much of the data we use rely on self-reported zygosity.[10] Some of the twins in these samples may have misperceived their own zygosity. Critics of this research program also assert that the particular kind of twin study we use cannot imply causality. That is not an unreasonable criticism insofar as we are using these data from cross sections, estimating and comparing shares of variation. However, our findings are consistent with the hypothesis that media use is influenced by genetic backgrounds in a population because more genetically similar people are more alike than less genetically similar people. The data lends support to our expectations, but in the best

light demonstrate genetic effects, rather than trace out genetic mechanisms. More precise genome-wide association studies and other research identifying detailed mechanisms for complex social behaviors and judgments are just underway (e.g., Fowler & Dawes 2008, Hatemi et al., 2011, McDermott, Tingley, Cowden, Frazzetto, & Johnson, 2009). Additional concerns such as the fact that twin samples are convenience samples (Hibbing & Smith, 2011) rather than representative of an underlying population of interest[11] (e.g. residents of the United States) ultimately contribute to concerns about the degree of influence from each of these quarters (genes, culture, environments).

The CTD also assumes limited or absent interactions between the gene and environment (a GxE interaction) and no correlation between genes and the environment (rGE). There is evidence that environments can be sufficiently influential on a population or subset of a population to severely depress the expression of heritability, among other moderating effects (Shanahan & Hofer, 2005). For instance, scholars have demonstrated that the estimated heritability of intelligence decreases as family socioeconomic status decreases (Turkheimer, Haley, Waldron, D'Onofrio, & Gotesman, 2003; Harden, Turkheimer, & Loehlin, 2006). This basic pattern of environment moderating the genetic influences has been demonstrated on other traits (Boomsma, de Geus, van Baal, & Koopmans, 1999; Heath et al., 1985; Caspi et al., 2002). Students of media psychology should be attentive to the possibility that environments moderate heritability as it will be an important avenue for future research. That said, preliminarily we expect environments will affect the degree to which genes and environments are influential, not whether each has influence at all.

We echo points made by Alford, Funk, and Hibbing (2008) who defend that behavior geneticists engage in "honest attempts to wrestle with the limitations of the twin design along with empirical tests" (Alford et al., 2008, p. 793). Classic (Kendler, Neale, Kessler, Heath, & Eaves, 1993) and contemporary (Hibbing & Smith, 2011) research shows broad empirical support for the EEA, but behavior geneticists remain circumspect about their own research. Second, while skepticism is in order, wholesale rejection of this research program is not. Alford et al. (2008) argue most of the criticisms we have cataloged here ultimately raise a host of active empirical questions. Clearly we have more to learn about the interplay of social, cultural, and genetic influences on behavior.

Media consumption and communication traits are strong predictors of a variety of political and social behaviors. Understanding their origin is a key component of this research. This project seeks to provide more insight into this nuanced and complex research area. By incorporating genetic components into our research, we are better able to explain many facets of human behavior including communication behaviors. Previous scholars (Sherry, 2004; Weber, Sherry, & Mathiak, 2008) have urged scholars to expand our study of communication-related behaviors beyond social learning theory in order to provide a more complex and complete epistemology. As DeFleur (1998) argues, communication scholars gain in advancing out theoretical understanding by expanding our methodology to include the methods

and epistemology of the natural sciences. Only with this greater degree of scrutiny, through a better recognition of the role that genes and environment play in social life, will we more fully understand the factors that contribute to staying informed and cultivating and maintaining connections to others.

Notes

1 It is important to note that while Loehlin and Nichols' (1976) study included thousands of different items, it measured television viewing using a sole indicator. In addition, correlation between identical twins was only slightly higher ($r = 0.49$) than it was for fraternal twins ($r = 0.38$).
2 Inside a cell's DNA, genes are gathered into long strings, or chromosomes. These are ordered sequentially with genes residing in specific places along a chromosome. The site a gene occupies on a chromosome is its locus. The forms of genes occupying the same loci on a chromosome are alleles (see, e.g., Alford et al., 2005; Fowler & Dawes, 2008).
3 Dominant variance results from the nonadditive (i.e. interactive) genetic influence (Plomin, Defries, Graig, & McGuffin, 2001).
4 We compared the ACE to the ADE model for all analyzed variables and our analysis can be found in the Appendix. Since the models are not nested, one cannot conduct a likelihood ratio test. However, it is possible to compare the Akaike Information Criterion (AIC) and the Bayesian Information Criterion (BIC). The change in model fit is often negligible rendering it difficult to empirically favor one model over the other. Therefore, our results presented are from the ACE models but the ADE parameter estimates are located in the Appendix.
5 An ADE model is similar. The *D* covariance for MZ twins is constrained to 1 and DZ twins is constrained to 0.25 because MZ twins inherit the same genes, while DZ twins inherit the same pattern of genes a quarter of the time (on average).
6 This is because the reduced AE and CE models are nested in the ACE model. The LR test is distributed chi-squared with one-degree of freedom.
7 For the variables coded from 0 to 7, 1 to 5, and 1 to 10, we estimated a liability-threshold model, which is used when data are categorical (Neale, 2009). Specifying the models in this way also did not substantially change the ACE estimates. We also re-estimated the models that were natural log transformed. The parameter estimates again were quite similar, with the exception of the Internet and TV items in Add Health, Wave IV. We find that on the raw data, a CE model provides the best fit, though this should be interpreted with caution, since the data are extremely skewed. Finally, we transformed the variables where responses were given in hours. Specifically, we created two category variables based on a median split. Again, we find that the point estimates in the ACE models are remarkably similar, though the standard errors are greater, which is expected given the information that is lost in converting a continuous variable to a categorical variable. We take this to suggest that our findings are robust to different model specifications and not a function of the way that data are distributed.
8 As Tryon and Lewis (2008) note, overlapping confidence intervals (descriptive or inferential) are not sufficient to conclude statistical equivalence (see also, Tryon, 2001). As Tryon (2001, p. 379) notes, "the absence of positive evidence for statistical difference does not constitute presence of positive evidence for statistical equivalence." Building

on Schuirmann (1987), Tryon and Lewis (2008) note that one can test for statistical equivalence by (1) first calculating Δ from the upper and lower bounds of the descriptive and inferential confidence interval for MZ and DZ twins (see Tryon & Lewis, 2008, for formulae); and (2) using Δ to test the null hypothesis that the difference between means is greater than Δ, where $[t_1 = \frac{(\bar{Y}_{hi}-\bar{Y}_{low})-\Delta}{s_{dif}}, t_2 = \frac{(\bar{Y}_{hi}-\bar{Y}_{low})-(-\Delta)}{s_{dif}}]$ and each t-value is compared to $\pm t_{\alpha/2}$. If the two calculated t-value exceeds the critical t-values one can conclude there is statistical equivalence (Tryon & Lewis, 2008). For most of our variables, we are unable to conclude that the means are equivalent, since t_1 and t_2 are not larger than $\pm t_{\alpha/2}$. For these variables, we are in the "indeterminate" range, where we have evidence to reject the null that the means are not equal, but insufficient evidence to posit that the means are equal. Thus, we can conclude that the equal environments assumption (EEA) is not violated, but cannot simultaneously conclude that the EEA is true. We proceed assuming no violation of the EEA.

9 As previously mentioned, several previous CTD studies have found gender differences across the traits. In order to test for this, we analyzed whether the ACE estimates varied across male and females. For seven of the nine variables, we find no evidence of sex differences with respect to additive genetic, common environment, and unique environmental estimates. For computer use in Add Health, Wave III, we find very small differences in ACE estimates for males and females ($\chi^2[3] = 6.34$, $p = .10$) and the importance of the news in MIDUS ($\chi^2[3] = 7.42$, $p = .06$). In both cases, the estimate for a^2 is higher for males than females, whereas c^2 is slightly higher for females than males.

10 The MIDUS zygosity data is based on self-report and while some of the Add Health respondents were paired with their twin and classified using a genetic test, many were matched and coded as MZ or DZ by self-report.

11 We rely on samples that are both slightly younger (Add Health) and older (MIDUS) than the general U.S. population. Of greater concern is that twins are themselves systematically different from the overall population. For example, twins tend to have slightly lower performance on standardized IQ tests than singletons, (Ronalds, De Stavola, & Leon, 2005). However, this IQ difference does not account for the differences across *types* of twins we observe. Nonetheless, it may suggest the need for additional research on how physiological development may affect communication behaviors.

Appendix

Question wording

Add health: Wave III

[Computer Use; H3DA3] In the past 7 days how many times did you watch a movie, play video or computer games, or use a computer for surfing the web, exchanging e-mail or participating in a chat room?

[Video; H3DA4] On the average, how many hours a week do you spend watching videos?

[Days TV; H3DA6] In the past 7 days, how many times did you watch television?

[Hours TV; H3DA7] On the average, how many hours a week do you spend watching television?

Add health: Wave IV

[Internet; H4DA22] In the past 7 days, how many hours did you spend using the Internet? For example, accessing your e-mail and using the web. Do not count Internet use for work or school.

[Hours TV; H4DA1] In the past 7 days how many hours did you watch television or videos, including VHS, DVDs or music videos?

[Talk; H4PE9] How much do you agree with each statement about you as you generally are now, not as you wish to be in the future? I don't talk a lot.

[Talk to others at Parties; H4PE17] I talk to a lot of different people at parties. MIDUS

[Importance of News; A1SK7J] How important is it to you to keep informed about national news?

Alternative models

In addition to the ACE models presented in the table, we also present ADE models, which model the effect of additive and dominant genetic factors. The ADE model follows a specification similar to the ACE, with the exception of the specified cross-twin covariances for the *D* term (which replaces the *C* term). For MZ twins, the covariance is specified at 1, as identical twins share 100% of dominant genes; for DZ twins, the covariance is set at 0.25, as fraternal twins share 25% of dominant genes, on average. We present the path diagram for the ADE specification in Figure A1.

Typically, an ADE model is specified when two times the correlation for DZ twins is less than the correlation for MZ twins. For some of our variables, this is the case (see Table 1). We subsequently reran all our models as ADE models and input the results in Table A1. Across the board, we do not find any evidence to suggest that an ADE model fit the data better than an ACE model. By comparing the AIC and BIC across model specifications, the change never exceeds 2. Thus, we opted to present all ACE models for consistency, subsequently leaving the ADE models to the Appendix.

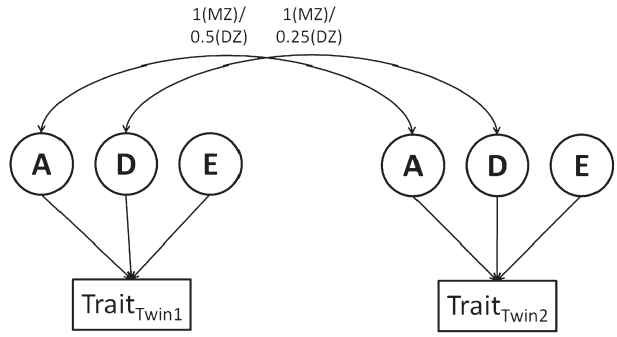

Figure A1 Model specification for the ADE model.

Table A1 Comparing ACE and ADE Models

Variable	Model	a^2	c^2	d^2	e^2	AIC	BIC	−2LL
Computer use	ACE	0.34 (0.23, 0.45)	0 (0.00,0.00)	—	0.66 (0.55, 0.77)	3,955.42	3,971.91	3,947.42
	ADE	0.31 (−0.23, 0.86)	—	0.03 (−0.54, 0.32)	0.65 (0.54, 0.61)	3,955.41	3,971.90	3,947.40
Video	ACE	0.30 (0.19, 0.41)	0 (0.00,0.00)	—	0.70 (0.59, 0.82)	2,190.67	2,207.15	2,182.68
	ADE	0.01 (−0.57, 0.59)	—	0.3 (−0.30, 0.82)	0.68 (0.56, 0.80)	2,189.67	2,206.15	2,181.68
Days TV	ACE	0.24 (0.13, 0.36)	0 (0.00,0.00)	—	0.76 (0.64, 0.87)	3,777.29	3,793.79	3,769.30
	ADE	0.10 (−0.48, 0.68)	—	0.15 (−0.46, 0.77)	0.75 (0.62, 0.87)	3,777.05	3,793.54	3,769.06
Hours TV	ACE	0.35 (0.25, 0.47)	0 (0.00,0.00)	—	0.64 (0.53, 0.75)	2,216.37	2,232.85	2,200.00
	ADE	0 (−0.01, 0.01)	—	0.40 (0.28, 0.51)	0.61 (0.49, 0.72)	2,214.24	2,230.72	2,206.24
Internet	ACE	0.10 (0.26, 0.41)	0.23 (0.07, 0.52)	—	0.67 (0.55, −0.80)	2,345.29	2,361.81	2,312.40
	ADE	0.36 (0.25, 0.47)	—	0 (0.00, 0.00)	0.64 (0.53, 0.75)	2,347.40	2,363.40	2,327.96
Hours TV	ACE	0.23 (0.11, 0.35)	0 (0.00,0.00)	—	0.77 (0.65, 0.89)	2,307.36	2,323.93	2,299.36
	ADE	0.23 (−0.32, 0.78)	—	0.01 (−0.59, 0.60)	0.77 (0.63, 0.91)	2,307.36	2,323.93	2,299.36
Talk	ACE	0.29 (0.18, 0.40)	0 (0.00,0.00)	—	0.71 (0.60, 0.82)	2,643.69	2,660.25	2,635.68
	ADE	0.34 (0.23, 0.45)		0 (0.00, 0.00)	0.66 (0.55, 0.77)	3,955.42	3,971.91	3,947.42
Parties	ACE	0.09 (−0.29, 0.47)	0.10 (−0.21, 0.41)	—	0.81 (0.69, 0.93)	2,585.24	2,601.81	2,577.24
	ADE	0.21 (0.10, 0.33)	—	0 (−0.01, 0.01)	0.79 (0.68, 0.91)	2,585.65	2,602.22	2,577.65
News	ACE	0.18 (−0.10, 0.46)	0.13 (−0.10, 0.36)	—	0.66 (0.57, 0.76)	5,823.28	5,841.35	5,815.28
	ADE	0.35 (0.27, 0.44)	—	0 (0.00, 0.00)	0.65 (0.56, 0.73)	5,823.91	5,841.97	5,815.9

Note: Wave III, Wave IV (Add Health), and MIDUS. ACE and ADE models. AIC = Akaike Information Criterion, BIC = Bayesian Information Criterion. −2 LL is −2 times the log-likelihood. 95% confidence intervals are in parentheses. a^2 = additive genetic factors, c^2 = common environmental factors, d^2 = dominant genetic factors, and e^2 = unique environmental factors.

References

Alford, J., Funk, C., & Hibbing, J. (2005). Are political orientations genetically transmitted? *American Political Science Review*, **99**, 153–167.

Alford, J. R., Funk, C. L., & Hibbing, J. R. (2008). Twin studies, molecular genetics, politics, and tolerance: A response to Beckwith and Morris. *Perspectives on Politics*, **6**, 793–797.

Althaus, S., & Tewksbury, D. (2000). Patterns of Internet and traditional news media use in a networked community. *Political Communication*, **17**(1), 21–45.

Bates, J. (1989). Concepts and measures of temperament. Temperament in childhood. In G. A. Kohnstamm, J. Bates, & M. Klevjord (Eds.), *Temperament in childhood* (pp. 3–26). Oxford, UK: John Wiley & Sons.

Beckwith, J., & Morris, C. (2008). Twin studies of political behavior: Untenable assumptions? *Perspectives on Politics*, **6**, 785–792.

Bennett, W. L., & Iyengar, S. (2008). A new era of minimal effects? The changing foundations of political communication. *Journal of Communication*, **58**, 707–731.

Boomsma, D., Busjahn, A., & Peltonen, L. (2002). Classical twin studies and beyond. *Nature Reviews Genetics*, **3**, 872–882.

Boomsma, D. I., de Geus, E. J. C., van Baal, G. C. M., & Koopmans, J. R. (1999). A religious upbringing reduces the influence of genetic factors on disinhibition: Evidence for interaction between genotype and environment on personality. *Twin Research*, **2**, 115–125.

Blumler, J. G. (1985). The social character of media gratifications. In K. E. Rosengren, L. A. Wenner, & P. Palmgreen (Eds.), *Media gratifications research: Current perspectives* (pp. 41–59). Beverly Hills, CA: Sage.

Bouchard, T. (2004). Genetic influence on human psychological traits: A survey. *Current Directions in Psychological Science*, **13**, 148–151.

Bouchard, T., & McGue, M. (2003). Genetic and environmental influences on human psychological differences. *Journal of Neurobiology*, **54**(1), 4–45.

Brady, H., Schlozman, K., & Verba, S. (1999). Prospecting for participants: A rational expectations and the recruitment of political activists. *American Political Science Review*, **93**, 153–168.

Brody, N. (1994). Heritability of traits. *Psychology Inquiry*, **5**, 117–119.

Bryant, J., & Oliver, M. B. (Eds.) (2008). *Media effects: Advances in theory and research* (3rd ed.). New York, NY: Routledge.

Cantril, H. (1942). Professor quiz: A gratifications study. In P. F. Lazarsfeld & F. Stanton (Eds.), *Radio research 1941* (pp. 34–45). New York, NY: Duell, Sloan & Pearce.

Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig, I., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT. *Gene Science*, **18**(July), 386–389.

Correa, T., Hinsley, A. W., & de Zuniga, H. G. (2010). Who interacts on the web?: The intersection of users' personality and social media use. *Computers in Human Behavior*, **26**, 247–253.

DeFleur, M. (1998). Where have all the milestones gone? The decline of significant research on the process and effects of mass communication. *Mass Communication and Society*, **1**(1/2), 85–98.

Delli Carpini, M. X., & Keeter, S. (1996). *What Americans know about politics and why it matters.* New Haven, CT: Yale University.

Donohew, L., Palmgreen, P., & Rayburn, J. D. (1987). Social and psychological origins of media use: A lifestyle analysis. *Journal of Broadcasting and Electronic Media*, **31**, 255–278.

Eaves, L. J., Eyseneck, H. J., & Martin, N. G. (1989). *Genes, culture, and personality: An empirical approach.* San Diego, CA: Academic Press.

Eaves, L. J., Silberg, J. L., & Maes, H. H. (2005). Revisiting the children of twins: Can they be used to resolve the environmental effects of dyadic parental treatment on child behavior? *Twin Research and Human Genetics*, **8**, 283–290.

Ehrenberg, A., Juckes, S., White, K. M., & Walsh, S. P. (2008). Personality and self-esteem as predictors of young people's technology use. *CyberPyschology and Behavior*, **11**, 739–741.

Falconer, D. S. (1960). *Introduction to quantitative genetics* (1st ed.). Edinburgh, UK: Oliver & Boyd.

Flaherty, L. M., Pearce, K. J., & Rubin, R. (1998). Internet and face-to-face communication: Not functional alternatives. *Communication Quarterly*, **46**, 250–268.

Fowler, J., Baker, L., & Dawes, C. (2008). Genetic variation in political participation. *American Political Science Review*, **102**, 233–248.

Fowler, J., & Dawes, C. (2008). Two genes predict voter turnout. *Journal of Politics*, **70**, 579–594.

Fowler, J. H., Dawes, C. T., & Christakis, N. A. (2009). Model of genetic variation in human social networks. *Proceedings of the National Academy of Sciences*, **106**, 1720–1724.

Freese, J. (2008). Genetics and the social science explanation of individual outcomes. *American Journal of Sociology*, **114**, S1–S35.

Freese, J., & Shostak, S. (2009). Genetics and social inquiry. *Annual Review of Sociology*, **35**, 107–128.

Fuller, J. L., & Thompson, W. R. (1978). *Foundations of behavior genetics.* St. Louis, MO: Mosby.

Funk, C. L., Smith, K. B., Alford, J., Hibbing, M. V., Hatemi, P. K., & Hibbing, J. R. (2010). *Toward a modern view of political man: Genetic and environmental sources of political orientations and participation*. Paper presented at the APSA 2010 Annual Meeting, Washington, DC.

Gerber, A., Huber, G., Doherty, D., & Dowling, C. (2010). *Personality traits and the consumption of political information*. Unpublished manuscript.

Gerson, W. (1966). Mass media socialization behavior: Negro-whites differences. *Social Forces*, **45**, 40–50.

Hall, J. G. (2003). Twinning. *Lancet*, **362**, 735–743.

Hamburger, Y. A., & Ben-Artzi, E. (2000). The relationship between extraversion and neuroticism and the different uses of the Internet. *Computers in Human Behavior*, **16**, 441–449.

Harden P. K., Turkheimer, E., & Loehlin, J. C. (2006). Genotype by environment interaction in adolescents' cognitive aptitude. *Behavior Genetics*, **37**, 1573–3297.

Harris, K. M. (2008). *The National Longitudinal Study of Adolescent Health (Add Health), Waves I & II, 1994–1996; Wave III, 2001–2002* [machine-readable data file and documentation]. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill.

Hatemi, P., Alford, J., Hibbing, J., Martin, N., & Eaves, L. (2008). Is there a "party" in your genes? *Political Research Quarterly*. doi: 10.1177/1065912908327606.

Hatemi, P. K., Gillespie, N., Eaves, L. J., Maher, B., Webb, B., Heath, A. C., et al. (2011). Genome-wide analysis of political attitudes. *Journal of Politics*, **73**(1), 1–15.

Heath, A. C., Berg, K., Eaves, L. J., Solass, M., Berg, L. J., Eaves, M. H., et al. (1985). Education policy and the heritability of educational attainment. *Nature*, **314**, 734–736.

Hetherington, M. (1998). The political relevance of political trust. *American Political Science Review*, **92**, 791–808.

Hibbing, M. V., Ritchie, M., & Anderson, M. R. (2010, November 5). Personality and political discussion. *Political Behavior* [Epub ahead of print].

Hibbing, J. R., & Smith, K. B. (2011). *Biology, ideology and epistemology: How do we know political attitudes are inherited and why should we care?* Unpublished manuscript, University of Nebraska-Lincoln, Lincoln, NE.

Horwitz, A., Videon, T., Schmitz, M., & Davis, D. (2003). Rethinking twins and environments: Possible social sources for assumed genetics influences in twin research. *Journal of Health and Social Behavior*, **44**, 111–129.

Kaplan, D. (2000). *Structural equation modeling: Foundations and extensions.* Thousand Oaks, CA: Sage.

Katz, E., Blumler, J. G., & Gurevitch, M. (1974). Utilization of mass communication by the individual. In J. G. Blumler, & E. Katz (Eds.), *The uses of mass communications: Current perspectives on gratifications research* (pp. 19–32). Beverly Hills, CA: Sage Publishing.

Katz, E., & Foulkes, D. (1962). On the use of mass media as escape: Clarification of a concept. *Public Opinion Quarterly*, **26**, 377–388.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A test of the equal- environment assumption in twin studies of psychiatric illness. *Behavior Genetics*, **23**(1), 21–27.

LaRose, R. (2010). The problem of media habits. *Communication Theory*, **20**, 194–222.

LaRose, R., & Atkin, D. (1991). Cable access: Market concerns amidst the marketplace of ideas. *Journalism Quarterly*, **68**, 354–362.

LaRose, R., & Eastin, M. (2004). A social cognitive theory of Internet uses and gratifications: Toward a new model of media attendance. *Journal of Broadcasting and Electronic Media*, **48**, 358–377.

Levine, T., Weber, R., Hullett, C., Park, H. S., & Massi Lindsey, L. (2008). A critical assessment of null hypothesis significance testing in quantitative communication research. *Human Communication Research*, **34**, 171–187.

Levine, T., Weber, R., Park, H. S., & Hullett, C. (2008). A communication researchers' guide to null hypothesis significance testing and alternatives. *Human Communication Research*, **34**, 188–209.

Lin, C. (1993). Exploring the role of VCR use in the emerging home entertainment culture. *Journalism Quarterly*, **70**, 833–842.

Loehlin, J. C. (1992). *Genes and environment in personality development.* Newbury Park, CA: Sage.

Loehlin, J. C., & Nichols, R. C. (1976). *Heredity, environment, and personality: A study of 850 sets of twins.* Austin, TX: University of Texas Press.

Loewen, P. J., & Dawes, C. T. (2010). Heritability of duty and voter turnout. *Political Psychology*.

Lowery, S., & DeFleur, M. (1995). *Milestones in mass communication research* (3rd ed.). White Plains, NY: Longman.

Lull, J. (1995). *Media, communication, culture: A global approach.* New York: Columbia University Press.
Luskin, R. C. (1990). Explaining political sophistication. *Political Behavior*, **12**, 331–336.
Martin, N. G., & Eaves, L. J. (1977). The genetical analysis of covariance structure. *Heredity*, **38**, 79–95.
McDermott, R., Tingley, D., Cowden, J., Frazzetto, G., & Johnson, D. D. P. (2009). Monoamine oxidase A gene (MAOA) predicts behavioral aggression following provocation. *Proceedings of the National Academy of Sciences*, **106**, 2118–2123.
McQuail, D. (1994). The rise of media of mass communication. In D. McQuail (Ed.), *Mass communication theory: An introduction* (pp. 1–29). London: Sage.
Medland, S., & Hatemi, P. (2009). Political science, behavior genetics and twin studies: A methodological primer. *Political Analysis*, **17**, 191–214.
Mendelsohn, H. (1964). Listening to the radio. In L. A. Dexter, & D. M. White (Eds.), *People, society and mass communication* (pp. 239–248). New York: Free Press.
Mondak, J. J., & Halperin, K. D. (2008). A framework for the study of personality and political behavior. *British Journal of Political Science*, **38**, 335–362.
Mondak, J. J., Hibbing, M. V., Canache, D., Seligson, M. A., & Anderson, M. R. (2010). Personality and civic engagement: An integrative framework for the study of trait effects on political behavior. *American Political Science Review*, **104**, 85–110.
Muthén, L. K., & Muthén, B. O. (1998–2007). *Mplus user's guide* (5th ed.). Los Angeles: Muthén & Muthén.
Neale, M. (2009). Biometrical models in behavioral genetics. In Y.-K. Kim (Ed.), *Handbook of behavior genetics.* New York: Springer.
Neale, M., & Cardon, L. (1992). *Methodology for genetics studies of twins and families.* Boston: Kluwer Academic.
Neale, M. C., & Maes, H. M. (2004). *Methodology for genetic studies of twins and families.* Dordrecht, NL: Kluwer Academic.
Oellette, J., & Wood, W. (1998). Habit and intention in everyday life: The multiple processes by which past behavior predicts future behavior. *Psychological Bulletin*, **124**(1), 54–74.
Papacharissi, Z., & Rubin, A. (2000). Predictors of internet use. *Journal of Broadcasting Electronic Media*, **44**, 175–196.
Plomin, R., Corley, R., DeFries, J. C., & Fulker, D. W. (1990). Individual differences in television viewing in early childhood: Nature and well as nurture. *Psychological Science*, **1**, 371–377.
Plomin, R., DeFries, J. C., McClearn, G., & McGuffin, P. (2001). *Behavior genetics.* New York: Worth Publishers.
Posthuma, D. (2009). Multivariate genetic analysis. In Y.-K. Kim (Ed.), *Handbook of behavior genetics*. New York: Springer.
Prior, M. (2007). *Post-broadcast democracy: How media choice increases inequality in political involvement and polarizes elections.* Cambridge, NY: Cambridge University Press.
Prior, M. (2009). Improving media effects research through better measurement of news exposure. *Journal of Politics*, **71**, 893–908.
Prior, M. (2010). You've either got it or you don't? The stability of political interest over the life cycle. *Journal of Politics*, **72**, 747–766.
Ridley, M. (2003). *Nature via nurture.* New York: Harper Collins.

Ronalds, G. A., De Stavola, B. L., & Leon, D. A. (2005). The cognitive cost of being a twin: Evidence from comparisons within families in the Aberdeen children of the 1950s cohort study. *British Medical Journal*, **331**, 1306.

Rosengren, K. E. (1974). Uses and gratifications: A paradigm outline. In J. G. Blumler, & E. Katz (Eds.), *The use of mass communications: Current perspectives of gratifications research* (269–286). Beverly Hills, CA: Sage.

Rosengren, K. E., Wenner, L., & Palmgren, P. (Eds.). (1985). *Media gratifications research: Current perspectives.* Beverly Hills, CA: Sage.

Rosenstein, A. W., & Grant, A. E. (1997). Reconceptualizing the role of habit: A new model of television audience. *Journal of Broadcasting & Electronic Media*, **41**, 324–344.

Ross, C., Orr, E. S., Sisic, M., Arseneault, J. M., Simmering, M. G., & Orr, R. R. (2009). Personality and motivations associated with Facebook use. *Computers in Human Behavior*, **25**, 578–586.

Rubin, A. (1994). Media uses and effects: A uses and gratifications perspective. In J. Bryant, & D. Zillmann (Eds.), *Media effects: Advances in theory and research.* Hillsdale, NJ: Laurence Erlbaum.

Rubin, R., Perse, E., & Barbato, C. (1988). Conceptualization and measurement of interpersonal communication motives. *Human Communication Research*, **14**, 602–628.

Rubin, A., & Rubin, R. (1989). Social and psychological antecedents of VCR use. In M. R. Levy (Ed.), *The VCR age: Home video and mass communication* (pp. 92–111). Newbury Park, CA: Sage.

Ruggiero, T. (2000). Uses and gratifications in the 21st century. *Mass Communication and Society*, **3**(1), 3–37.

Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development*, **63**, 1–19.

Schuirmann, D. J. (1987). A comparison of the two one-sided tests procedure and the power approach for assessing equivalence of average bioavailability. *Journal of Pharmacokinetics and Biopharmaceutics*, **15**, 657–680.

Shanahan, M. J., & Hofer, S. M. (2005). Social context in gene-environment interactions: Retrospect and prospect. *Journals of Gerontology: Series B*, **60B**(Special Issue I), 65–76.

Sherry, J. (2001). Toward an etiology of media use motivations: The role of temperament in media use. *Communication Monographs*, **68**, 274–288.

Sherry, J. (2004). Media effects theory and the nature/nurture debate: A historical overview and directions for future research. *Media Psychology*, **6**, 83–109.

Snieder, H., Wang, X., & MacGregor, A. (2010). Twin methodology. In H. Kehrer-Sawatzki (Ed.), *Encyclopedia of life sciences: Evolution and diversity of life.* Chichester, UK: John Wiley Sons, Ltd.

Tryon, W. W. (2001). Evaluating statistical difference, equivalence, and indeterminacy using inferential confidence intervals: An integrated alternative method of conducting null hypothesis statistical tests. *Psychological Methods*, **6**, 371–386.

Tryon, W. W., & Lewis, C. (2008). An inferential confidence interval method of establishing statistical equivalence that corrects Tryon's (2001) reduction factor. *Psychological Methods*, **13**, 272–277.

Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B. M., & Gottesman, I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, **14**, 623–628.

Vink, J. M., Willemsen, G., & Boomsma, D. I. (2005). Heritability of smoking initiation and nicotine dependence. *Behavior Genetics*, **35**, 397–406.

Weaver, D. H. (1996, July). What voters learn from the media. *Annual of the AAPSS*, 34–47.

Weaver, J. B. (2000). Personality and entertainment preferences. In D. Zillman, & P. Vorderer (Eds.), *Media entertainment: The psychology of its appeal.* Mahwah, NJ: Erlbaum.

Weaver, J. B. (2003). Individual differences in television viewing motives. *Personality and individual differences*, **35**, 1427–1437.

Weber, R., Sherry, J., & Mathiak, K. (2008). The neurophysiological perspective in mass communication research. Theoretical rationale, methods, and applications. In M. J. Beatty, J. C. McCroskey, & K. Floyd (Eds.), *Biological dimensions of communication: Perspectives, methods, and research.* (pp. 41–71). Cresskill, NJ: Hampton Press.

Wimmer, R., & Dominick, J. (1994). *Mass media research: An introduction.* Belmont, CA: Wadsworth Publishing.

Zaller, J. (1996). The myth of massive media impact revived: New support for a discredited idea. In D. Mutz, R. Brody, & P. Sniderman (Eds.), *Political persuasion and attitude change.* (pp. 17–79). Ann Arbor, MI: University of Michigan Press.

Zhang, X., Gainetdinov, P., Beaulieu, J. M., Sotnikova, T., Burch, L., Williams, R., et al. (2005). Loss of function mutation in tryptophan hydroxlylase-2 identified in unipolar major depression. *Neuron*, **6**(January), 11–16.

Zywica, J., & Danowski, J. (2008). The faces of facebookers: Investigating social enhancement and social compensation hypotheses; predicting Facebook and offline popularity from sociability and self-esteem, and mapping the meanings of popularity with semantic networks. *Journal of Computer-Mediated Communication*, **14**(1), 1–34.

基因和环境对媒介使用和传播行为的影响

【摘要：】

许多学术研究已经探索了媒体消费的动机和其他各种传播特征。然而，很少有人研究这些动机的来源，几乎没有任何研究考虑其潜在的基因基础。本文应用行为遗传学领域的研究，通过“经典双子设计”（CTD），探讨遗传和环境对九个传播行为的影响。研究结果表明，媒体习惯中总体变量中的相当一部分, 在某些情况下甚至高达总体变量三分之一的变量，可由基因所解释。在当代社会，更进一步地关注遗传基因将有助于大众传播学者思考媒体特征的原因和结果。

Les facteurs génétiques et environnementaux qui influencent l'usage des médias et les comportements de communication

Beaucoup de recherches ont exploré les motivations sous-tendant la consommation médiatique et plusieurs autres traits de communication. Cependant, peu d'études ont cherché à connaître les sources de ces motivations, et pratiquement aucune recherche ne considère leurs possibles fondements génétiques. Puisant dans la recherche en génétique des comportements, nous utilisons un modèle classique d'étude de jumeaux pour examiner les influences génétiques et environnementales sur neuf comportements de communication. Nos résultats indiquent qu'une part importante de la variance totale des habitudes de comportements médiatiques peut être attribuée aux gènes, dans une proportion aussi élevée qu'un tiers de la variance, dans certains cas. Les chercheurs en communication de masse tireraient avantage à porter une plus grande attention à l'héritabilité lorsqu'ils considèrent les causes et les conséquences des traits médiatiques dans la société contemporaine.

Mots clés : usage des médias, traits de communication, génétique des comportements, modèle classique d'étude de jumeaux

Genetische und umweltbezogene Einflüsse auf Mediennutzung und Kommunikationsverhalten

Eine große Zahl wissenschaftlicher Arbeiten hat die Motive für die Nutzung von Medien und andere Kommunikationseigenschaften untersucht. Nur wenige Arbeiten haben sich mit den Ursachen dieser Motive befasst und praktisch keine Forschung existiert zu einer möglichen genetischen Veranlagung. Wir orientieren uns an verhaltensgenetischer Forschung und nutzen ein klassisches Zwillingsforschungsdesign, um die genetischen und umweltbezogenen Einflüsse auf neun Kommunikationsverhaltensweisen zu untersuchen. Unsere Ergebnisse zeigen, dass ein beträchtlicher Teil der Gesamtvarianz von Mediengewohnheiten den Genen zugeschrieben werden kann – zum Teil bis zu einem Drittel der Varianz. Wissenschaftler im Bereich der Massenkommunikationsforschung würden davon profitieren, diese erbliche Komponente stärker zu berücksichtigen, wenn sie über die Gründe und auch die Konsequenzen von Medieneigenschaften in der heutigen Gesellschaft nachdenken.

Schlüsselbegriffe: Mediennutzung, Kommunikationseigenschaften, Verhaltensgenetik, Klassisches Zwillingsdesign

미디어 사용과 커뮤니케이션 행위들에 대한 유전적 그리고 환경적 영향력들에 관한 연구

기존의 많은 학문적 연구들이 미디어 소비와 여러 커뮤니케이션 특징들을 설명하는 동기들을 연구해 왔다. 그러나, 이들 동기들의 출처를 연구한 논문은 별로 없었으며, 그들의 잠재적인 유전적 토대를 설명하는 논문은 사실상 없었다. 행태 유전학의 영역에 기초하여, 우리는 CTD 연구기법을 사용하며 9 개의 커뮤니케이션 행태들에 대한 유전적 그리고 환경적 영향력들을 연구하였다. 우리의 발견들은 미디어 행태에서의 전체변수의 상당한 정도가 유전적 요소에 기인하며, 약 3 분의 1 정도는 다른 상황을 설명한다는 것을 보여주고 있다. 따라서, 본 논문은 매스 커뮤니케이션 학자들이 현사회에서 미디어 특성들에 대한 원인과 결과들을 고려할때 유전적 요소를 보다 밀접하게 고려하는것에 의해 큰 도움을 받을수 있을 것으로 보고 있다.

Las Influencias Genéticas y del Medio Ambiente sobre el Uso de los Medios y los Comportamientos Comunicacionales

Un gran número de trabajos de eruditos ha explorado las motivaciones detrás del consumo de los medios y otras varios rasgos de la comunicación. Sin embargo, poca investigación ha indagado los recursos de las motivaciones y virtualmente ninguna investigación considera los fundamentos genéticos potenciales. Basados en el campo de la genética del comportamiento, usamos un Estudio de Diseño Clásico Gemelo (CTD) para examinar las influencias genéticas y del medio ambiente sobre nueve comportamientos comunicativos. Nuestros hallazgos indican que una porción substancial del total de la varianza en los hábitos de los medios puede ser atribuido a los genes, tanto como un tercio de la varianza en algunas instancias. Los estudiosos de los medios de comunicación se beneficiarían en prestar atención a la herencia cuando piensan en las causas, así como también las consecuencias de los rasgos de los medios en la sociedad contemporánea.

Palabras Claves: uso de los medios, rasgos de comunicación, genéticas del comportamiento, diseño clásico gemelo