Published in final edited form as:

Behav Genet. 2019 March ; 49(2): 211–220. doi:10.1007/s10519-018-9934-y.

The Role of Emergence in Genetically Informed Relationships Research: A Methodological Analysis

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Abstract

This paper provides a critical analysis of genetically informed research on relationships, with an emphasis on relationships among unrelated individuals (e.g., spouses). To date, research in this area has used traditional behavioral genetic frameworks to either partition the variance in relationship-related outcomes into genetic and environmental components, or to examine gene-environment interplay between relationship factors and other outcomes. However, this conventional approach is at odds with the long-standing understanding from the field of relationship science that both partners' characteristics matter when predicting shared outcomes—that is, outcomes that are emergent. We examine briefly the philosophical concept of emergence, and discuss ways to model dyadic outcomes in genetically informed relationships research. We also review the related topic of social genetic effects, which refer to the influence of a social partner's genotype on a proband's phenotype. A genetically informed dyadic perspective has potentially important consequences for our understanding of the pathways from genotype→shared or individual-level phenotypes, and more fully recognizes the complexity of how genetic and social/environmental factors come together to influence human behavior.

The Role of Emergence in Genetically Informative Relationships Research Consider an interaction between two spouses who have been together for over a decade. The day before, Tim offered to drive Alice to the airport for her 7:45 flight, not realizing that she had a morning, not evening, departure. He recognizes the miscommunication when Alice shakes him awake, saying that they need to get on the road for the 40-minute drive to the airport. Alice is annoyed that Tim is not ready to go, and Tim is furious with Alice for not being

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Compliance with Ethical Standards

Disclosure of potential conflicts of interest. The authors declare no conflicts of interest.

Research involving human participants. Not applicable.

Informed consent. Not applicable.

clear about the time of her flight, as he will now be late for a staff meeting on the opposite side of town. Each is convinced that the situation is the other person's fault, but knows that saying so will only worsen the tension. Despite being otherwise well-adjusted and happy individuals, Tim and Alice sit in angry silence on the way to the airport.

Tim and Alice's silent car ride is the complex product of their interactions in various situations across the years. This web of actions, thoughts, and emotions reminds us that the whole is often greater than the sum of its parts when studying relationships. And although this principle is readily intuited by most, it is largely absent from genetically informed relationships research. The purpose of this article is to reflect on the state of the science for genetically informed relationships research, and to outline future directions for research in this area. To this end, we: *i*) review the conventional methods used in this area; *ii*) critically evaluate whether conventional models are appropriate for relationships research given the emergent nature of relationship phenotypes; and *iii*) advance the argument that a dyadic perspective is needed to improve genetically informed relationships research. The argument put forth here—namely, the need to incorporate a dyadic perspective that considers the characteristics of both partners—is intended to be applicable to a range of close relationships (e.g., parent-child, peer, and romantic); however, in the interest of providing a coherent set of examples, we primarily draw from the area of romantic relationships.

Genetically Informed Research on Relationships: The Field Today

The goal of this section is to briefly review the types of analytic models that are typically used in genetically informed relationships research, rather than to provide an exhaustive review of specific study results (readers interested in a review of findings are referred to Whisman and South 2016). The majority of genetically informed research on relationships is conducted in twin samples, where the patterns of resemblance for monozygotic and dizygotic twins are used to make inferences about the degree to which genetic and environmental factors influence outcomes. Figure 1 illustrates this model for a phenotype, *T*. Phenotypic variance in *T* is partitioned into additive genetic (*A*), shared environmental (*C*), and nonshared environmental (*E*) components. Monozygotic (MZ) twins share 100% of their genetic variation, while dizygotic (DZ) twins share half of their genetic variation on average. Accordingly, the genetic correlations (r_A) between twins in monozygotic and dizygotic pairs are 1.0 and 0.5, respectively. The shared environmental correlation (r_C), which reflects the environmental factors that increase similarity between siblings reared together, is 1.0 for both MZ and DZ twins. The non-shared environmental factors, which reflects environmental exposures that make siblings different from one another, are uncorrelated.

As with most behavioral outcomes, these studies show modest to moderate genetic influences on relationship measures including marriage (Jerskey et al. 2010; Johnson et al. 2004), divorce (McGue and Lykken 1992; Salvatore et al. 2017), as well as aspects of self-reported relationship functioning including satisfaction, conflict, strain and support (Spotts et al. 2004; Spotts et al. 2006; Jarnecke and South 2014; Kuschke et al. 2018). There is one exception to the general finding that relationship outcomes are heritable, which is that there are minimal genetic influences on observer-rated measures of relationship functioning (Spotts et al. 2005). One possible explanation for this discrepancy is that genetic influences

on relationship processes may operate through perceptions of relationship functioning rather than objectively measured indicators of interpersonal behavior.

Studies of twins and their spouses have also demonstrated that one spouse's heritable characteristics account for variation in the other spouse's relationship perceptions. In analyses of female twins and their spouses, 14–23% of the variation in husbands' perception of relationship functioning was attributable to genetic differences between their wives (Spotts et al. 2004). Although such findings may seem counterintuitive, they illustrate the idea that genes influence phenotypes beyond the body as part of what Dawkins (1982) termed “the extended phenotype”. We return to this idea shortly as part of a discussion on social genetic effects. These spousal genetic effects are suggestive of evocative gene-environment correlation (Scarr and McCartney 1983), whereby an individual's genetically influenced traits or behaviors may elicit particular responses from others in their social environment. Subsequent analyses were consistent with this interpretation, and demonstrated that genetic influences on wives' personality characteristics overlapped, to modest degree, with wives' genetic influences on husbands' marital satisfaction (Spotts et al. 2005).

Above and beyond partitioning the variance in relationship outcomes into genetic and environmental components, twin methods are also often used to examine the associations between relationship factors and other behavioral, emotional, and health outcomes (Whisman and South 2016). One common question is the nature of the associations between relationship factors and these other outcomes. Studies along these lines show that relationship factors share (in part) genetic influences with internalizing and externalizing problems like depressive and anxiety symptoms (Spotts et al. 2004; Whisman et al. 2018), alcohol use disorder (Salvatore et al. 2017; Salvatore et al. 2015), and self-rated physical health (South and Krueger 2013). Some have expanded these models to include information from twins' spouses to characterize the nature of associations between spouse characteristics and mental health outcomes. Findings from these studies indicate that non-shared environmental influences explained the covariation between husbands' reports of marital quality and female twins' reports of positive mental health and depressive symptoms (Spotts et al. 2004; Spotts et al. 2005). Parallel effects were observed in a study of marital quality and depressive symptoms, whereby non-shared environmental influences primarily accounted for the association between low levels of spouse-reported dyadic adjustment and high levels of twins' depressive symptoms (Whisman et al. 2018). Significant covariation between spouse characteristics and one's own mental health that is attributable to unique environmental influences could be consistent with the causal hypothesis that spousal characteristics and cause poorer mental health (or the reverse).

An important consideration when interpreting findings from twin studies that incorporate data from spouses is that the modeling approaches assume random mating for the focal variables. Violations of this assumption can bias results. For example, when there is assortative mating for a genetically-influenced trait or behavior, whereby partners select others similar to them, this can result in a genetic correlation between partners. The consequence of this for the models described above is that MZ twins will have spouses who are more alike one another than the spouses of DZ twins, inflating the heritability estimates for spousal reports. There is consistent evidence of assortative mating across a wide variety

of traits and behaviors. Spousal concordance is moderate for physical characteristics (e.g., height, eye color) and psychiatric disorders (Nordsletten et al. 2016; Vandenberg 1971), and higher for traditional values (e.g., church attendance) and indicators of cognitive ability (e.g., IQ and education) (Lykken and Tellegen 1993; Watson et al. 2004). Spousal concordance for personality traits tend to be more modest (Vandenberg 1971). Attenuating concerns about bias due to assortative mating is evidence that the spouses of MZ and DZ twins do not resemble one another more than random pairs of individuals on many psychological variables (Lykken and Tellegen 1993).

A second question is whether relationship factors moderate genetic influences on these outcomes in what is commonly known as gene-environment interaction. In twin studies, latent gene-environment interaction is inferred by comparing MZ and DZ twin pair correlations across different levels of the environment. When the magnitude of additive genetic effects changes across levels of the environment, this suggests gene-environment interaction. In an early example from the relationships literature, Heath et al. (1989) found that genetic influences on alcohol outcomes were attenuated in women who were married or in a marriage-like relationship. In contrast, genetic influences were more pronounced among women who were not in marriage-like relationships. A number of other studies using these methods find that relationship factors like support from one's spouse moderate genetic influences on outcomes including depression (South and Krueger 2008), physical health (South and Krueger 2013; Finkel et al. 2016), and alcohol problems (Jarnecke and South 2014).

Although the preponderance of genetically informed relationships research comes from twin studies, it is worth noting that other methods have been employed, including children-of-twin, co-relative, and adoption designs (Salvatore et al. 2018; D'Onofrio et al. 2007; Kendler et al. 2017; Kendler et al. 2016). As with twin studies, these methods use patterns of resemblance among relatives of known genetic relatedness and environmental sharing to make inferences about genetic influences on relationship outcomes, and the nature of the associations between relationship factors and other behavioral and emotional health outcomes. There are fewer studies of relationships using measured genotypes. Those that exist (e.g., Maher et al. 2011; Walum et al. 2012; Schoebi et al. 2012) have used a candidate gene approach to examine whether individual differences in a single gene/genetic variant of purported biological relevance are associated with relationship outcomes. However, these studies should be interpreted with caution given the controversy surrounding candidate gene research (Duncan and Keller 2011), with Type I error and the selection of the candidate gene/variant being of particular concern.

Are conventional behavior genetic models appropriate for emergent outcomes in relationships research?

As detailed above, the majority of genetically informative studies on relationships take an individual-focused perspective to map individual differences in inferred or measured genotype onto relationship outcomes or to examine genetic correlation or gene-environment interaction. This provides a useful starting point for genetically informed relationships

research; however, this approach is incomplete when considering interpersonal processes and outcomes that are emergent, and arise only through the interactions of partners and their characteristics (Sroufe et al. 2005).

The concept of emergence has a long history in philosophy (O'Connor and Wong 2015; Kim 1999). Although there are many definitions of emergence, most typically include two components (O'Connor and Wong 2015). The first is that a higher-level system demonstrates novel properties that go beyond those of the lower-level constituent systems or components. The second is that the higher-level system behaves in ways that are not predictable (in a linear, additive sense) based on the lower-level systems. Common examples of emergence from the natural world include flocking birds or ant colonies. These illustrate emergence in that the higher-level system of the flock or the colony exhibits novel, unpredictable properties that transcend the properties of individual birds or ants. Thus, an emergentist perspective sits somewhere between reductionism (which posits that complex systems can only be understood in terms of their simpler component parts) and holism (which posits that complex systems can only be understood in their entirety).

There are a few key concepts and distinctions in the philosophical literature that are particularly relevant for our discussion of the role of emergence in genetically informed relationships research. First is the concept of novelty, and the related distinction between emergent and resultant phenomena. In an emergentist perspective, *novelty* is not limited to the phenomenon being unexpected or surprising. Rather, novelty more broadly refers to the idea that emergent phenomena are not simply an additive combination of the lower-level systems that are involved. This is in contrast to a *resultant property*, which is an additive function of the lower-level systems or components. Water is often used as an example of this distinction between emergent and resultant properties (Hempel and Oppenheim 2008). Some physical characteristics of water, such as its being a liquid at room temperature, can be considered emergent given that this feature would not have been predictable from knowing about hydrogen and oxygen. However, other characteristics of liquid water, such as its weight, are considered resultant because weight can be predicted as an additive function of hydrogen and oxygen atoms.

Second is the distinction between ontological and epistemological emergence. Ontological emergence, also referred to as *supervenience emergentism* or *strong emergence* posits that lower-level systems undergird the higher-level systems, but that the higher-level systems cannot be reduced to these lower-level systems. That is, something new, in an ontological sense, arises from the interaction of the lower-level components. For example, the psychological process of feeling happy supervenes on neural processes, but cannot be reduced to neural processes. Likewise, the meaning of the word “dog” is not reducible to the sound waves that constitute it but rather depends on the emergence of human language and symbolic processes.

In contrast, *epistemological emergentism* or *weak emergence* defines emergence with respect to the limits of human knowledge and understanding of complex systems. Thus, according to this perspective, emergent phenomena are not fundamentally “new”, but rather cannot be easily predicted from even a thorough understanding of the constituent

components. Instead, there must be laws or theories linking the higher- and lower-level systems. To use an example from Sawyer (2002), no single molecule gives a volume of gas its emergent property of pressure. Yet, statistical mechanics makes it possible to reduce pressure to lawful statements about how molecules move and interact.

The concept of emergence plays an influential role in psychological theories and concepts of social behavior, such as group dynamics (Lewin 1947), attachment theory (Bowlby 1969/1982), and interdependence theory (Thibaut and Kelley 1959). Although these theories explain different social phenomena, they converge in suggesting that dyadic outcomes are best understood as a function of both individuals' characteristics. That is, these social outcomes are not predetermined by any one partner. Rather, something novel and unpredictable (in a linear sense) emerges. Thus, these theories are consistent with an emergentist perspective in that they suggest that both partners' characteristics contribute to shared outcomes. These theories are examples of weak emergence, given that their primary goal is to explain and predict how the characteristics of the lower-level components (i.e., individuals) predict these novel, emergent higher-level systems (i.e., dyadic relationship outcomes).

There are numerous examples of emergent outcomes in romantic relationships. These range from relationship formation and dissolution (Sprecher et al. 2006); the strategies that couples adopt to resolve conflict (e.g., mutual avoidance versus conflict engagement) (Christensen and Shenk 1991); decisions about how a shared resource (e.g., money, leisure time) are allocated (Smith et al. 2017); and decisions about condom use (Karney et al. 2010). These outcomes illustrate weakly emergent phenomena in that they depend on the contributions of both partners, and would not be predictable from one partner's characteristics alone. (A note about relationship dissolution: we recognize that one partner's actions, such as filing divorce papers, can dissolve a relationship. However, it is commonly recognized in relationship science that relationship dissolution is a process that typically involves both partners, rather than a discrete, unilateral event (Amato 2010)). This matters for genetically informed relationships research because it suggests that we are missing something when we simply map one individual's genetic contribution onto outcomes that are co-constructed or influenced by both partners.

We illustrate this in Figure 2, which is a simple expansion of Figure 1 to include twins' spouses. In this figure, a dyadic outcome, T, depends on the variance components of both the twin proband, P, and their unrelated spouse, S. (For simplicity, we assume random mating). In this model, it becomes clear that there are two sources of genetic influence on a dyadic outcome, like relationship dissolution, reflecting the contributions of both the twin and their spouse. The addition of the spousal component in Figure 2 opens a series of new questions for our understanding of the genetic contributions to shared outcomes, including whether the relative contributions of A, C, and E for each partner can be assumed to be equivalent, and whether partners' A, C, and E components have additive or interactive effects. Just as preformationist ideas about "genes for" complex behavioral outcomes are likely to be inappropriate (Kendler 2005), this simple illustration cautions against considering the contributions of only one partner's genotype to emergent phenotypes. We recognize that this simple additive model does not capture the full dynamic complexity of dyadic, emergent

outcomes. However, we believe that this model is useful for beginning to think about how to incorporate a dyadic perspective in genetically informed research.

What Tools are Available to Study Emergent, Dyadic Phenotypes in Genetically Informed Relationships Research?

The study of emergence is not new to behavioral science. For example, the work of Gottlieb (1998) and others (Smith and Thelen 2003; Vallacher et al. 2015; Lewis 2000; Kelso 1995; Gottman et al. 2002) has long recognized the probabilistic nature of how people think, feel, and behave. Much of this work relies on dynamic systems modeling approaches to describe how an organism or system changes over time. This typically involves state-space modeling of intensive repeated measures data, such as using partners' verbal and non-verbal behavior during an interaction task to classify the couple as distressed or non-distressed (Gottman et al. 1977). Although we recognize that dynamic systems modeling is the gold standard for modeling emergent outcomes in the behavioral sciences, in our view these types of statistical tools would be impractical to implement (at present) in the context of genetically informed relationships research given the difficulty of collecting intensive repeated measures data in the sample sizes required. For this reason, although the genetically-informed methods that we highlight below consider both partners' contributions to shared, dyadic outcomes, they rely on additive and interactive models that are currently empirically tractable within the context of genetically informed research. In short, we focus on simpler additive and interactive models in the spirit of beginning to incorporate a dyadic perspective in genetically informed research, while acknowledging that these models do not fully capture emergent processes. We use the example of divorce given that this shared outcome has received attention in the behavior genetics literature in the past (Jerskey et al. 2010; McGue and Lykken 1992; Salvatore et al. 2018; D'Onofrio et al. 2007).

Characterizing the contributions of each partner's genome to a shared phenotype using an extension of genome-wide complex trait analysis.

Genome-wide complex trait analysis (GCTA; Yang et al. 2011) was developed as a method to use molecular genetic data to estimate the degree to which common genetic variation from across the genome accounts for variation in a trait or behavior (i.e., SNP heritability). The standard GCTA model uses a genetic relationship matrix based on identity-by-state calculations inferred from genome-wide data from unrelated individuals to estimate the contribution of genetic factors to variation in an outcome, expressed as $V = G + E$, where G represents the additive genetic variance, and E represents the unique environmental variance. Using an efficient restricted maximum likelihood (REML) estimator, the GCTA model estimates the degree to which each chromosome contributes to additive genetic variance in the outcome. More recently, a dyadic extension of GCTA was developed to allow one to quantify the contributions of partners in a dyad to a shared outcome (Eaves et al. 2014). This dyadic extension, known as M-GCTA, was originally developed for applications to mother-child dyads. Here, we tailor the description to specifically address spousal dyads for which there is genome-wide association data on both partners.

The basic path model for proband and partner effects in spousal dyads is presented in Figure 3. For simplicity, this model assumes random mating and additive genetic effects. The dyadic phenotype, T , is influenced by both the proband and spouse genotypes, G_p and G_s , as well as the unique environmental effects, E . Thus, the phenotype for dyad i is: $T_i = p(G_{p,i}) + s(G_{s,i}) + e(E_i)$. To conduct the GCTA analysis for this example, one would first arrange the pedigree and genotype files such that data for all probands are followed by data for all spouses (with the same ordering of dyad identifiers within probands and spouses). Then, one would calculate the genetic relationship matrix. The top left quadrant of this matrix will include the genetic relationship matrix describing the relatedness among probands, whereas the bottom right quadrant will include describe the relatedness among their spouses. These quadrants can then be extracted and fit in linear mixed models using the standard GCTA algorithm (Yang et al. 2011) to examine the proportion of variance that each partner's genome makes to the shared phenotype.

Weighted familial risk scores.

The extended GCTA method detailed above describes the proportion of variance that each partner's genotype accounts for in the shared phenotype. One might also be interested in examining how personalized measures of partners' predispositions may contribute to a shared outcome. One way to do this would be to construct personalized "familial risk" indicators for each partner based on information regarding their family history for the trait under study. Using divorce as an example, one could calculate additive linear composites of each individual's familial loading for divorce based on pedigree information (e.g., divorce history among parents and grandparents) from self-reports or population-based registries. Scoring routines can be weighted based on degree of genetic sharing; for example, a history of divorce in parents is weighted twice as strongly as a history of divorce in grandparents or aunts/uncles. We note that although these scores can be weighted by degree of genetic sharing, they reflect both genetic and environmental sources of familial influence for individuals raised in their biological families.

These familial risk scores can be treated as one would any other manifest variable, making it possible to incorporate them into standard regression-based models to understand the pathways from partners' familial loading to their shared outcome. For example, one can examine whether partners' familial risk scores contribute equally to a dyadic outcome; whether partners' risk scores have additive or interactive effects; and whether other factors (e.g., sex, power, satisfaction, presence of children) moderate the effects of each partner's familial risk score. A spouse's weighted familial risk score for divorce could also be incorporated into a conventional twin design to examine whether genetic and environmental variation in divorce (as estimated from the twin model) changes as a function of the spouse's familial risk. Although familial risk scores are an imperfect proxy of the spouse's genotype, evidence for a $G \times$ spousal familial risk effect could be interpreted as evidence that partners' genetic predispositions interact to predict variation in their shared phenotype.

Genome-wide polygenic scores.

The availability of relatively low-cost genome-wide genotyping arrays has made genome-wide polygenic scores an increasingly common way to incorporate molecular genetic

measures into behavioral studies (Bogdan et al. 2018; Wray et al. 2014; Dudbridge 2016). In samples where genome-wide data are available on both partners it is thus possible to examine the contribution of each partner's polygenic scores (derived from a theoretically related phenotype) to a shared outcome. Genome-wide polygenic scores are personalized estimates of the number of alleles an individual carries that are associated with a trait of interest (Wray et al. 2014). The alleles for any particular variant are weighted by their effect sizes from an independent genome-wide association study, such that variants with larger effect sizes are upweighted. As with the weighted familial risk scores described above, polygenic scores can be incorporated into standard regression-based models.

Although there is a great degree of enthusiasm surrounding genome-wide polygenic scores (Maier et al. 2017), there are several concerns one should consider before using them to predict dyadic outcomes. One concern is that polygenic scores typically account for a minimal proportion of the variance (i.e., $< 1\%$), even when looking at the same phenotype (for an exception, see educational attainment (Lee et al. 2018), which accounts for 11–13% of the variance). Thus, large sample sizes are needed to detect these small effects. A related concern is the incremental validity and theoretical meaning of a polygenic score above and beyond the individual-level phenotype from which the polygenic score is derived. Consider the example of whether partners' polygenic scores for neuroticism (Lo et al. 2017) predict marital dissolution. Under the assumption that these polygenic effects are likely to be mediated via partner personality, for which there is already a robust association with relationship instability (Karney and Bradbury 1995), whether the added expense and effort of generating genome-wide association data and calculating polygenic scores adds much explanatory value is unclear.

Other Methods for Incorporating a Dyadic Perspective in Genetically Informed Research

Although our focus in this paper is primarily on the need to incorporate information from both partners in genetically informed research on joint outcomes, we would also like to briefly touch on other methods for incorporating a dyadic perspective in genetically informed research. Specifically, we turn our attention to social genetic effects, which refer to the influence of a social partner's genotype on a proband's phenotype (Wolf et al. 1998). Although these types of effects would not necessarily be considered emergent, we felt this topic was important to include as part of the broader discussion on incorporating a dyadic perspective in genetically informed research given the increasing interest in these methods.

By way of background, we ground this discussion in the literature on direct and indirect genetic effects. The field's current approach to characterizing genetic influences on complex traits and behaviors takes an individual perspective, mapping one's own genetic variation onto phenotypic variation. This reflects an implicit *direct genetic effects assumption*, which posits that one's genotype influences *only* one's own phenotype (shown in Figure 4A). In contrast is the concept of *social* or *indirect genetic effects*. As shown in Figure 4B, social genetic effects refer to the effect of a social partner's genotype on a proband's phenotype (i.e., the effect of Person 1's genotype a_j on Person 2 is transmitted indirectly through

Person 1's phenotype Z_j). Thus, from the point of view of a focal individual, social genetic effects are considered environmental effects. Social genetic effects have a long history in evolutionary and human genetics (Wolf et al. 1998; Moore et al. 1997), and have typically been studied in the context of related individuals, with an example being the effect of maternal genotype on an offspring's outcome (Kirkpatrick and Lande 1989), such as gestational age at birth (York et al. 2013). Only recently have researchers begun to investigate social genetic effects in unrelated individuals, with intriguing preliminary findings demonstrating that the genotypes of friends and schoolmates predict one's own educational attainment (Domingue et al. 2018) and the effects of nontransmitted parental alleles on children's educational outcomes (Bates et al. 2018; Kong et al. 2018).

In a compelling preclinical demonstration of the significance of social genetic effects, Baud et al. (2017) used inbred strains of mice to quantify direct and indirect (i.e., social) genetic effects for 50 biomedical-related phenotypes related to anxiety, helplessness, body weight, stress, wound healing, and social dominance. To do this, they co-housed C57BL/6J (B6) and DBA/2J (D2) mice in like-strain and different-strain pairs (e.g., B6/B6, B6/D2, and D2/D2) for six weeks prior to collecting the phenotypic measures. Standard variance partitioning models were then applied to each phenotype, considering three sources of genetic variation: the focal mouse's, their cage mate's, and the interaction between the two. In total, there was evidence for social genetic effects for 11 of these phenotypes, with the cage mate's genotype accounting for 8–18% of the variation in the focal mouse's phenotype. In addition, there was evidence for an interaction between the focal mouse and cage mate's genotypes to predict variation in the anxiety, helplessness, and wound healing measures. Thus, this study provides some of the most persuasive experimental evidence for social genetic effects for health-related outcomes.

With respect to human studies, the availability of low-cost genotyping has also stimulated interest in examining social genetic effects. In an early example of social genetic effects from a small sample, investigators found that women's sexual responsivity and unfaithfulness changed as a function of how similar they were to their romantic partner across a series of major histocompatibility complex (MHC) alleles (Garver-Apgar et al. 2006). In a more recent example, the GCTA model discussed previously was applied to genome-wide association data to decompose variation in offspring length at birth into direct and indirect (i.e., social or maternal) genetic effects (Eaves et al. 2014).

The increasing popularity of genome-wide polygenic scores has also stimulated interest in whether the genotype of a social partner or partners predicts individual phenotypic outcomes in samples that include genome-wide data on dyads or social networks. In samples of unrelated individuals, social genetic effects have been examined using multilevel modeling approaches (which account for the nested structure of individuals within dyads or groups). In an application of this method to school-based data from the Add Health sample, friends' and classmates' polygenic scores for educational attainment predicted individuals' own educational attainment above and beyond their own polygenic scores (Domingue et al. 2018).

Studies of social genetic effects using polygenic methods have also been extended to family-based designs. Of particular interest has been the question of whether the alleles that are not transmitted from parents to offspring are associated with offspring outcomes. From the perspective of the offspring, these non-transmitted alleles represent a social or environmental effect. To examine this, investigators construct polygenic scores for parents that include only the variants that were not transmitted to their offspring, and examine whether these are associated offspring outcomes. Across two independent samples, polygenic scores for educational attainment derived from non-transmitted parental alleles were associated with offspring's educational attainment (Bates et al. 2018; Kong et al. 2018). Such findings have been interpreted as the genetic basis of nurture.

These methods provide novel ways to incorporate genetic data into relationships and social networks research; however, it is again worth asking about the incremental validity of polygenic scores in these types of studies of social influence given that we experience our social partners' genotypes through their phenotypes. For example, the non-transmitted parental alleles for educational attainment were associated with socioeconomic status (Bates et al. 2018), raising the question of whether social genetic effects are simply duplicative of phenotypic effects. Although this remains an open question, one possibility for exploring this would be examine social genetic effects in scenarios where a social partner's genotype has become separated from their phenotype. Consider the example of individuals who have high polygenic scores for alcohol use disorder but who do not drink to excess. If there is an association between these individuals' polygenic scores and their spouse's problematic drinking (beyond the spouse's own polygenic score), this could be evidence that the polygenic score has incremental validity above and beyond the proband's own drinking phenotype.

Summary and Conclusion

Behavioral genetics has much to offer relationships research; likewise, the unique perspective of relationships researchers has much to offer those interested in understanding the genetic influences on behavior. To date, the majority of genetically informed relationships research has taken an individual perspective to decompose the variance in relationship-related outcomes into genetic and environmental influences, or examine whether relationship factors moderate the genetic influences on health-related outcomes. We advocate for the incorporation of a dyadic perspective in genetically informed relationships research to begin to better reflect the emergent nature of relationship outcomes and the contributions of both partners. Such an approach would provide a more complete picture of how genetic factors influence relationship outcomes, and the complex pathways from genotype to phenotypes that are influenced by more than one individual.

Acknowledgements

This project was supported by a Genetics and Human Agency Junior Investigator Award to JES. Additional support comes from K01AA024152 from the National Institute on Alcohol Abuse and Alcoholism. This work is solely the responsibility of the authors and does not necessarily represent the official view of the funders.

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Figure 1.

Illustrative univariate twin model for a phenotype, T . MZ, monozygotic; DZ, dizygotic; A , additive genetic factors; C , shared environmental factors; E , nonshared environmental factors; T_1 , phenotype for twin 1; T_2 , phenotype for twin 2. Genetic, shared environmental or unique environmental factors contribute to the variation in phenotype T if model fit is worsened when a' , c' , or e' parameters (respectively) are removed.

Figure 2.

Extension of the univariate twin model that considers the genetic and environmental contributions of twins and their spouses to a shared phenotype, T. MZ, monozygotic; DZ, dizygotic; A, additive genetic factors; C, shared environmental factors; E, nonshared environmental factors; T_1 , phenotype for twin 1 and spouse 1; T_2 , phenotype for twin 2 and spouse 2; S, spouse; P, twin proband. Genetic, shared environmental or unique environmental factors contribute to the variation in phenotype T if model fit is worsened when a' , c' , or e' parameters (respectively) are removed.

Figure 3.

Basic path model for proband and spouse effects for a shared phenotype, T. G_P , proband genotype for loci that contribute to genetic variation in T; G_S , spouse genotype for loci that contribute to genetic variation in T; E, random environmental effects that contribute to variation in T.

Figure 4.

Illustration of direct and social genetic effects (adapted from Wolf et al., 1998). Primes denote values for a partner of the focal individual (person 2). The effect of the environment provided by person 1 on person 2 is represented as $e_{Z'j}$. The dashed line denotes the effect of Z'_j on Z_i .