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Assessing causal pathways between physical formidability and aggression in human males

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ABSTRACT

Studies suggest the existence of an association between the physical formidability of human males and their level of aggression. This association is theoretically predictable from animal models of conflict behavior but could emerge from multiple different causal pathways. Previous studies have not been able to tease apart these paths, as they have almost exclusively relied on bivariate correlations and cross-sectional data. Here, we apply longitudinal twin data from two different samples to (1) estimate the direction of causality between formidability and aggression by means of quasi-experimental methods and (2) estimate the relative contribution of genetic and environmental factors by means of twin modeling. Importantly, the results suggest, on the one hand, that the association between formidability and aggression is less reliable than previously thought. On the other hand, the results also suggest that when the association occurs, the causal direction is from formidability to aggression and the primary part of the causal relationship is genetic in nature. These latter findings are consistent with adaptionist models suggesting that human male aggression is 'reactively heritable' to genetic components of formidability.

1. Introduction

A number of recent studies have demonstrated that aggressiveness in human males is associated with physical formidability (Archer & Thanzami, 2007; Gallup, White, & Gallup, 2007; Petersen, Sznycer, Sell, Cosmides, & Tooby, 2013; Price, Kang, Dunn, & Hopkins, 2010; Price, Dunn, Hopkins, & Kang, 2012; Raine, Reynolds, Venables, Mednick, & Farrington, 1998; Sell, Tooby, & Cosmides, 2009; Tremblay, 1998). Higher physical formidability – in particular upper-body strength – is associated with more aggressive predispositions as indexed by a number of different measures such as aggressiveness (Archer & Thanzami, 2007; Gallup et al., 2007; Raine et al., 1998; Tremblay, 1998) and social dominance orientation (Price et al., 2012). A functional relationship between formidability and aggressiveness is straightforwardly predicted by models of conflict behavior within evolutionary biology; in particular, the Asymmetric War of Attrition model (Hammerstein & Parker, 1982; Maynard Smith & Parker, 1976). Because relatively larger animals can inflict greater costs than relatively smaller animals, a larger animal should be more inclined to escalate conflicts. In line with this, an association between size and success in conflicts has been reliably established across a number of animal species (Kelly, 2008), and the relationship between formidability and aggressiveness in humans can be viewed as yet another piece of evidence of the general selection pressure captured by the Asymmetric War of Attrition model (Puts, 2010; Sell et al., 2009).

While the association between formidability and aggressiveness is theoretically predictable and seems to be empirically robust (Durkee & Goetz, 2017), the underlying causal mechanism remains unclear. This is in particular due to a number of methodological limitations in previous research that prevents discerning between the different potential causal mechanisms (see also Isen, McGue, & Iacono, 2015). Most previous studies of the association between formidability and aggressiveness have relied on cross-sectional data and tested the predictions by way of correlation (see Archer & Thanzami, 2007; Gallup et al., 2007; Petersen et al., 2013; Price et al., 2010; Price et al., 2012; Sell et al., 2009; however, see Raine et al., 1998; Tremblay, 1998; Isen et al., 2015), so the direction of causality remains unclear. Furthermore, behavior genetics divides the causes of individual variation in traits such as physical formidability and aggression into environmental and genetic causes. At present, we know little about the relative role of these causes for the formidability-aggression link due to specialized data requirements for adjudicating between genetic and environmental factors (although see Lukaszewski & Roney, 2011). In this manuscript, we seek to use samples with unique properties for simultaneously assessing (1) the direction of causality and (2) the relative role of genetic and environmental causes for the formidability-aggression link.

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On one potential view, the correlation between formidability and aggression could arise from individual differences in aggression exerting a causal effect on formidability. Hence, humans can, albeit slowly, build upper-body strength through focused training or physical labor (Abe, DeHoyos, Pollock, & Garzarella, 2000). If formidability is a bargaining tool, it is possible that motivations to bargain for a greater share of resources activate motivations to build muscle mass to more successfully implement the strategy (Isen et al., 2015). Consistent with this, one study finds that social dominance orientation in British males correlates positively with drive for muscularity, that is, the desire to build up muscle mass (Swami et al., 2013). Also, another recent study found that boys, that teachers characterized as antisocial and aggressive, developed more physical strength during puberty than boys characterized as social and non-aggressive, potentially suggesting that aggressiveness is causally prior to strength (Isen et al., 2015).

This account would suggest (1) that the direction of causality goes from aggression to formidability and (2) that this link is primarily environmental in nature (i.e., reflecting physical labor or training). However, both empirical observations and adaptationist logic speak against this argument. First, against this explanation speaks the anthropological observation that dedicated training focused on building muscle mass is not a human universal and, in particular, not observed in forager cultures (Eaton & Eaton, 2003). If trait aggressiveness triggers an adaptation to onset motivations to build up strength, we would expect to see such activity even among foragers. Second, physiologically, building muscle does not require using the muscles. All our muscles are built in the womb without resistance training. The fact that muscles grow in response to resistance training is because of an adaptation that adds muscle mass when muscles experience microtrauma (Charge & Rudnicki, 2004). An adaptation to grow more muscle in response to motivations for more bargaining would be inefficient if it simply motivated wasting energy tearing one's muscles in order to grow more.

Given such observations, evolutionary psychologists have primarily pursued another argument. This adaptationist argument has been referred to as “facultative calibration” and focuses on how variation in personality traits are regulated by contextual and other individual differences in fitness-enhancing ways (Buss, 2009; Tooby & Cosmides, 1990). In the specific context of the formidability-aggression link, the application of this argument privileges (1) a causal effect from formidability to aggression and (2), in some versions, genetic causes over environmental causes.

According to the facultative calibration account, differences in basic physiological traits are important because they provide the basis for adaptive differences in personality (Buss, 2009; Gosling & John, 1999; Tooby & Cosmides, 1990). In noisy information environments where organisms occasionally misjudge situations, adaptations will emerge that allow individual differences in personality and strategy (i.e., stability in behavior across situations) to emerge from lower-level trait differences that influence the costs of different misjudgments (McElreath & Strimling, 2006). For example, a less formidable individual will, all else equal, pay greater costs from a misjudged act of conflict escalation relative to a more formidable individual. Accordingly, it pays for a non-formidable individual to err on the side of caution and be consistently less aggressive than a formidable individual. Whereas a training-oriented account suggests that causality could flow from aggression to formidability, facultative calibration entails that formidability causally influences aggression rather than vice versa.

In principle, this calibrational process of aggression to formidability could be both environmental in nature and genetic in nature. A number of adaptionist researchers have, however, pointed to a potential important role for genetic factors in this regard. Heritable personality (here, aggression) could emerge, at least in part, because the input conditions of universal, adaptive mechanisms for regulating strategies are reactive to input that is heritable (here, physical formidability). This mechanism has been referred to as ‘reactive heritability’ (Buss, 2009; Tooby & Cosmides, 1990). Indeed, research shows that between 50 and 60% of individual variation in physical strength is caused by genetic differences (Silventoinen, Magnusson, Tynelius, Kaprio, & Rasmussen, 2008). In the context of the association between physical formidability and aggression, the reactive heritability version of facultative calibration suggests that the effect of formidability on aggression is to a significant extent caused by underlying individual differences in genetic dispositions for formidability.

All accounts considered at this point suggests that there is indeed a causal relationship between formidability and aggression and disagree on the direction and nature of this relationship. Importantly, however, a final possibility should be raised. It could be that the relationship is not causal but, instead, that there is a common cause that simultaneously shape both aggression and formidability. One candidate for a common cause is pleiotropic gene effects. For example, some studies suggest that particular genetic polymorphisms affect both formidability and the personality trait of extraversion (Lukaszewski & Roney, 2011) and similar patterns could occur for formidability and aggression. Another potential sets of common causes could be hormonal in nature with, for example, early testosterone exposure shaping the developmental trajectory of both aggression and formidability (see, e.g., Isen et al., 2015).

In sum, this manuscript aims to decompose the association between formidability and aggression identified in the previous literature. The focus is to answer two distinct questions: (1) what is the direction of causality and (2) what is the relative role of genetic and environmental causes? The adaptionist theory of reactive heritability, in particular, proposes that aggression is calibrated to genetic components of formidability and thus hypothesizes, first, that causality flows from formidability to aggression and, second, that genetic causes dominate environmental ones. In addition, there is the possibility that associations are influenced by causes common to both aggression and formidability. We return to this final possibility in the discussion.

2. Methods

To investigate the role of reactive heritability in the aggression-formidability link, we use The Minnesota Twin Family Study (MTFS) (see also Isen et al., 2015). The MTFS is a population-based, longitudinal study of over 1800 twin pairs and their parents (Iacono, McGue, & Krueger, 2006). The samples within the study have unique properties that provide us with exceptional possibilities of testing the two claims of a reactive heritability perspective on the formidability-aggression link: First, they have a panel structure with multiple waves that should allow the use of quasi-experimental designs to gauge causality between formidability and aggression. Second, they have a twin component that should allow us to differentiate between genetic and environmental accounts of the formidability-aggression link.

2.1. Samples

The MTFS has a longitudinal component that allows us to shed light on the causal direction of the link (see below under ‘Analysis’). Specifically, data was collected on the twin pairs at an initial assessment as well as follow-ups for the children occurring at roughly 3-year intervals. The MTFS is comprised of two separate age cohorts, one in which subjects were 11 years old at the time of their initial assessment and one in which subjects were 17 years old. The younger cohort was born between 1977 and 1984, and the older cohort was born between 1972 and 1979. Participants in the samples have been shown to be comparable to the overall Minnesota population (Iacono et al., 2006). To maximally utilize the longitudinal data for causal inference, we need measures of both formidability and aggression at two time points. This is only available for the older cohort, in which both types of measures are available at ages 17 and 24. In the younger cohort measures of both formidability and aggression are available at age 17 and a measure of aggression is available at age 24.