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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.

Highlights

- Male physical formidability is correlated with trait aggressiveness.
- Correlations are not as reliable as suggested by previous research.
- Cross-lagged regression models show that formidability has a causal effect on aggressiveness.
- Analyses of twins show that the effect of formidability on aggressiveness is driven by genes.
- Overall, analyses support the theoretical notion of “reactive heritability”.

A number of recent studies have demonstrated that aggressiveness in human males is associated with physical formidability (Archer & Thanzami, 2006; Gallup, White & Gallup, 2007; Petersen et al., 2013; Price et al., 2010; Price et al., 2012; Raine et al., 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, 2006; Gallup, White & Gallup, 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, Tooby & Cosmides, 2009).

While the association between formidability and aggressiveness is theoretically predictable and seems to be empirically robust, 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, 2006; Gallup, White & Gallup, 2007; Petersen et al., 2013; Price et al., 2010; Price et al., 2012; Sell, Tooby & Cosmides, 2009; however, see Raine et al., 1998; Tremblay, 1998; Isen, McGue, & Iacono, 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 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.

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 et al., 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, McGue, & Iacono, 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., 2000). Also, another recent study found that boys, that teachers characterized as antisocial and aggressive, developed more physically during puberty than boys characterized as social and non-aggressive, potentially suggesting that aggressiveness is causally prior to strength (Isen, McGue, & Iacono, 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 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 adaptationist 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 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 50 and 60 % of individual variation in physical strength is caused by genetic differences (Silventoinen et al., 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, McGue, & Iacono, 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.

Methods

To investigate the role of reactive heritability in the aggression-formidability link, we use The Minnesota Twin Family Study (MTFS) (see also Isen, McGue, & Iacono,

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 perception 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.

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, McGue, & Krueger, 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.

From these cohorts, we select all males as we seek to shed light on the link between physical formidability and aggression that past studies have uncovered in

human males specifically. We end with a sample of 578 male twins from the older cohort (378 monozygotic twins and 200 dizygotic twins) and an additional 752 male twins (504 monozygotic twins and 248 dizygotic twins) from the younger cohort. Because of missing data on one or more measures, fewer twins are included in specific analyses.

Measures

To test the reactive heritability account, we need measures of individual differences in aggression and formidability.

The MTFS has longitudinally collected detailed measures of personality traits for participants, which allows us to measure aggression. As part of the study, subjects completed the 262-question Multidimensional Personality Questionnaire (MPQ) (Tellegen & Waller, 2008). The MPQ is comprised of 11 primary factors that are aggregated up to three higher factors (positive emotionality, negative emotionality, and constraint). Those scoring high for the MPQ primary factor *aggression* self-report being physically aggressive and tend to enjoy observing violence.¹ We use the MPQ aggression scale collected for each individual at around ages 17 and 24.

To measure formidability, we rely on measures used in previous studies. Specifically, we computed three measures of physical formidability: (1) handgrip strength of the right hand to directly gauge strength (Gallup, White, & Gallup, 2007; Sell, Tooby, & Cosmides, 2009), (2) bicep circumference (Petersen et al., 2013; Price et al., 2011; Price et al., 2012; Sell, Tooby, & Cosmides, 2009),² and (3) bulk

¹ Those scoring high on the aggression scale agree with statements such as ‘I enjoy violent movies’, ‘When I get angry I am often ready to hit someone’, ‘I admit that I sometimes take pleasure in hurting someone physically, and ‘Sometimes I just like to hit someone’.

² To truly gauge muscularity using the measure of bicep circumference, it would be important to correct for upper-arm skinfold thickness. Unfortunately, this measure is not available for the samples. In addition, some previous studies have used flexed bicep circumference (Petersen et al., 2013; Sell,

measured as the product of height (in centimeters) and weight (in kilograms) to gauge overall physical size (Raine et al., 1998). At age 17, all measures are available for both cohorts. At age 24, all measures are available for the older cohort but only bulk is available for the younger cohort. Our main analyses are based on scales of these measures and, hence, we z-scored the measures and then computed their arithmetic mean for each subject. At both ages, the scale reliability for our formidability measure is lower than optimal but sufficient to proceed (age 17: $\alpha=.67$; age 24: $\alpha=.66$).

Analyses

We seek to investigate two arguments: First, whether formidability is the causal driver of any formidability-aggression link; second, whether a substantial part of any effect is genetic in nature. We use different parts of the study design and set up different models to test of each of these claims.

Direction of causality. To test the direction of causality between aggression and formidability, we use the panel structure of the data. In general, questions about causality are difficult to answer without experimental data and random assignment, but the two-wave longitudinal study provides us with significant traction on the issue (Campbell & Stanley, 1963). Specifically, it allows us to use a quasi-experimental design: a cross-lagged regression model (Kenny, 1975). The existence of a causal effect of formidability on aggression requires us to find (1) a significant effect of formidability at age 17 on aggression at age 24, controlling for aggression at age 17, (2) a non-significant effect of aggression at age 17 on formidability at age 24, controlling for formidability at age 17, and (3) that (1) is significantly stronger than

Tooby, & Cosmides, 2009); others have used relaxed bicep circumference (Price et al., 2011; Price et al., 2012). Only relaxed circumference is available at both ages, so we rely on this measure. At age 24, both flexed and relaxed circumference are available, and we can observe that they are highly correlated ($r=.93$, $p<.001$).

(2). In common sense terms, the existence of this set of effects (with the predicted sign) would suggest that changes in aggression from age 17 to age 24 move the personality trait into greater synchronization with formidability, implying that formidability exerts a causal pressure on an individual's personality. While a few previous studies on the aggression-formidability link have used longitudinal observations (Raine et al., 1998; Tremblay, 1998), these studies have not used an analytical framework that allows for causal identification.

The cross-lagged models are estimated using structural equation modeling. We report standardized coefficients (betas) estimated using structural equation modeling and reported two-sided p-values estimated on the basis of cluster robust standard errors with twin pair ID as cluster variable (to correct for within-pair autocorrelation). Because the twins are surveyed within a relative wide age range (e.g., for measures taken around age 17, the youngest subject is 16 and the oldest is 20), we also control for respondents' ages.

Genetic versus environmental effects. To identify whether any effect of formidability on aggression has a substantial genetic origin, we rely on the twin component of the sample. The classic twin design relies on monozygotic (MZ) twins, who share 100% of their genes, and dizygotic (DZ) twins, who are no more genetically similar than ordinary siblings. Imposing the assumption that MZ and DZ twins experience comparable exogenous environmental conditions (known as the equal environments assumption), the similarity for a trait among MZ twin pairs to that of DZ twin pairs can be compared in order to obtain an estimate of the degree to which genes influence that trait.

The so-called univariate twin model assumes that the variance in an observed trait can be partitioned into additive genetic factors (A), environmental factors that are

shared or common to co-twins (C), and unique environmental factors (E). Shared environment includes the family environment in which both twins were raised and any other factor to which both twins were equally exposed. In contrast, the unique environment includes influences that are experienced individually. The proportion of the overall variance of a trait attributed to additive genetic factors is known as *heritability*.

The bivariate twin model estimates how much of the covariation between two traits – in this case, formidability and aggression – can be attributed to the same genetic and environmental sources. Specifically, we utilize a bivariate Cholesky decomposition model (Martin & Eaves, 1977). This model assumes that the environmental and genetic factors underlying formidability influence aggression but not vice versa. In other words, if the results from the cross-lagged models show that formidability influences aggressions and not vice versa, this assumption for the Cholesky model is met. In addition, the model assumes that the latent genetic and environmental factors underlying formidability and aggression are uncorrelated with one another across and within individuals.

The parameter estimates generated by the bivariate Cholesky model can be used to construct two quantities of interest. First, the genetic correlation and environmental correlations (r_A , r_C , r_E) capture the degree to which the genetic endowment of two traits covary. An $r_A = 0$ means the two traits are influenced by completely different genes, and a $r_A = 1$ means the same genes influence both traits. Second, the phenotypic correlation between two traits can be partitioned into the proportion explained by additive genetic and environmental factors (Proportion_A , Proportion_C , Proportion_E). A large estimate for Proportion_A suggests that the

relationship between formidability and aggression is primarily driven by genetic factors.

We estimate the Cholesky model using structural equation modeling. To the extent that the cross-lagged model is consistent with predictions, it will have established a causal effect of formidability at age 17 on aggression at 24. In the Cholesky model, we therefore specifically decompose this effect. To take individual differences in age, the aggression and formidability traits are residualized on age and cohort before analysis.

Initial sample validation for causal analysis

Our focus in this manuscript is to discern between different possible causal pathways between formidability and aggression. In order to validate that the available samples allow us to decompose the association between formidability and aggression into these pathways, the first necessary step is to examine whether we are indeed able to replicate previous findings of an association between formidability and aggression in these specific cohorts. Specifically, for each cohort, we estimate the correlation between the measures of formidability, on the one hand, and the two measures of aggression on the other. The findings appear in Table 1. This initial replication effort leads to the conclusion that the formidability-aggression link is less robust than expected. The link is detectable in the older cohort (the upper rows of Table 1) but not in the younger cohort (the middle rows of Table 1). In addition, the effect sizes for the older cohort are relatively small.

Table 1

Correlations between measures of formidability and aggression for different cohorts and ages

Cohort	Aggression	<u>Formidability</u>	
		Age 17	Age 24
Older	Age 17	$r=.12$; $p=.04$	$r=.13$; $p=.02$
	Age 24	$r=.17$; $p=.002$	$r=.15$; $p=.009$
Younger	Age 17	$r=-.04$; $p=.44$	-
	Age 24	$r=.02$; $p=.69$	-

Note. N (Older cohort) = 303, N (Younger cohort) = 420. Entries are pearson correlation coefficients. Reported p-values are two-sided.

It is unclear why there is this difference between the two cohorts. This difference does, however, imply that we can only decompose the association between formidability and aggression for the older cohort. This will be our focus in the following analyses. We therefore emphasize that all of the conclusions of the subsequent analyses become conditional: if there--given the set of circumstances--is a link between formidability and aggression, what then is its nature? For completeness, we provide parallel analyses for the younger cohort in the online supplementary information. We return to this difference between the cohorts in the conclusion.

Results

What is the direction of causality between formidability and aggression?

The association between formidability and aggression in the older cohort is consistent with both an account that stresses the facultative calibration of aggression to

formidability and an account stressing that trait aggression activates a drive for building formidability. These two accounts, however, differ sharply in their prediction on the direction of causality with facultative calibration implying that formidability is the causal driver while the alternate account implies that aggression is the causal driver.

To discern between these possibilities, we used cross-lagged regression models to predict aggression and formidability, respectively, at age 24 on the basis of these traits at age 17. Figure 1 shows the results of this analysis for the older cohort where an association between formidability and aggression was identified. In the older cohort the results provided clear support for the role of reactive heritability over the role of an aggression-driven motivation for formidability. Controlling for aggression at age 17, formidability at age 17 had a positive and significant effect on aggression at age 24. In reverse, controlling for formidability at age 17, aggression at age 17 was statistically unrelated to formidability at age 24. Further analysis showed that this significant effect of formidability on later aggression is furthermore significantly stronger than the non-significant effect of aggression on later formidability ($\chi^2=3.96$, $df=1$, $p = .047$).

Overall these analyses suggest that if there is a link between formidability and aggression, the effect flows from formidability to aggression. Hence, for the older cohort where the initial correlational tests identified a link, the cross-lagged analyses suggested that if formidability and aggression are asynchronous at age 17, later developments in aggression are pulled in the direction of formidability rather than vice versa. In other words, consistent with the theory of facultative calibration, formidability is the causal driver of any formidability-aggression association, not aggression.

Figure 1. Cross-lagged effects of formidability on aggression and aggression on formidability. $N = 303$. Entries are standardized coefficients (betas) estimated using structural equation modeling. Coefficients have been controlled for age when ‘age 17’ measures were obtained. Reported p -values are two-sided p -values estimated on the basis of cluster robust standard errors with twin pair ID as cluster variable.

Is aggression calibrated by genetic or environmental variation in formidability?

The analyses so far suggest that when the association between formidability and aggression occurs it is driven by a causal effect of formidability on aggression. This finding is consistent with the theory of facultative calibration, suggesting that the mind contains a system for the adaptive regulation of social strategies (i.e., personality) on the basis of lower-level features. In the final test, we turn towards the question whether aggression is calibrated to the environmental or genetic components of formidability.

To this end, we applied the twin component of our study design. As a first step, we establish the univariate estimates of heritability, common environment, and unique environment for our focal variables, formidability at age 17 and aggression at age 24. As these analyses do not hinge on the existence of an association between formidability and aggression, we estimated univariate heritability on the basis of models that pooled the younger and older cohort. In total, we had 253 complete monozygotic twin pairs and 137 complete dizygotic twin pairs available for this analyses (see Table A2 in the online appendix for descriptives). The results are

displayed in Table 2. As reflected in the confidence intervals, the heritability and unique environment estimates for both were significantly different from zero, with the former being larger than the latter in both cases. The estimate for shared environment was not statistically distinguishable from zero, implying that family socialization does not influence either of the traits strongly. These findings are consistent with previous twin studies on the heritability of both formidability (e.g., Silventoinen et al., 2008) and aggression (Miles & Carey, 1997).

Table 2

Heritability estimates for formidability and aggression

	Formidability (age 17)	Aggression (age 24)
Heritability (A)	0.70 (0.48, 0.85)	0.54 (0.31, 0.62)
Shared Environment (C)	0.12 (0.00, 0.34)	0.00 (0.00, 0.20)
Unique Environment (E)	0.18 (0.15, 0.22)	0.46 (0.38, 0.55)

Note. N = 253 MZ pairs and 137 DZ pairs. Entries are parameter estimates and 95% confidence intervals (in parentheses) for a univariate ACE model. Both traits are residualized on cohort and age.

On the basis of these results, we turned towards decomposing the effect of formidability on aggression for the older cohort where we identified the effect. Since the common environment estimates for aggression is zero and non-significant for both traits in the univariate model, we estimate a bivariate Cholesky model. The genetic and environmental correlations for both traits are presented in Table 3.

Table 3

Bivariate Cholesky decomposition model of effect of formidability (age 17) on aggression (24)

	Estimates
r_A	0.35 (0.03, 0.86)
r_C	-1.00 (-1.00, 1.00)
r_E	-0.01 (-0.19, 0.16)

Note. N = 120 MZ pairs and 65 DZ pairs. The entries the Additive Genetic (r_A), Common Environmental (r_C) and Unique Environmental (r_E) Correlation and 95% CIs (in parentheses) from a bivariate Cholesky model of formidability and aggression. The traits are residualized on age.

For the older cohort, where a causal association between formidability and aggression was identified, we find a significant genetic correlation between formidability and aggression and non-significant environmental correlations. In itself, these results would merely suggest that the relationship between formidability and aggression is primarily genetic in nature but would still leave open to interpretation the precise nature of this relationship. However, when combined with the evidence from the cross-lagged model establishing a clear causal effect from formidability on aggression but not vice versa, the Cholesky model provides supportive evidence in favor of reactive heritability: for this sample, individual differences in aggression are calibrated by heritable differences in physical formidability.

Discussion and Conclusion

A substantial number of studies have investigated and found an association between men's physical formidability and their level of aggressiveness with more formidable men being more aggressive. In this manuscript, we sought to answer important

outstanding questions about the formidability-aggression link by utilizing a dataset with unique design features: a panel study of the physical formidability and aggression of male twins. Such a dataset provides the possibility of investigating (1) the direction of causality in the formidability-aggression link and (2) the relative role of genetic and environmental factors.

The analyses returned mixed insights. On the one hand, in relation to the two questions guiding this research, the present analyses used a quasi-experimental setup and suggested (1) that formidability in males has a causal effect on aggression and (2) that this causal effect is particularly tied to the genetic components of individual differences in formidability. This is consistent with the adaptationist notions of the facultative calibration and the reactive heritability of personality traits. On this account, evolution by natural selection has installed mechanisms in humans (and other animals) designed to calibrate trait aggression to formidability, and because of heritable differences in formidability, differences in aggression emerge as a result. This provides an illustrative case of how evolved mechanisms and genetic, individual differences work in tandem to produce individual behavior (Tooby & Cosmides, 1990). In providing empirical support for the theoretical notion of reactive heritability, these results challenge an environment-oriented account on the aggression-formidability link that would suggest that people who are aggressive seek out opportunities to increase muscle mass (see, e.g., Swami et al., 2000).

On the other hand, these empirical patterns were only detectable in one out of two twin cohorts. In the younger cohort, no simple association between formidability and aggression was detectable. As consequence, as detailed in the Online Appendix, no further evidence of the direction of causality or the influence of genetic and environmental factors in creating this (lack of) association could be generated in this

younger cohort. This suggests that the link between formidability and aggression is less reliable than previous studies have suggested.

Any deeper interpretation of the lack of effect in one but not the other cohort is, of course, speculative. That said, one possibility is that the effect of formidability on aggression is moderated by some contextual factors and, hence, only emerges under specific circumstances. One study, for example, finds that while there is no link between formidability and aggression among Western females, there is such a link among Aka females, who live under more severe conditions than Westerners (Hess et al., 2010). Some unspecified event could have occurred in Minnesota between the older and younger cohort, which muted the relationship for the latter. At the same time, the plausibility of this explanation is low as the similarities in the living conditions of the two cohorts most likely is much larger than the differences.

A second possibility is that there is in fact no relationship between formidability and aggression and that the association in the older cohort is a false-positive. Thus, the “replication crisis” in psychology has suggested that a number of otherwise well-established effects are artifacts generated by selective reporting of data (Open Science Collaboration, 2015). Meta-analysis is an important tool to identify such processes. A recent meta-analysis on the formidability-aggression link reported the results from 32 studies, both published and unpublished (Durkee & Goetz, 2017). Across these studies, a significant positive association with a mean effect size of .13 was found between measures of aggression and formidability. Importantly, methods designed to detect publication bias suggested that this association was not driven by selective publication of positive findings.

In this light, a third possibility is plausible: that the association is real but small and, therefore, difficult to detect reliably. The mean effect size from the meta-

analysis corresponds well to the identified effect size in the older cohort. This is indeed a small effect. Furthermore, the meta-analysis suggests that the effect is even smaller in samples with an age of less than 18 years, as is the case for the younger cohort (in the older cohort we also have the measures at age 24) (Durkee & Goetz, 2017). When an effect is small it is more sensitive to the occurrence of sampling and measurement noise. That said, the finding of a lack effect calls for further replications of the basic formidability-aggression link.

Overall, the conclusions of the empirical investigations are mixed. On that basis, a tenable overall conclusion would be the following: if there is an association between formidability and aggression in a specific sample, then the present study supports the involvement of processes of reactive heritability: that the association (at least, for males) emerges because individual differences in aggressiveness are being causally calibrated to heritable components of physical formidability.

At the same time, it is important to view this conclusion in the light of some limitations of the present study: First, the analyses rests on a limited dataset with lower than optimal statistical power – especially in the light of the small size of the effect. This calls for future replication studies on the direction of causality and the role of genetics in the formidability-aggression link, although the uniqueness of the present data makes such replication attempts difficult. Second, in future studies, it would be worthwhile to obtain even more valid measures of formidability. For example, the bicep circumference measure that was utilized confounds muscularity and fatness (see footnote 2) and in future studies it would be important to, for example, correct this measure for skinfold thickness of the upper-arm. Third, the cross-lagged analysis was based on data with a lag of seven years between observations. On the one hand, this is a significant lag and it would be relevant to seek

to replicate the finding using data with a shorter lag between observations. On the other hand, that formidability is able to exert an effect on aggression over a seven year period suggests that the causal pull stems from highly stable features of formidability that does not fluctuate significantly between observations. This suggestion is consistent with the findings from the bivariate twin model that shows that the key driver is genetic in nature. Finally, as also noted above, the bivariate twin model does not in itself imply the existence of a causal effect between aggression and formidability. A genetic correlation could emerge from the existence of common causes (e.g., related to androgens, see Lukaszewski & Roney, 2011) that influence both formidability and aggression. In this respect, however, it is important to combine the insights from the two key tests in the manuscript: (1) the cross-lagged model suggests that a correlation between aggression-formidability reflects an effect of formidability on aggression and (2), in light of this conclusion, the bivariate twin model suggest that this causal effect is grounded in genetic factors. Hence, the support for the reactive heritability account emerges from the combined evidence of the two tests.

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ONLINE APPENDIX

**Assessing Causal Pathways between
Physical Formidability and Aggression in Human Males**

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A1. Associations Between Measures of Formidability and Aggression Across Cohorts

Table 1 in the main text shows the correlations between the formidability index and aggression for the younger and older cohorts. In this part of the online appendix, we provide a more detailed view of the associations. Specifically, we estimate the associations for each of the components of the index for the younger cohort, the older cohort and the cohorts combined. Furthermore, rather than bivariate correlations, we use statistical models that corresponds to those utilized for estimating the cross-lagged models. Specifically, we report standardized coefficients (betas) estimated using structural equation modeling and report two-sided p-values estimated on the basis of cluster robust standard errors with twin pair ID as cluster variable (to correct for within-pair autocorrelation). Because the twins are surveyed within a relative wide age range (e.g., for measures taken around age 17, the youngest subject is 16 and the oldest is 20), we also control for respondents' ages. The associations are reported in Table A1.

Table A1. Associations between Measures of Formidability and Aggression for Different Cohorts and Ages.

		Formidability (age 17)				Formidability (age 24)			
		Handgrip	Bicep	Bulk	Index	Handgrip	Bicep	Bulk	Index
Older Cohort	Aggression (age 17)	$\beta=.11$; $p=.032$, $n=391$	$\beta=.12$; $p=.027$, $n=394$	$\beta=.09$; $p=.062$, $n=392$	$\beta=.13$; $p=.008$, $n=394$	$\beta=.08$; $p=.158$, $n=303$	$\beta=.16$; $p=.008$, $n=302$	$\beta=.06$; $p=.337$, $n=297$	$\beta=.13$; $p=.040$, $n=303$
	Aggression (age 24)	$\beta=.07$; $p=.211$, $n=391$	$\beta=.18$; $p=.004$, $n=394$	$\beta=.12$; $p=.113$, $n=392$	$\beta=.15$; $p=.018$, $n=394$	$\beta=.07$; $p=.368$; $n=303$	$\beta=.16$; $p=.034$, $n=302$	$\beta=.13$; $p=.099$, $n=297$	$\beta=.15$; $p=.062$, $n=303$
Younger Cohort	Aggression (age 17)	$\beta=-.017$; $p=.752$, $n=416$	$\beta=-.020$; $p=.703$, $n=414$	$\beta=-.066$; $p=.212$, $n=416$	$\beta=-.04$; $p=.473$, $n=420$	-	-	$\beta=.01$; $p=.799$, $n=459$	-
	Aggression (age 24)	$\beta=.08$; $p=.116$; $n=416$	$\beta=.003$; $p=.946$, $n=414$	$\beta=-.03$; $p=.598$, $n=416$	$\beta=.03$; $p=.632$, $n=420$	-	-	$\beta=.03$; $p=.658$, $n=459$	-
Cohorts Combined	Aggression (age 17)	$\beta=.07$; $p=.047$; $n=807$	$\beta=.04$; $p=.309$; $n=808$	$\beta=.002$; $p=.967$; $n=808$	$\beta=.04$; $p=.280$, $n=814$	-	-	$\beta=.03$; $p=.459$, $n=756$	-
	Aggression (age 24)	$\beta=.08$; $p=.045$; $n=807$	$\beta=.08$; $p=.046$; $n=808$	$\beta=.04$; $p=.425$; $n=808$	$\beta=.09$; $p=.045$, $n=814$	-	-	$\beta=.06$; $p=.175$, $n=756$	-

Notes. Entries are standardized coefficients (betas) estimated using structural equation modeling. Coefficients have been controlled for age when the relevant formidability measure was obtained. Reported p-values are two-sided p-values estimated on the basis of cluster robust standard errors with twin pair ID as cluster variable. Cells with bold entries include effects that are significant or marginally significant. Cells with bold borders include the correlations that will be used to assess causality.

A2. Cross-Lagged Effects in The Younger Cohort

Figure A1 replicates the analysis in Figure 1 for the younger cohort and for data that combines the two cohorts. Specifically, Figure A1, Panel A and Panel C, shows the lagged effect of formidability at age 17 on aggression at age 24 controlling for aggression at age. For the younger cohort (and, hence, for cohorts combined), formidability was not measured at age 24 and, hence, we cannot estimate the full cross-lagged effects. Consistent with the levels of association between formidability and aggression in the younger and combined cohorts (see Table A1), there is no evidence of a lagged effect of formidability on aggression in the younger cohort and only a smaller, marginally significant effect on the combined cohorts.

Figure A1. Cross-Lagged Effects of Formidability on Aggression and Aggression on Formidability

Panel A. Younger Cohort

Panel B. Cohorts Combined

Notes. N (Panel A) = 420; N (Panel B) = 814. Entries are standardized coefficients (betas) estimated using structural equation modeling. Coefficients have been controlled for age when ‘age 17’ measures were obtained. In Panel B effects on Aggression at age 24 are also controlled for cohort. Reported p-values are two-sided p-values estimated on the basis of cluster robust standard errors with twin pair ID as cluster variable.

A3. Supplemental Analyses for the Twin Analyses

Table A2 provides the descriptive statistics for the twins utilized in the univariate twin models. Table A3 provides the bivariate twin models for the younger cohort and the two cohorts combined. As there is no association to decompose for the younger cohort, the results returned from these bivariate models are inconclusive.

Table A2. Descriptive Statistics for Twins.

	N	Formidability (age 17)		Aggression (age 24)	
		Mean	Std. Dev	Mean	Std. Dev
Monozygotic (MZ) twins	553	-.01	.79	35.40	8.18
Dizygotic (DZ) twins	314	.07	.82	35.85	8.27

Notes. Table shows descriptives for twins from both the older and the younger cohort who have complete information on formidability measured at age 17 and aggression measured at age 24.

Table 3. Bivariate Cholesky Decomposition Model of Effect of Formidability (age 17) on Aggression (24).

	Younger Cohort	Cohorts Combined
r_A	0.05 (-0.53, 0.53)	0.21 (-0.06, 0.52)
r_C	-0.99 (-1.00, 1.00)	-1.00 (-1.00, 1.00)
r_E	0.10 (-0.07, 0.26)	0.05 (-0.07, 0.17)

Notes. N (Younger Cohort) = 133 MZ pairs and 72 DZ pairs; N (Cohorts Combined) = 253 MZ pairs and 137 DZ pairs. The entries the Additive Genetic (r_A), Common Environmental (r_C) and Unique Environmental (r_E) Correlation and 95% CIs (in parentheses) from a bivariate Cholesky model of formidability and aggression. For the analysis of the younger cohort, the traits are residualized on age. For the analysis of the combined cohorts, the traits are residualized on cohort and age.