# Individual differences in multiple dimensions of aggression: a univariate and multivariate genetic analysis

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Previous behaviour genetic studies of aggression have yielded inconsistent results: reported heritabilities for different types of aggressive behaviour ranging from 0 to 0.98. In the present study, 247 adult twin pairs (183 MZ pairs; 64 same-sex DZ pairs) were administered seven self-report questionnaires which yielded 18 measures of aggression. Univariate genetic analyses showed moderate to high heritabilities for 14 of these 18 measures and for a general aggression factor and three correlated aggression factors extracted from the measures. Multivariate genetic analyses showed sizeable genetic correlations between the different dimensions of aggression. Thus, individual differences in many types of aggressive behaviour are attributable to some extent to genetic factors and there is considerable overlap between the genes that operate on different types of aggressive behaviour.

Keywords: aggression, genetic analyses, twins, heritability, individual differences

## Introduction

Despite the fact that biological factors have been shown to play a role in aggressive behaviours both in animals<sup>1</sup> and in humans,<sup>2</sup> surprisingly few behaviour genetic studies of aggression in humans have been conducted. Moreover, the results of those studies that have investigated the possible genetic basis of individual differences among humans in aggression have been quite inconsistent.<sup>3</sup> The present study was designed in an attempt to clarify the contradictory findings of previous work in this area.

One area of contention in studies of aggression relates to the fact that, in addition to such types of aggressive behaviour as physical or verbal aggression, many other different types of behaviour have been proposed either as components, or at least correlates, of aggression. These include such behaviour as self-harm, hostility, anger, and impatience, which might reasonably be considered to be related to aggression, but have also included behaviour such as suspiciousness, nonconformity, and boldness, which are perhaps less obviously tied into a taxonomy of aggression. Thus, inconsistencies between studies that have each purportedly investigated 'aggression' may in part be attributable to the sometimes quite different behaviours that have actually been focused on or measured.

A related issue concerns differences in opinion regarding the dimensionality of aggression: some studies suggesting that aggression is a unitary trait, whilst others suggest that it is multidimensional.<sup>4,5</sup> A resolution of the dimensionality debate was recently provided by Choynowski<sup>6</sup> who, through a series of item and factor analyses, grouped an initial pool of over 900 aggression items into 13 scales which themselves factored into four correlated factors: Rebelliousness (comprising the nonconformity, verbal aggression, malice, and negativism scales), Spontaneous Aggressiveness (comprising physical aggression, boldness, and vicarious aggression), Intra-Aggressiveness (comprising self-aggression, resentment, and suspiciousness), and Irritable Aggressiveness (comprising irritability, lack of selfcontrol, and vengefulness). All 13 of Choynowski's scales are quite highly intercorrelated (multiple correlations between each scale and the other 12 scales range from 0.43 to 0.86), as are the four higher-order factors (which on average correlate about 0.50 with one another). As  $Eysenck^7$  points out, these intercorrelations indicate the presence of a still higher order general aggression factor. As in the realm of mental abilities, aggression may be viewed as having a 'g' or general component which itself, however, comprises a number of quite diverse (albeit types of aggressive correlated) aspects or behaviour.

Turning to behaviour genetic studies of aggression in humans,<sup>1,8-25</sup> most that we were able to locate focused on measures of physical aggression; others

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looked at verbal aggression, anger, irritability, hostility, and indirect aggression, or used a measure of overall aggression such as the aggression scale from Jackson's Personality Research Form.<sup>26</sup> Sample sizes ranged from 18 to over 700 pairs of monozygotic or dizygotic twins or adoptees, and the ages of subjects ranged from 4 years to adulthood. Across studies and measures, heritabilities of different types of aggression ranged from 0 to 0.98, with a median of 0.53. Generally, studies which looked at physical aggression, measured either through self-report or by direct observation, reported lower heritabilities than studies which looked at other dimensions of aggression.

Given the wide range of heritabilities reported for different types of aggressive behaviors, the fact that most previous studies focused on only one dimension of aggression (eg physical), and Choynowski's<sup>6</sup> demonstration that aggression is a multifaceted trait, it seemed it would be worthwhile to conduct a behaviour genetic investigation of aggression which employed multiple measures tapping many different dimensions of aggressive behaviour. Not only would this allow the heritability of a wide range of aggressive behaviours to be estimated in a single sample, it would also allow a genetic analysis of higher order factors of aggression derived from the multiple individual measures. Moreover, such a study would also allow multivariate genetic analyses to be performed in order to investigate the extent to which any phenotypic correlations between different types of aggression are themselves attributable to common genetic and/or environmental factors. To these ends the present study was conducted.

## Materials and methods

Subjects were 247 adult twin pairs: 183 pairs of monozygotic (MZ) twins (149 female twin pairs, mean age 45.1 years, s = 16.5; 34 male twin pairs, mean age 45.1 years, s = 15.8) and 64 same sex dizygotic (DZ) twin pairs (55 female twin pairs, mean age 42.8 years, s = 17.6; 9 male twin pairs, mean age 33.9 years, s = 8.9), recruited either from the University of British Columbia Twin Registry or from among participants in the Twinsburg, Ohio, annual Twins Day Festival: an event which each year attracts some 3000 pairs of twins from across North America and around the world. The subjects represent a wide variety of backgrounds and levels of education.

Subjects completed seven self-report questionnaires which each assessed from one to six dimensions of aggression: the *Aggression Questionnaire*  $(AQ^{27})$ , a 29-item scale tapping physical and verbal aggression, anger, and hostility; the *Aggression*  Inventory (AI<sup>28</sup>), a 22-item scale tapping physical and verbal aggression, impulsivity, and impatience; the Personality Assessment Inventory  $(PAI^{29})$ , a 38-item scale tapping physical and verbal aggression, affective instability, anti-social behaviour, selfharm, and aggressive attitudes; the aggression scale from the Multidimensional Personality Questionnaire (MPQ), a 20-item scale tapping overall aggression (Tellegen A, 1982, unpublished ms); the aggression scale from the Personality Research Form-E (PRF<sup>26</sup>), a 16-item scale tapping overall aggression; and two Adjective Check Lists (ACL1 and ACL2)<sup>30</sup>, each of which contained a total of 300 descriptive adjectives, 44 of which pertained to aggressive behaviour and provided a measure of overall aggression. In total, 18 measures of aggression were obtained, the reliabilities of which range from moderate (PAI verbal aggression: alpha = 0.60) to high (ACL2: alpha = 0.88); median alpha = 0.75. Subjects also completed a zygosity questionnaire,<sup>31</sup> which has a reported accuracy of 93% compared with the results of blood typing.<sup>32</sup>

Items from the first five scales listed above were responded to on a 5-point Likert scale. In one form of the *Adjective Check List* (ACL1), subjects simply ticked any adjective they felt was at all descriptive of them. In the second *Adjective Check List* (ACL2), subjects rated the degree to which they felt each adjective was descriptive of them on a 5-point Likert scale. It was hoped (and subsequently confirmed) that the latter version would yield a more reliable measure.

Subjects were mailed the questionnaires along with instructions for their completion and the promise of a chance to win one of 10 cash prizes of \$100.00 in return for completing the questionnaires. Subjects completed the questionnaires at home and returned them in stamped, pre-addressed envelopes. On completion of the study, subjects were sent a copy of the results and an inscribed pen. Subsequently, 10 subjects were randomly selected from among those who had returned their completed questionnaires and these subjects were sent the prize money.

## Results

Consistent with what would be expected from a nonclinical sample, mean scores on the 18 measures of aggression indicated low to moderate average levels of aggression (mean scores on the 5-point scales ranging from 1.38 to 2.43: higher values denoting more aggression), but with scores ranging from 1 to 5 on every measure (and standard deviations ranging from 0.49 to 0.80), indicating sizeable

variability or individual differences within the sample. There were no significant mean score differences between MZ and DZ twins on any of the measures.

Using LISREL 8,<sup>33</sup> univariate genetic analyses were conducted to assess the relative contributions of genetic and environmental effects to individual differences on the 18 aggression measures, after separating out age and sex. For each measure, a full A (additive genetic), C (common environment), and E (nonshared environment) model was fit to the data, followed by reduced models (AE, CE, and E only) which systematically removed one source of variance. The model whose goodness-of-fit was not significantly poorer than that of the full ACE model, as evaluated by a comparison of chi squares, and which yielded the best goodness-of-fit, was selected as the 'best' model. When MZ twin correlations were more than twice as large as DZ correlations, the presence of nonadditive (dominance) genetic effects (D) was also investigated with an ADE model.

Table 1 shows the MZ and DZ correlations on the 18 aggression measures and estimates from the bestfitting models of the contributions of additive genetic effects ( $a^2$ ), dominance genetic effects ( $d^2$ ), common environmental effects ( $c^2$ ), and non-shared environmental effects ( $e^2$ ) to individual differences on the measures. MZ correlations are equal to or larger than DZ correlations for all measures and 14 of the 18 measures show significant heritabilities ( $a^2$ and/or  $d^2$ ). For these 14 measures, heritabilities range from 0.26 to 0.56 (median = 0.38); dominance genetic effects are evident for five of these measures. Across all 18 measures, common environmental effects (c<sup>2</sup>) are present for only four measures: the largest source of environmental variance for all measures is non-shared ( $e^2$  ranging from 0.44 to 0.74, median = 0.63).

To investigate the factorial structure underlying the measures, intercorrelations among the 18 aggression measures were subjected to principal components analysis with oblimin rotation. This analysis yielded a strong first unrotated or general aggression factor, accounting for 48% of the variance, on which all of the measures loaded highly and positively (see Table 2). The analysis also yielded three correlated factors, accounting for a total of 65% of the variance, with loadings (> 0.25) as shown in Table 2. The first of these three correlated factors is similar to Choynowski's Spontaneous Aggression factor: receiving high loadings from measures of physical aggression and also including some verbal aggression measures and a measure of antisocial behaviour. The second correlated factor receives its highest loadings from measures of impulsivity, affective instability, hostility, and anger: it is interpreted as representing Aggressive Attitudes. The third correlated factor receives loadings primarily from the two ACL measures and lower loadings from all three of the verbal aggression measures and from the Jackson PRF aggression scale. It is interpreted as a verbal aggression factor.

MZ and DZ twin correlations and the results of univariate genetic analyses conducted on factor scores from the general aggression factor and the three correlated aggression factors are presented in Table 3. At this more reliable level of analysis, MZ correlations are greater than DZ correlations for all

**Table 1**MZ and DZ correlations and genetic analyses<sup>a</sup> of 18 aggression measures

Variables <sup>b</sup>	MZ	DZ	$a^2$	$d^2$	$C^2$	$e^2$
AQ-PHYS	0.50	0.02		0.49		0.51
AQ-VERB	0.43	0.41			0.42	0.58
AQ-HOS	0.33	0.24	0.36			0.64
AQ-ANG	0.35	0.16	0.36			0.64
AI-PHYS	0.41	0.05		0.39		0.61
AI-IMPUL	0.26	0.14	0.26			0.74
AI-VERB	0.44	0.14	0.42			0.58
AI-IMPAT	0.33	0.19	0.32			0.68
PAI-VERB	0.35	0.22	0.36			0.64
PAI-AFFINS	0.46	0.09		0.47		0.53
PAI-SHARM	0.36	0.29			0.34	0.66
PAI-ANBEH	0.53	0.14		0.56		0.44
PAI-PHYS	0.25	0.14	0.27			0.73
PAI-AGATT	0.46	0.44			0.45	0.55
MPQ-AGG	0.42	0.28	0.42			0.58
PRF-AGG	0.34	0.05		0.34		0.66
ACL1-AGG	0.39	0.31	0.42			0.58
ACL2-AGG	0.35	0.26			0.33	0.67

<sup>a</sup>All reported genetic and environmental effects are significant, with standard errors ranging from 0.03 to 0.07.

<sup>b</sup>Variable names, in the order they appear are: Aggression Questionnaire: Physical, Verbal, Hostility, Anger; Aggression Inventory: Physical, Impulsivity, Verbal, Impatience; Personality Assessment Inventory: Verbal, Affective Instability, Self Harm, Antisocial Behaviour, Physical, Aggressive Attitudes, and the aggression scales from the Multidimensional Personality Questionnaire, the Personality Research Form, and two Adjective Check Lists.

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Table 2Loadings of 18 aggression measures on the firstunrotated and 3 oblique rotated factors

	Oblique rotated factors First				
Variables <sup>a</sup>	unrotated	First	Second	Third	
AQ-PHYS	0.77	0.83			
AQ-VERB	0.68	0.45		0.48	
AQ-HOS	0.62		0.77		
AQ-ANG	0.79		0.72	0.34	
AI-PHYS	0.78	0.88			
AI-IMPUL	0.57		0.81		
AI-VERB	0.68	0.73		0.26	
AI-IMPAT	0.65		0.53	0.40	
PAI-VERB	0.56	0.51		0.36	
PAI-AFFINS	0.77		0.78		
PAI-SELFHARM	0.63		0.64		
PAI-ANTBEH	0.68	0.62			
PAI-PHYS	0.76	0.49	0.45		
PAI-AGGATT	0.77		0.62		
MPQ-AGG	0.75	0.84			
PRF-AGG	0.74	0.37		0.46	
ACL1-AGG	0.56			0.77	
ACL2-AGG	0.67			0.77	

<sup>a</sup>Variable names, in the order they appear are: Aggression Questionnaire: Physical, Verbal, Hostility, Anger; Aggression Inventory: Physical, Impulsivity, Verbal, Impatience; Personality Assessment Inventory: Verbal, Affective Instability, Self Harm, Antisocial Behaviour, Physical, Aggressive Attitudes, and the aggression scales from the Multidimensional Personality Questionnaire, the Personality Research Form, and two Adjective Check Lists.

factors, and in each case an AE (or ADE) model provides the best fit to the data. Heritabilities for the factors range from 0.44 to 0.54 and non-shared environmental effects account for 46–56% of the variance. None of the factors shows any influence of shared environmental effects.

Phenotypic correlations between the correlated factors are presented in Table 4, where it can be seen

Table 3  $\,$  MZ and DZ correlations and genetic analyses  $^{\rm a}$  of aggression factors

<i>Factors</i> <sup>b</sup>	MZ	DZ	$a^2$	$d^2$	$C^2$	$e^2$
GEN FAC	0.54	0.25	0.54			0.46
ROTFAC1 ROTFAC2	$0.50 \\ 0.53$	$0.26 \\ 0.08$	0.52	0.52		$\begin{array}{c} 0.48 \\ 0.48 \end{array}$
ROTFAC3	0.44	0.31	0.44			0.56

<sup>a</sup>All reported genetic and environmental effects are significant, with standard errors ranging from 0.04 to 0.06.

<sup>b</sup>Factors, in the order they appear, are the first unrotated general aggression factor, followed by the three correlated oblimin rotated factors: spontaneous aggression, aggressive attitudes, and verbal aggression, respectively.

	ROTFAC1	ROTFAC2	ROTFAC3
ROTFAC1	1.0	0.44	0.26
ROTFAC2	0.50	1.0	0.40
ROTFAC3	0.31	0.51	1.0

<sup>a</sup>Phenotypic correlations appear above the main diagonal; genetic correlations appear below the main diagonal.

that all three factors are moderately highly intercorrelated (average r = 0.37), a not surprising finding in light of the large first unrotated or general factor that was extracted earlier. Table 4 also presents the results of multivariate genetic analyses which were conducted to estimate the genetic correlations between the factors. In these analyses, Cholesky or triangular decomposition<sup>34</sup> was applied to MZ and DZ mean square between and within pair covariance matrices to calculate the genetic correlations, which estimate the extent to which phenotypic correlations between variables are attributable to common genetic influences. As can be seen in Table 4, all the genetic correlations are moderately large (average r = 0.44), indicating considerable genetic overlap between these factors of aggression.

## Discussion

Univariate and multivariate genetic analyses revealed that multiple measures of different dimensions of aggressive behaviour have moderate to quite large heritabilities. Unlike previous behaviour genetic studies of aggression, measures of physical aggression in the present study did not show lower heritabilities than other types of aggression. In fact, some of our measures of physical aggression were among the more highly heritable, as might be expected from an evolutionary perspective.

Factor analysis of 18 measures of aggression yielded similar results to those reported by Choynowski:<sup>6</sup> a strong general factor comprised of a number of (in our case, three) moderately highly correlated group factors. Univariate genetic analyses revealed that all the aggression factors were heritable and multivariate analyses showed that there were moderately large genetic correlations between the factors. These results indicate both that individual differences in aggression are attributable to some extent to genetic factors and that there is considerable overlap between the genes that operate on different types of aggressive behaviour.

Positive features of the present study are its inclusion of a large number of measures tapping a variety of different types of aggression. This in turn allowed genetic analyses to be performed at two levels: on the measures themselves and on the more stable higher order factors. To our knowledge, ours is the first behaviour genetic study of aggression which has administered as many different measures to the same groups of subjects or which, as a result, has been able to investigate the genetic basis of phenotypic correlations among measures.

Limitations of our study include the fact that the majority of our sample was female and that we had many more MZ than DZ pairs. Not only do these Genetic analysis of aggression PA Vernon et al

considerations, and the absence of any opposite-sex DZ pairs, limit somewhat the generalisability of our findings but they prevented us from performing analyses separately among males and females. The questions as to whether different patterns of heritabilities for different types of aggression would be found in males and females, or whether there is any nonscalar sex limitation (ie whether the same genes for aggression operate in males and females), would be interesting and potentially important for future studies to address; from an evolutionary point of view it is not hard to imagine that the sexes may have been subject to different degrees of pressure to develop and to maintain different levels of aggressive behaviour.

These limitations aside, the present study has clearly demonstrated that individual differences in many manifestations of aggressive behaviour have a sizeable genetic component. This is consistent with the results of studies that have identified biological factors underlying aggression: for example, Brunner and colleagues<sup>35,36</sup> recently reported an association between monoamine oxidase A (MAO-A) deficiency and aggressive behaviour (and borderline mental retardation) in several males from a single large Dutch kindred. Moreover, the present study demonstrated that those genes that contribute to higher or lower levels of one type of aggression also contribute to individual differences in other types of aggressive behaviour: also consistent with Brunner's report<sup>35</sup> that MAO-A deficient males showed several types of abnormally aggressive behaviour, including increased impulsive behaviours, abnormal sexual behaviour, and arson. High heritability, of course, does not preclude the role of environmental factors in the development of aggressive behaviour. However, it is important to note that common or shared environmental effects were present for only a few of the aggression measures and for none of the aggression factors. Thus, those environmental factors that contribute to individual differences in aggression are largely non-shared and within family and not such between family factors as SES which affect all siblings in a family to the same degree.

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