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Etiology of Triarchic Psychopathy Dimensions in Chimpanzees (*Pan troglodytes*)

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Abstract

The current study undertook analyses of genealogical data from a sample of 178 socially housed chimpanzees (*Pan troglodytes*) with well-documented pedigrees to clarify the etiologic bases of triarchic psychopathy dimensions and the influence of early social rearing experiences. Whereas biometric analyses for the full sample indicated significant heritability for the boldness dimension of psychopathy only, heritability estimates varied by early rearing, with all three triarchic dimensions showing significant heritabilities among mother-reared but not nursery-reared apes. For mother-reared apes, both genes and environment contributed to covariance between meanness and disinhibition, whereas environment contributed mainly to covariation between these dimensions and boldness. Results indicate contributions of both genes and environment to psychopathic tendencies, with an important role for early rearing in their relative contributions to distinct phenotypic subdimensions. In conjunction with findings from human studies, results provide valuable insights into core biobehavioral processes relevant to psychological illness and health.

Keywords

psychopathic personality, heritability, early rearing, chimpanzees, nonhuman primate models

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Psychopathy is a form of personality pathology that entails severe disturbances in behavioral control, social relations, and emotional experiences (Cleckley, 1941) and that appears to have clear genetic foundations (e.g., Blonigen, Carlson, Krueger, & Patrick, 2003; Larsson, Andershed, & Lichtenstein, 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003; Tuvblad, Wang, Bezdjian, Raine, & Baker, 2016). Although historically studied largely in criminal populations, a growing literature indicates that psychopathy represents a multidimensional construct grounded in basic biobehavioral dispositions that vary continuously within the neurotypical human population (Lilienfeld, Watts, Smith, Berg, & Latzman, in press; Patrick, Fowles, & Krueger, 2009) and, as such, differs from normality in degree, rather than in kind (Edens, Marcus, Lilienfeld, & Poythress, 2006; Walters, Marcus, Edens, Knight, & Sanford, 2011). Viewed in this way, understanding of psychopathy can be advanced through study of these basic dispositional dimensions in a range of populations, including both clinical and nonclinical samples (Hall & Benning, 2006; Lilienfeld, 1994; Salekin, 2006).

Recently, investigation of these dispositional dimensions of psychopathy has been extended to our closest living relatives, chimpanzees (Latzman, Drislane, et al., 2016), providing a basis for comparative research on their behavioral and neurobiological aspects. The current

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study was undertaken to further clarify the etiologic bases of psychopathic personality dimensions by examining, for the first time in a nonhuman primate sample, the contribution of genetic influences to individual variation in differing psychopathy subdimensions, along with that of early rearing experiences—both alone and in interaction with genetic influences.

Recent theoretical and empirical work has sought to more accurately capture the dimensions of psychopathy through clearer delineation of its component dispositional or trait dimensions (e.g., Lilienfeld & Widows, 2005; Marcus, Fulton, & Edens, 2011; Patrick et al., 2009; Poythress & Hall, 2011). One prominent conceptualization, the triarchic model (Patrick et al., 2009; Patrick & Drislane, 2015), characterizes the symptomatic components of psychopathy in terms of three biobehavioral trait constructs: boldness, meanness, and disinhibition. Disinhibition and meanness (callous-aggression) correspond to impulsive conduct problem and callous-unemotional symptom dimensions in the youth psychopathy literature (Frick, Ray, Thornton, & Kahn, 2014) and disinhibitory and callous-aggressive dimensions in the adult literature on externalizing disorders (Krueger, Markon, Patrick, Benning, & Kramer, 2007). The third construct of the triarchic model, boldness, encompasses characteristics with adaptive as well as maladaptive qualities (i.e., social dominance, stress immunity, venturesomeness) that can be viewed, in turn, as facets of dispositional fear/fearlessness (Kramer, Patrick, Krueger, & Gasperi, 2012; Patrick & Drislane, 2015).

Extending this human research on the triarchic model, Latzman, Drislane, et al. (2016) developed a chimpanzee operationalization of psychopathic personality organized around the triarchic conceptualization. Specifically, drawing on caretaker-rated items from an existing primate personality instrument, Latzman et al. used a three-stage consensus rating approach to formulate scale measures of the three triarchic model constructs for use with chimpanzees. These Chimpanzee Triarchic (CHMP-Tri) scales were then validated both with regard to their translational relevance to humans and their associations with performance on behavioral tasks. Importantly, scales indexing boldness and disinhibition showed expected differential associations with task-performance measures of impulsive and venturesome tendencies, respectively—indicating convergence with findings from the human literature (Patrick & Drislane, 2015).

Results from this work indicate that the triarchic model of psychopathy can be operationalized effectively in chimpanzees, an animal species uniquely well-suited for neurobiological investigations of individual variation in broad, transdiagnostic traits (Latzman, Young, & Hopkins, 2016). The current study further extends this literature by examining the interface of this nonhuman animal translation of the model of psychopathy with human findings by evaluating, in a sample of chimpanzee subjects, genetic and environmental contributions to variability in scores on the triarchic model dimensions as indexed by the CHMP-Tri scale measures.

The translational value of any animal model lies in the ability of findings for behavioral phenomena of interest to generalize between nonhuman animals and humans in ways that reflect basic processes in common. Given evidence that humans and chimpanzees share many affective–motivational processes in common (Phillips et al., 2014), this ape species provides a unique animal model for investigating genetic and environmental influences (particularly earlier environmental experiences) contributing to behavioral proclivities (Nelson & Winslow, 2009), including psychopathic traits. Whereas in humans sociocultural systems likely contribute importantly along with biological factors to variability on these dimensions (Farrington, 2006; Lykken, 1995), by imposing expectations on how humans should behave and react in various social situations beginning early in life, systematic social and cultural pressures of these types are largely absent in chimpanzees. Consequently, interindividual variation in psychopathic traits among individuals of this species can be presumed to reflect biological mechanisms more prominently (Latzman, Young, & Hopkins, 2016).

Appreciable heritabilities have been reported for psychopathic tendencies across a range of assessment methods and at differing points in the lifespan (e.g., Bezdjian, Tuvblad, Raine, & Baker, 2011; Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005, 2006; Brook et al., 2010; Tuvblad et al., 2016; Viding, Blair, Moffitt, & Plomin, 2005). Blonigen et al. (2005) estimated scores on component dimensions of psychopathy using subscales of the Multidimensional Personality Questionnaire (MPQ; Tellegen & Waller, 2008) in a young mixed-gender twin sample and found that genetic factors accounted for 45% of the variance in fearless dominance, a close counterpart to boldness (Patrick et al., 2009), and 49% of the variance in impulsive antisociality, a counterpart to the triarchic construct of disinhibition (Drislane, Patrick, & Arsal, 2014). A subsequent study by Brook et al. (2010) reported a somewhat higher heritability estimate for MPQ-estimated fearless dominance (.51) and a somewhat lower estimate for impulsive antisociality (.32). Although neither of these studies examined the triarchic construct of meanness, an investigation focusing on the etiology of callous–unemotional traits (Viding et al., 2005), a symptom dimension related to meanness, reported a heritability estimate of 67% among twins assessed in childhood.

Taken together, available research indicates a prominent contribution of genes to psychopathic tendencies, with differential heritabilities evident for differing symptom dimensions (facets). At the same time, the corollary

finding of a sizable portion of variance attributable to nonheritable factors raises the important question of what else may be contributing to variations in psychopathy symptoms, both independently of and in concert with genetic influences (e.g., through Gene × Environment [G×E] interactions).

The existing human research literature strongly supports a contribution of genetic as well as environmental influences to interindividual variability in psychopathic tendencies. Indeed, recent meta-analytic results of virtually all twin studies published in the past 50 years provide unquestionable evidence that *all* human traits are heritable (mean $h^2 = .49$) (Polderman et al., 2015). Similar findings have emerged among chimpanzees with a relatively wide range of traits appearing to have significant genetic contributions including, among other traits, tool use skills (Hopkins, Reamer, et al., 2014), handedness (Hopkins, Adams, & Weiss, 2013), and general intelligence (Hopkins, Russell, & Schaeffer, 2014). Further, a growing literature provides strong evidence for the translational value of broad chimpanzee personality dimensions to humans (Latzman, Sauvigne, & Hopkins, 2016) and the heritable nature of these dimensions (Latzman, Freeman, Schapiro, & Hopkins, 2015). Taken together, this work provides a strong basis for expecting partial heritability of dispositional dimensions corresponding to the triarchic model dimensions in chimpanzees.

However, it seems likely that genetic and environmental influences contribute not only individually but also interactively to variance in psychopathic symptomatology. Indeed, in both human and nonhuman animals, the heritability of particular traits likely depends on distinct factors in the environment, resulting in the relevance of genes in some environments but not in others (Charmantier & Garant, 2005; Rutter, Moffitt, & Caspi, 2006). That is, the genetic contributions to various outcomes may differ depending on the environment. In humans, evidence has been presented for an interactive contribution of environmental adversity and genetic variation to a broad range of psychopathological and related outcomes (Moffitt, Caspi, & Rutter, 2006). For example, Miles, Silberg, Pickens, and Eaves (2005) reported that genetic effects on adolescent alcohol use varied according to the quality of parental relationships. Similarly, Krueger, South, Johnson, and Iacono (2008) found that genetic contributions to individual variation in emotionality was moderated by the quality of adolescents' relationships with their parents. Further, research in nonhuman animals (i.e., Charmantier & Garant, 2005), including chimpanzees (Latzman et al., 2015), has revealed similar variability in heritability estimates as a function of familial environmental differences. Among chimpanzees, Latzman et al. (2015) found strong evidence that the heritability of personality characteristics varied by early social rearing experiences (human- versus mother-reared), as evidenced by a significant G×E interaction for personality traits as rated by human caretakers. Specifically, Latzman et al. found affective dimensions of personality (i.e., negative emotionality, positive emotionality) to be significantly heritable among mother-, but not nursery-reared, apes. This work, together with human research findings, indicates that consideration of the way in which heritabilities vary across environments is critical. As described below, differing members of the chimpanzee colonies used in the current study were exposed to contrasting early rearing experiences, resulting in a unique opportunity to evaluate the effects of differences in early rearing on variability in psychopathy dimensions.

Focusing on a relatively large sample of socially housed captive chimpanzees and using an unparalleled animal model, the current study sought to investigate genetic and environmental contributions to psychopathy dimensions. Given evidence of a genetic foundation for a higher order psychopathic personality factor (i.e., Larsson et al., 2006), genetic correlations among individual psychopathy subdimensions were examined, as was the heritability of a single extracted psychopathy factor. In addition, a distinct focus of the study was on the role of a major environmental variable, differential early rearing (i.e., mother- versus nonmother nursery rearing), on the contribution of genetic influences to measured psychopathic tendencies.

Although the current investigation is the first to examine the heritability of psychopathy subdimensions in nonhuman primates, considerable evidence exists for translational correspondence of basic personality traits between chimpanzees and humans (Latzman et al., 2015; Latzman, Sauvigne, & Hopkins, 2016), and thus we predicted on the basis of existing human work (e.g., Blonigen et al., 2003; Larsson et al., 2006; Taylor et al., 2003; Tuvblab et al., 2016) that the three psychopathy subdimensions would each show a genetic contribution. Further, within the human literature, genetic influences have been shown to vary as a function of environmental enrichment versus impoverishment. For example, the heritability of intelligence has been found to vary by the socioeconomic status (SES) of the home within which children grew up, with high heritability estimates evident for individuals from affluent families, and estimates approaching zero for those from impoverished families (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). By contrast, Hicks, South, DiRago, Iacono, and McGue (2009) reported evidence for increased heritability of tendencies toward externalizing problems in individuals exposed to more adverse rearing environments. The implication is that adverse early rearing may have opposing effects on heritabilities for adaptive versus maladaptive outcomes (e.g., intellectual ability versus disinhibitory psychopathology). Based on findings of these

types from human studies, we hypothesized that genetic contributions to individual variability in triarchic dimension scores would differ as a function of early social rearing experiences in chimpanzees. More specifically, based on effects reported by Latzman et al. (2015) for broad personality dimensions in chimpanzees, we predicted that heritabilities of psychopathy dimensions would be lower among nursery-reared as compared to mother-reared apes. A final study prediction, based on the concept of disinhibition and meanness as subdimensions of a broad externalizing spectrum (Krueger et al., 2007; Patrick et al., 2009) along with heritability findings for psychopathy as a whole in humans (Larsson et al., 2006), was that these two triarchic dimensions would load together more than boldness on a strongly heritable general psychopathy factor.

Method

Subjects

Chimpanzees were members of two colonies of apes housed at the Yerkes National Primate Research Center (YNPRC) in Atlanta, Georgia, and The University of Texas MD Anderson Cancer Center (UTMDACC) in Bastrop, Texas. As described in previous work utilizing this chimpanzee sample (Latzman et al., 2016), personality ratings were available for 95 adult and subadult chimpanzees at YNPRC, including 68 females and 27 males ranging in age from 9 to 53 years (M_{age} = 24.79, SD = 10.90). Ratings were available for 143 adult and subadult chimpanzees at UTMDACC, including 74 females and 69 males ranging in age from 8 to 51 years (M_{age} = 28.58, SD = 10.60). All apes in both colonies were well and not undergoing any medical experiments. All subjects were combined into a single sample for analyses, resulting in a final sample of 238 chimpanzees.

Early rearing experiences varied among individuals in this final sample, with 119 being mother-reared, 59 human nursery-reared, and 60 wild-born. For the purposes of the current study and consistent with previous research (i.e., Bogart, Bennett, Schapiro, Reamer, & Hopkins, 2014; Latzman et al., 2015), wild-born animals were excluded from the current analyses because of their restricted age-range (i.e., all were appreciably older than other subjects, by more than 17 years on average) and because information was lacking regarding their relatedness—resulting in a final sample of 178 apes for all analyses.

Nursery-reared chimpanzees were separated from their mothers within the first 30 days of life due to unresponsive care, injury, or illness. Although there was little variability during this period, information concerning details of any variability (e.g., time spent with their mother prior to removal; mother–offspring interactions) was unfortunately not available for use in the current study. These chimpanzees were placed in incubators, fed standard human infant formula, and cared for by humans until they could care adequately for themselves, at which time they were placed with other infants of the same age until they were 3 years old (Bard, 1994; Bard, Platzman, Lester, & Suomi, 1992). At 3 years of age, the nursery-reared chimpanzees were integrated into larger social groups of adult and subadult chimpanzees. During this time, they remained with their peer group 24 hours per day, 7 days per week. Mother-reared chimpanzees remained under the care of their mothers for at least 2.5 years of life and were raised in "nuclear" family groups of chimpanzees, with group sizes ranging from 4 to 20 individuals. It should be noted that all of the nursery-reared chimpanzees were raised in this manner because their biological mothers did not exhibit adequate maternal care at birth and thus required intervention in order to protect the infants' well-being. That is, the chimpanzees in this study were not nursery-reared by design, with the goal of subsequently determining the effects of early life experiences on development. The data for these subjects are therefore *ex post facto* and opportunistic; indeed, we capitalized on the fact that some of the chimpanzees received different rearing experiences in order to evaluate whether this might have long-term consequences on personality development. Importantly, as described previously (Bogart et al., 2014), based on the composition of the rearing groups, potential rearing differences are likely not conflated with familial environment. Specifically, the genetic diversity within each group was comparable. With regard to relatedness within each rearing group, 52 different sires and 79 different dams contributed to the mother-reared group, and 34 different sires and 42 different dams contributed to the nursery-reared group. Within each group, 21 and 7 parent–child dyads and 23 and 8 full siblings were represented in the mother- and nursery-reared groups, respectively. Importantly, though some variability in relatedness structure appears to be present when these basic descriptors are considered, when accounting for colony (YNPRC versus UTMDACC), relatedness coefficients did not differ between the two rearing groups (F = .82, p > .35). This suggests that (a) group membership reflects early experiences rather than familial aggregation of group placement decisions and (b) variance decomposition analyses are similarly meaningful in each group.

All aspects of the research complied with the American Psychological Association's *Guidelines for Ethical Conduct in the Care and Use of Nonhuman Animals in Research* (American Psychological Association [APA], 2012), followed the Institute of Medicine (2011) guidelines for research with chimpanzees, and was done with

the approval of the Institutional Animal Care and Use Committees of the universities at which the research was conducted. All chimpanzees are housed in social groups ranging from 2 to 16 individuals in indoor–outdoor compounds, with free access to both portions of their enclosures 24 hours a day. During the winter seasons, the indoor facilities are heated, whereas air conditioning or fans and misters are provided in the hotter summer months. Lighting in the outdoor facility follows the typical seasonal cyclic change in sunrise and sunset. Standard tungsten lighting is provided in the indoor facility, and the lights are on a 12-hour on–off cycle. The chimpanzees are fed two to five times per day with a diet that consists of fruits, vegetables, and commercially produced primate chow. In addition, they receive a number of foraging and enrichment opportunities each day. Environmental enrichment, such as simulated tool use tasks or nonnutritive substrates, is provided to the chimpanzees on a daily basis. At no time are the subjects ever food- or water-deprived.

Assessment of triarchic psychopathy dimensions

CHMP-Tri scales previously developed through a consensus-based approach (Lazman, Drislane, et al., 2016) were used in the current study. Consistent with the triarchic model of psychopathy, the three CHMP-Tri scales assess Boldness (six items), Meanness (five items), and Disinhibition (seven items). As described by Latzman et al. (2016), chimpanzees were rated by colony-staff members who had worked with the animals for an extended period of time and reported having "enough experience for an accurate rating" (Freeman et al., 2013, p. 1044). Items for each scale were rated using a 7-point Likert-type format, with response options ranging from 1 (*least descriptive of the chimpanzee*) to 7 (*most descriptive of the chimpanzee*). With the exception of one of the YNPRC animals, two to three independent raters rated each chimpanzee, and ratings were averaged for all analyses; mean interrater reliability using ICC (3,k) across all items included in the CHMP-Tri scales was .63 and .65 for the YNPRC and UTMDACC colonies, respectively. Further, as reported by Latzman et al. (2016), internal consistencies (Cronbach's alpha) for the three scales were acceptable, especially considering their brevity: .77 for Boldness, .67 for Meanness, and .82 for Disinhibition.

Data analysis

After first conducting preliminary analyses of associations for scores on the triarchic psychopathy dimensions with age and sex, additive genetic contributions to each dimension were examined. Many of the chimpanzees in each colony are related, allowing for an analysis of heritability using quantitative genetics as applied to the pedigree hierarchy as a whole. To estimate heritability of the three psychopathy subdimensions, we used Sequential Oligogenic Linkage Analysis Routines (SOLAR; Almasy & Blangero, 1998). SOLAR uses a variance-components approach that relies on maximum likelihood estimation to compute a polygenic variance term for a dependent measure of interest when considering the entire pedigree (see Fears et al., 2009; Fears et al., 2011). We evaluated whether to include potential covariates (i.e., colony, age, sex, Age × Sex) in each model by testing the statistical significance of their associations with the three psychopathy dimensions, using the software's default probability criterion of 0.1. Covariates likely to be influential based on this criterion were retained in the final model. The SOLAR routine generates a total additive genetic variance (h^2) term, reflecting the proportion of total phenotypic variance that is attributable to all genetic sources. Total phenotypic variance is constrained to a value of 1; therefore, all nongenetic contributions to the phenotype are computed as $1 - h^2$. Further, in line with previous nonhuman primate research (e.g., Fairbanks, Bailey et al., 2011; Fairbanks, Jorgensen et al., 2011; Hopkins, Reamer et al., 2014), we quantified the effects of shared environments (c^2) by incorporating a matrix identifying individuals that were raised by the same mother. This creates a parameter corresponding to the fraction of the variance associated with the effect of a common maternal environment.

We next examined the phenotypic effects of early rearing experiences on the triarchic psychopathy dimensions through a series of Multivariate Analysis of Covariance Analyses (MANCOVAs). Specifically, while controlling statistically for sex and age, scores for each of the three CHMP-Tri scales were included as dependent variables with a dichotomous between-subjects factor of mother- versus nursery-rearing serving as the independent variable.

Finally, given data from both human (e.g., Moffitt et al., 2006; Rutter, 2005) and nonhuman (e.g., Charmantier & Garant, 2005) studies pointing to the importance of G×E interactions across a broad range of outcomes, including psychopathy-related tendencies (i.e., antisocial behavior; Jaffee et al., 2005), as well as prior work with chimpanzees demonstrating the importance of early rearing experiences in influencing the heritability of personality broadly (i.e., Latzman et al., 2015), analyses were conducted to separately estimate heritable influences for mother- and nursery-reared chimpanzees. Given consistent evidence for robust phenotypic correlations among distinguishable dimensions (facets) of psychopathy in both chimpanzees (Latzman, Drislane, et al., 2016) and humans (Hare, 2003; Skeem, Polaschek, Patrick, &

Table 1. CHMP-Tri Dimensions by Early Rearing Experiences

CHMP-Tri Dimension	Mother-Reared *M* (*SD*)	Nursery-Reared *M* (*SD*)	*F*	*p*	Partial eta^2
Boldness	3.99 (.08)	4.15 (.11)	1.33	.25	.01
Meanness	4.07 (.07)	3.98 (.10)	.67	.41	.00
Disinhibition	3.90 (.06)	4.15 (.08)	**6.33**	**.01**	**.04**

Note: Mother-reared $N = 119$; nursery-reared $N = 59$. As described in the text, all analytic models control for age and sex. *F* values shown in boldface are significant at $p < .05$.

Lilienfeld, 2011) and evidence for distinct etiological connections between particular triarchic dimensions and broad domains of psychopathology (i.e., Blonigen et al., 2005), we next evaluated genetic correlations (ρ_G) among CHMP-Tri dimensions found to be heritable. Specifically, we used SOLAR to quantify the degree of shared genetic variance by decomposing the covariance between CHMP-Tri dimensions into genetic and environmental factors and took the former as an estimate of the proportion of variability due to shared genetic effects (i.e., ρ_G). Finally, given previous evidence of significant genetic contributions to a general, higher order psychopathy dimension (Larsson et al., 2006), we subjected the three psychopathy subdimensions to a principal axis factor analysis and extracted a single common factor, saving out regression-estimated scores on this general factor. We then ran heritability analyses on this factor score.

Results

Preliminary analyses

Within the combined sample ($N = 178$), scores on the three CHMP-Tri scales were intercorrelated as follows: Boldness With Meanness, $r = .36$; Boldness With Disinhibition, $r = .25$; and Meanness With Disinhibition, $r = .46$ (all ps $< .01$). Age was correlated to a significant negative degree with Disinhibition ($r = -.31$, $p < .001$) but was unrelated to either Boldness ($r = -.09$, $p > .20$) or Meanness ($r = .12$, $p > .10$). Further, whereas sex was not associated with Boldness ($r = -.02$, $p > .80$), males evidenced slightly, albeit significantly, higher scores for both Meanness ($r = -.18$, $p < .05$) and Disinhibition ($r = -.15$, $p < .05$). As shown in Table 1, scores on the CHMP-Tri Disinhibition scale also differed as a function of early rearing background, $F = 6.33$, $p < .01$, $\eta^2 = .04$, with nursery-reared apes evidencing higher mean levels of disinhibitory tendencies than mother-reared apes. By contrast, neither Boldness nor Meanness scores differed as a function of early rearing (see Table 1).

Tests for potential covariates (colony, age, sex, Age × Sex) revealed no significant associations with Boldness. However, significant correlations were found for sex with Meanness and for age with Disinhibition; these variables were therefore included as covariates in the heritability analyses for these two psychopathy dimensions. Within the combined sample as a whole, heritability estimates were found to be significant for both Boldness ($h^2 = .43$, $SE = .16$, $p < .001$) and Meanness ($h^2 = .32$, $SE = .20$, $p < .05$). However, the heritability estimate for Disinhibition in the combined mother- and nursery-reared sample was appreciably lower and nonsignificant ($h^2 = .13$, $SE = .18$, $p > .20$). None of the c^2 estimates ($Mdn = .01$) approached significance.

Heritability of triarchic psychopathy dimensions by early rearing experiences

Estimates of heritability differed markedly as a function of early rearing experience. Indeed, whereas scores for all three dimensions showed significant heritability in the subsample of mother-reared subjects, scores were not found to be heritable in any case for the nursery-reared subjects. As shown in Table 2, heritability coefficients within the mother-reared subsample ($n = 119$) were high for Boldness and Meanness (h^2s = .66 and .65, respectively) and moderate for Disinhibition ($h^2 = .36$). For the nursery-reared apes ($n = 59$), the analysis resulted in the polygenic additive (genetic) and sporadic (nongenetic) estimates resulting in the same odds, suggesting no detectable genetic contribution; the SOLAR program assigns a

Table 2. Heritability of CHMP-Tri Dimensions for Mother-Reared Participants ($n = 119$)

	Mother-Reared		
CHMP-Tri Dimension	h^2	*SE*	*p*
Boldness	**0.66**	**0.17**	**<.01**
Meanness	**0.65**	**0.27**	**.01**
Disinhibition	**0.36**	**0.20**	**.02**

Note: h^2 = estimated additive genetic influence; *SE* = standard error. As described in the text, sex was included as a covariate in the analytic model for Meanness, and age was included as a covariate in the model for Disinhibition. h^2 estimates shown in boldface are significant at $p < .05$.

default heritability estimate of .00 (p = .50) in this case, so h^2 coefficients in this subsample were set to zero for each dimension and are not shown in Table 2. None of the c^2 estimates approached significance in the analyses for either sample, and none exceeded a value of zero.

Genetic correlations among triarchic psychopathy dimensions

Lastly, to evaluate the extent to which heritabilities for the differing triarchic dimensions in the mother-reared apes (n = 119) were attributable to shared versus separate genetic influences, genetic correlations (ρ_Gs) among scores for the CHMP-Tri scales were estimated. Although phenotypic scores for the three scales were all significantly intercorrelated within the mother-reared sample (Boldness/Meanness r = .34; Boldness/Disinhibition r = .19; Meanness/Disinhibition r = .47), genetic correlations for Boldness with the other triarchic dimensions were nonsignificant (see Table 3). However, Meanness and Disinhibition were strongly genetically correlated (ρ_G = .91), indicating that much of the modest genetic variance in Disinhibition scores (36% of overall phenotypic variance) overlapped with the more substantial genetic variance (65%) in Meanness.

As a complement to the genetic correlations, Table 3 also shows environmental correlations (ρ_Es) among the CHMP-Tri scales in the mother-reared subsample, reflecting associations between phenotypic variance in a given scale attributable to nongenetic sources (i.e., shared and nonshared environment, plus measurement error) and phenotypic variance in the other scales attributable to these sources. Notably, the environmental correlation between the Boldness scale and the Meanness scale was very high (ρ_E = .95); this indicates, in conjunction with the lack of a significant genetic association between the two, that the moderate phenotypic correlation between these scales in mother-reared participants was largely attributable to overlapping environmental influences and perhaps some degree of shared measurement error. The environmental correlation between scores on Meanness and Disinhibition was also very high (ρ_E = .92), in this case mirroring the very high genetic correlation between the two; the implication is that the smaller portion of phenotypic variance in Meanness attributable to nongenetic influence (35%) overlapped very strongly with the larger portion of phenotypic variance in Disinhibition (64%) attributable to nongenetic influence.

Heritability of general factor of psychopathy

Finally, results of a principal axis factor analysis of CHMP-Tri scale scores for mother-reared participants indicated the presence of a single, higher order factor accounting for variance across the three triarchic dimensions, with loadings for each as follows: Meanness = .90, Disinhibition = .52, Boldness = .37.[1] A heritability analysis of scores on this general psychopathy factor, estimated using the standard regression method and incorporating sex as a covariate, revealed a significant additive genetic component (h^2 = .64, SE = .26, p < .01) to these factor scores; the c^2 estimate was nonsignificant and was not above zero.

Table 3. Genetic (ρ_G; Below Diagonal) and Environmental (ρ_E; Above Diagonal) Correlations Among CHMP-Tri Dimensions in Mother-Reared Subsample (N = 119)

	Boldness	Meanness	Disinhibition
Boldness	—	**.95 (.41)**	.30 (.25)
Meanness	.14 (.23)	—	**.92 (.36)**
Disinhibition	.16 (.35)	**.91 (.29)**	—

Note: N = 119. *S*tandard error terms are shown in parentheses. ρ_G and ρ_E values shown in boldface are significant at p < .05.

Discussion

Consistent with previous findings in humans (e.g., Blonigen et al., 2003; Farrington, 2006; Larsson et al., 2006; Taylor et al., 2003; Tuvblab et al., 2016), results indicated both additive genetic and nonshared environmental contributions to interindividual variability in psychopathic tendencies. When examined separately by early rearing background, it became clear that the heritability of psychopathy dimensions varied by early social learning experiences: Whereas all three triarchic dimensions showed significant heritability among mother-reared participants, heritability was not evident for any dimension in the nursery-reared subsample. Additionally, examination of genetic correlations among the three dimensions for the mother-reared participants revealed a substantial proportion of shared genetic influence in scores for Disinhibition and Meanness but no significant genetic correlation for Boldness with either of these dimensions.[2] Lastly, scores on the three CHMP-Tri scales were found to load significantly on a general psychopathy factor in the mother-reared apes, which was found to be appreciably heritable. In what follows, we discuss implications of these findings for understanding of psychopathy as well as for personality, personality disorders, and psychopathology more broadly.

Partially consistent with previous findings regarding the heritability of psychopathic tendencies in humans (e.g., Blonigen et al., 2003; Larsson et al., 2006; Taylor et al., 2003; Tuvblab et al., in press), boldness and meanness, but not disinhibition, were found to be significantly

heritable in the combined mother- and nursery-reared sample when early rearing experiences were not considered. The results for disinhibition were unexpected given consistent evidence in the human literature for a strong genetic basis to this dispositional dimension (e.g., Krueger et al., 2002; Yancey, Venables, Hicks, & Patrick, 2013). Nonetheless, as described in more detail below, given clear evidence of rearing experiences moderating heritability estimates, results in this combined sample should be considered tentative.

As discussed below, effects of early rearing clearly contributed to the unexpected findings for the current sample as a whole. However, other factors could have contributed as well. For example, our genealogical approach to analysis differed from the twin-biometric approach used in most human studies. A notable positive feature of our approach is that it leveraged the full pedigree of each subject, accounting for every familial relationship within the pedigree. Further, although not entirely consistent with the existing human literature, the current findings are nonetheless consistent with prior chimpanzee findings of weak heritability estimates for broad-range personality dimensions when not accounting for rearing background (i.e., Latzman et al., 2015). Regardless of the full range of contributing sources, results from the current work clearly suggest an important role for nongenetic influences on variation in psychopathy dimensions (facets).

Analyses focusing on subsamples of apes were undertaken to evaluate the independent impact of early social rearing experiences (i.e., mother- versus nursery-rearing) on psychopathy scores and their heritability. Consistent with research in humans suggesting an important role for the family environment in the etiology of psychopathy (i.e., Farrington, 2006), early rearing experiences were found to be significantly associated with the disinhibition dimension of psychopathy, with nursery-reared apes exhibiting significantly higher CHMP-Tri Disinhibition scores than mother-reared apes; notably, no corresponding difference was evident for either Boldness or Meanness. Findings of differential heritability for psychopathy facet scores as a function of rearing experience are discussed in the next section.

Consistent with expectations, biometric analyses conducted separately for mother- versus nursery-reared participants revealed evidence of differing etiologies as a function of rearing background for all three triarchic dimensions: Whereas significant heritability was evident for scores on each dimension in mother-reared participants (*Mdn* h^2 = .65), none of the psychopathy dimensions were found to be heritable among the nursery-reared chimpanzees. Within the mother-reared subsample, the heritability for boldness was somewhat higher than its estimated heritability in humans (e.g., Blonigen et al., 2005), perhaps reflecting increased heritability of a more adaptive trait characteristic in an enriched environment. In contrast, the heritability for disinhibition in our chimpanzee sample was markedly lower than its estimated heritability in humans (e.g., Yancey et al., 2013), perhaps reflecting decreased heritability of a more maladaptive trait characteristic in an enriched environment. The heritability of meanness, on the other hand, was highly similar to previously reported heritability estimates for callous–unemotional traits, a closely-related dispositional construct (Viding, Frick, & Plomin, 2007).

The contrasting heritability results for mother-reared versus nursery-reared participants are consistent with accumulating evidence in the human (e.g., Krueger et al., 2008; Miles et al., 2005; Moffitt et al., 2006; Rutter et al., 2006) and nonhuman animal literatures (e.g., Charmantier & Garant, 2005) for variations in heritability as a function of environmental context. Indeed, Latzman et al. (2015) reported parallel findings (i.e., significant heritabilities among mother- but not nursery-reared chimpanzees) in their investigation of the heritability of broad trait dimensions across levels of the personality hierarchy.

In addition to evidence for G×E interactions in human research on psychopathy-related phenotypes (i.e., antisocial behavior; Jaffee et al., 2005), G×E interactions have been reported for other psychological phenotypes including intelligence. For example, as described earlier, Turkheimer et al. (2003) found that the heritability of intelligence varied by the SES of the home within which children grew up, with high heritability estimates evident for individuals from affluent families and estimates approaching zero for those from impoverished families. However, as described by Turkheimer et al., SES is likely not just an indicator of the environment, since it can be confounded by genetic factors (e.g., heritable attributes of parents that contribute to higher SES may also enhance opportunities for children to fulfill their individual genetic potential). Nonetheless, it is important to note that G×E findings for intelligence have been quite mixed (i.e., Hanscombe et al., 2012), underscoring the difficulties in unraveling genetic and environmental contributors.

The work of Turkheimer et al. (2003) illustrates that disentangling etiological influences on psychological phenotypes can be difficult in human samples, due to confounding of environmental and genetic influences. However, the findings for our primate sample are less likely to reflect this type of confounding. As described previously (i.e., Bogart et al., 2014), although offspring in each of the two early rearing groups were not entirely heterogeneous, the degree of genetic diversity was comparable between them. That is, early rearing differences in our sample of chimpanzees were not attributable to genetic differences between offspring born to mothers who were capable of raising them versus offspring born

to mothers who were unable to provide adequate care (Bogart et al., 2014). Nonetheless, it will be important in future research to work toward identifying nuances of alternative early-rearing conditions that give rise to the observed differences in heritability of psychopathy subdimensions. For example, as noted earlier, chimpanzees were removed from their mothers for a variety of reasons including unresponsive care, injury, or illness. Thus, the various reasons for placement in human nursery care, or similar differences in early experiences, could be investigated further in subsequent investigations.

Further analyses of data for the mother-reared subsample of the current study focused on the etiologic bases of observed overlap (covariation) among the three psychopathy dimensions. Examination of bivariate genetic correlations revealed an appreciable role for shared genetic influence in the phenotypic covariation between Disinhibition and Meanness, with the modest heritability of Disinhibition mainly attributable to genetic variance in common with Meanness but no genetic influence in common between either of these dimensions and Boldness. A counterpart examination of the environmental correlations among the CHMP-Tri dimensions revealed a very strong association between Boldness and Meanness, indicating that common environmental influences accounted for most of the moderate phenotypic correlation between these two scales. Importantly, the environmental correlation between scores on Meanness and Disinhibition was similarly high, indicating that the nonheritable portion of variance in Meanness was attributable mainly to environmental influences in common with Disinhibition.

Finally, consistent with previous findings of phenotypic correlations among distinguishable dimensions of psychopathy in both chimpanzees (Latzman, Drislane, et al., 2016) and humans (Hare, 2003; Skeem et al., 2011) as well as distinct etiological connections between particular Triarchic dimensions and broad domains of psychopathology (i.e., Blonigen et al., 2005), we found evidence for a significant genetic contribution to scores on a general, higher order psychopathy factor in the mother-reared subsample. Given the findings from genetic and environmental correlational analyses as described above, it can be inferred that (a) the heritable variance in this general psychopathy factor (64%) largely reflects genetic variance in common between meanness and disinhibition (i.e., because these dimensions shared substantial genetic variance with one another but not with boldness) and (b) the nonheritable variance in this general factor (36%) largely reflects environmental variance that boldness shares with *both* meanness and disinhibition (i.e., because the environmental *r*s for boldness with disinhibition, and in turn for disinhibition with meanness, each approached unity). In sum, it can be concluded that scores on the general psychopathy factor extracted from scale scores for the mother-reared sample are attributable partly to genetic influences in common between meanness and disinhibition and partly to environmental influences in common among all three triarchic dimensions.

These results are consistent with previous findings in humans of a genetic influence on a higher order psychopathy factor explaining shared variance among lower order subdimensions (Larsson et al., 2006). It is important to note that different psychopathy instruments vary in their coverage of the three triarchic model dimensions (Drislane et al., 2014), with a general psychopathy factor more likely to emerge for scales developed from inventories assessing psychopathic tendencies in terms of generally correlated facets—for example, triarchic scales developed using items from Andershed et al.'s (2002) Youth Psychopathy Traits Inventory (i.e., YPI-Tri scales; Drislane et al., 2015). Paralleling relations among the YPI-Tri scales, the CHMP-Tri's Boldness scale correlates to a moderate positive degree with both its Meanness and Disinhibition scales. As described previously (Latzman, Drislane, et al., 2016), the intercorrelations among triarchic dimension scales vary depending on the items used to index the constructs, with Boldness and Disinhibition scales correlating more when each contains items related to sensation-seeking, a trait that encompasses facets related to both of these triarchic constructs (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Drislane & Patrick, 2016). This appears to be true of the CHMP-Tri Boldness and Disinhibition, which contain items indicative of daring approach and restless novelty-seeking, respectively. Thus, the emergence of a general psychopathy factor for the CHMP-Tri scales reflects the generally intercorrelated nature of these rating-based operationalizations of the triarchic model dimensions.

The etiologic findings for the general factor in the current sample provide unique insight into the bases of linkages among phenotypic tendencies toward boldness, meanness, and disinhibition as facets of psychopathy. The finding of significant genetic covariation between meanness and disinhibition points to some innate interdependence in the behavioral expression of these two tendencies. Notably, the heritable variance in disinhibition was subsumed almost entirely by that of meanness, whereas meanness exhibited additional, nonoverlapping heritable variance. This could indicate, in line with current developmental theorizing on psychopathy (e.g., Frick & Marsee, in press), that impulsive-externalizing behavior associated with callous–unemotionality reflects the presence of a distinct genotypic liability. Turning to boldness, this subdimension showed strong heritability, comparable to that for meanness, but its heritable variance overlapped minimally with that of meanness or

disinhibition. By contrast, boldness showed significant environmental variance in common with both meanness and disinhibition. The implication is that experiential influences of certain types can shape the genotypic disposition that underlies boldness—theorized to entail a constitutional weakness in the brain's acute threat (fear) system (Patrick et al., 2009)—in a mean-disinhibited (i.e., aggressive–psychopathic) direction.

A further implication of the current results is that the meanness dimension represents the etiologic point of intersection among the three psychopathy dimensions—connecting to disinhibition at a genetic level and to both disinhibition and boldness via shaping effects of the environment. That is, beyond evidence from recent structural modeling work in humans indicating that "meanness operates as the 'phenotypic glue' that binds distinguishable facets of psychopathy together" (Drislane & Patrick, 2016), current study findings suggest that meanness may also be etiologically central to psychopathy, when considered as a broad trait dimension. This perspective is consistent in turn with the position of Miller and Lynam (2015), who argue that "antagonism" is the central trait disposition underlying psychopathy as an omnibus condition, and with Patrick et al.'s (2009) suggestion that meanness is the main source of overlap between subdimensions of psychopathy as it is commonly assessed in criminal offenders (cf. Hare, 2003).

A notable feature of the constructs of the triarchic model is that they reflect basic behavioral dispositions (i.e., threat sensitivity, affiliation/attachment, inhibitory control; Patrick et al., 2009) and are framed explicitly in neurobiological terms (Patrick & Drislane, 2015). As such, they are relevant to clinical-psychological conditions of many types (e.g., Nelson et al., 2016; Patrick, Durbin, & Moser, 2012) and can serve as valuable referents for biologically oriented studies of psychopathology (Patrick et al., 2013; Yancey, Venables, & Patrick, 2016). Considered together with other recent work (e.g., Latzman, Taglialatela, & Hopkins, 2015; Latzman, Young, & Hopkins, 2016), results from the current study provide clear support for primate-translational operationalizations of these biobehavioral trait constructs and highlight the strengths of a chimpanzee comparative-translational approach for clarifying how these traits arise and what they represent in neural-systems terms. Importantly, and notwithstanding recent decisions by the National Institutes of Health (NIH, 2011) to scale back primate research of some types, work undertaken for the current study fits clearly within the ethical framework of scientifically justifiable research with chimpanzees as outlined by the Institute of Medicine (IOM, 2011). In conjunction with findings from human studies, work of this kind can provide enormously valuable insights into core biobehavioral processes relevant to psychological illness and health (Latzman & Hopkins, 2016).

The current study is not without limitations. First, the sample size, particularly in the case of analyses focusing on early-rearing groups, was relatively modest. This was particularly true for the nursery-reared apes, potentially limiting power to detect heritability in this subsample. Further, the number of parent–offspring dyads and full siblings appears to have been slightly lower in the nursery-reared group, which could have limited our ability to detect heritabilities in this subsample. Importantly, however, average relatedness coefficients did not differ between rearing groups, bolstering our confidence in the findings. Regardless, additional research is needed to replicate the current findings and establish more stable estimates for contributions of genetic and environmental influences to psychopathy among chimpanzees. Nonetheless, it is important to note that our overall sample is one of the largest reported in the nonhuman primate personality literature. Additionally, although widely used in both the human and nonhuman primate literatures, our use of scores on the CHMP-Tri triarchic scales, derived from caretaker ratings of a set of adjective descriptors with accompanying narrative definitions, is only one of a number of potential approaches to assessing the dimensions described within the triarchic model. It will be important for future research to replicate the current findings through, for example, the use of structured behavioral observations based within well-defined ethograms such as the *ChimpanZoo Observer's Guide* (Jane Goodall Institute, 1991). Underscoring the potential for such an approach are findings from human research indicating that key features of psychopathy can be reliably and validly assessed from small samples of behavior (i.e., thin slices; Fowler, Lilienfeld, & Patrick, 2009).

Additionally, it is important to note that chimpanzees encounter a variety of potentially impactful early experiences, whether raised by their biological mothers or in human-managed nursery settings. Given this, as noted previously (i.e., Latzman et al., 2015), our classification of subjects into subgroups based on the ostensibly topographical manner in which they were raised likely obscures important variability within each group. Indeed, previous research with chimpanzees has demonstrated that within the mother-reared group, maternal competence varies across mothers (i.e., Bard, 1994). Notably, however, our approach of grouping chimpanzee participants in this manner likely resulted in a more conservative indication of the role of early social rearing experiences, potentially enhancing confidence in conclusions advanced from current findings. Further, as noted earlier, the genetic diversity was similar across rearing groups, suggesting that rearing group distinctions are a reflection of early experiences rather than merely a reflection of familial aggregation of neglect (i.e., heritable variance). It is also important to note that nursery-reared apes were raised by a different species than the mother-reared

apes (i.e., humans). Although cross-fostering research is possible among rodents and other animal model species, such an approach is not possible in apes. Indeed, unlike in rodents where estimates of environmental influence are unlikely to be a reflection of early social rearing per se (i.e., because rodents do not form similar early mother–child bonds to primates), ape mothers and their offspring develop a clear bond within the first few days of life, resulting in an inability to cross-foster offspring (i.e., foster mothers will not accept the infants).

Relatedly, although human studies typically consider the impact of early adversity on genetic contributions, it is important to note that the degree of adversity within the nursery-rearing environment is likely not parallel to the environment typically studied in humans. Indeed, nursery-reared apes are explicitly removed from their biological mothers as a result of inadequate care, resulting in placement in an environment less adverse than remaining with their inadequate mother, a situation that could be life-threatening. Nonetheless, the distinction between physical and social adversity is an important one. Indeed, whereas nursery-reared chimpanzees are fully provided for with regard to nutrition, shelter, etc., they grow up in the absence of all adult chimpanzee figures. In the wild, chimpanzees spend approximately the first 5 years of their lives largely inseparable from their mothers (Goodall, 1986), with maternal deprivation at an early age associated with a number of dysfunctional behaviors, including, for example, coprophagy (eating of feces) and repetitive rocking stereotyped body movements, among others (for a review, see Bloomsmith, Baker, Ross, & Lambeth, 2006). Thus, although it is unclear whether nursery-rearing represents an experience akin to the early adversity encountered by many humans, it does not appear to be an enrichment environment and is instead likely more similar to many of the familial environments studied in humans and described earlier (e.g., Krueger et al., 2008; Miles et al., 2005). Finally, the sample of apes in the current study is a subset of the sample used by Latzman, Drislane, et al. (2016) in the CHMP-Tri development study. It will thus be important for results to be replicated in additional samples of apes to ensure that results are not sample- or measurement-specific.

Author Contributions

R. D. Latzman, C. J. Patrick, and W. D. Hopkins developed the study concept and contributed to the study design. Testing and data collection were performed by R. D. Latzman, W. D. Hopkins, H. J. Freeman, and S. J. Schapiro. R. D. Latzman, C. J. Patrick, and W. D. Hopkins performed the data analysis and interpretation. R. D. Latzman drafted the paper, and C. J. Patrick and W. D. Hopkins provided critical revisions. All authors approved the final version of the paper for submission.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Notes

1. Results of a principal axis factor analysis in the nursery-reared apes also indicated the presence of a single, higher order factor on which all three CHMP-Tri scales loaded substantially, as follows: Meanness = .78, Disinhibition = .61, and Boldness = .50.
2. It is important to note that the phenotypic correlation between Boldness and Disinhibition may have been too small to decompose.

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