Counterproductive Behaviors: Relations and Heritabilities of Counterproductivity across the Life Domains

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Dedication

To my family and friends, who have given me more than I can ever repay.

Abstract

A sample of 500 male twins is used to demonstrate that counterproductive behaviors across developmental periods and several life domains, including school, non-work, substance use, and work are related. Biometric analyses show that most of the variance in the counterproductivity scales/domains examined, including counterproductivity at work, is attributable to genetic and unique environmental factors. It is also found that a general counterproductivity factor accounts for approximately half of the variance in the specific counterproductivity scales. This general counterproductivity factor is also mostly affected by genetic (75.4%) and unique environmental factors (24.6%).

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Multiple areas within psychology have been interested in a constellation of behaviors that include rule-breaking, aggression, impulsivity, and substance misuse. School and educational psychologists examine disruptive behaviors, juvenile conduct problems, and delinquency (e.g., Loeber & Dishion, 1983). Clinical and counseling psychologists investigate antisocial and externalizing psychopathology (e.g., Krueger, Hicks, Patrick, Iacono, & McGue, 2002; Rhee & Waldman, 2002) as well as drug and alcohol abuse (e.g., Kandel, Yamaguchi, & Chen, 1992). Industrial and organizational psychologists examine counterproductive work behaviors (CWB, e.g., Dilchert, Ones, Davis, & Rostow, 2007; Ones & Dilchert, in press). Although the specific clusters of behaviors are referred to by various terms, the essential, conceptual underpinning of all these phenomena may be characterized as an "impairment of [normative] social and moral behavior" (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999, p. 1032); in short, the transgression of social norms. Regardless of the specific form that they take and the life domain they pertain to, these behaviors are under the individual's control and may involve both cognition (i.e., whether or not explicit reasoning is involved) and motive (e.g., malevolence, clumsiness, habit, or some other factor) in varying degrees (Ones & Dilchert, in press). In general, such behaviors detract from leading a productive life and thus may be termed counterproductivity (Cp). Counterproductivity at school (e.g., cheating) detracts from success in reaching educational goals such as learning. Counterproductivity in adult life (e.g., writing bad checks) has societal costs and detracts from functioning well as a member of society at large. Illicit drug use and alcohol abuse detract from well-being for the individual and others around that individual to the

detriment of leading a productive life. Counterproductivity in work settings detracts from organizational productivity and goals, as well as well-being.

However, there is scant research that explicitly crosses sub-discipline boundaries and links Cp in these multiple life domains. The first purpose of this study is to examine interrelations among Cp in the following domains: school, adult non-work, adult alcohol misuse and illicit drug use, and work. Of particular interest to I/O psychologists, we aim to link Cp in work settings with Cp at school and adult non-work settings. Although these domains span developmental stages and distinct areas of an individual's life, we expect that Cp in these domains will relate positively to one another. Our expectation stems primarily from two sets of previous psychological research: (1) linking adolescent delinquency to adult externalizing behaviors (Blazei, Iacono, & Krueger, 2006) and (2) sizable positive relations among disparate types of CWB in the I/O literature (e.g., Berry, Ones, & Sackett, 2007; Ones & Viswesvaran, 2003; Sackett & DeVore, 2001). Meta-analyses of I/O studies have provided evidence that interpersonal deviance and organizational deviance are highly correlated (Berry et al., 2007). Although there appear to be unique aspects of counterproductive work behavior and they may correlate differently with other factors (e.g., personality traits; Berry et al., 2007), there is also strong evidence that they are highly related, like the various forms of externalizing behaviors included in the spectrum described by Krueger et al. (2002).

Research increasingly indicates that Cp behaviors may not be restricted to a single domain of an individual's life. In fact, many forms of Cp, such as oppositional defiant disorder and substance use have already been incorporated into continua that recognize various externalizing/disinhibited behaviors as being significantly related (Krueger et al.,

2002; Young, Stallings, Corley, Krauter, & Hewitt, 2000). The clinical perspective of these previous efforts is largely based on the observations of high rates of comorbidity among severely disordered Cp behavior in the non-work domain, and thus is largely silent on behavior more germane to the work domain and to behavior less severe than clinical symptoms. It remains to be examined if comorbidity among non-work domains of deviance extend to and connect with forms of workplace deviance. Given the serious consequences counterproductive acts can have for individuals, organizations, and societies, deepening our understanding of the relations among and predictability of different forms of Cp is critical. For this reason, cross-domain investigations of Cp are essential.

There is also evidence that Cp persists across developmental boundaries. Several large scale studies have demonstrated that antisocial behavior in childhood (e.g., conduct disorder) is one of the best predictors of antisocial behavior in adulthood (Moffitt, Caspi, Rutter, & Silva, 2001; Roberts, Harms, Caspi, & Moffitt, 2007). Despite fluctuations in prevalence and specific behavioral manifestations/severity[1], those who are most delinquent in adolescence (age 18) also tend to be more delinquent in adulthood (age 32) (Farrington, 1995).

Three descriptive frameworks also support these conclusions. First, Moffitt's (1993) distinction between early-onset, life course persistent delinquency and adolescence limited delinquency suggests that at least some individuals (approximately 5%) who behave antisocially in childhood and adolescence will persist into adulthood. Dichotomizing antisocial behavior according to this distinction may not be the most

[1] Cp prevalence is highest in early childhood and adolescence with almost all individuals showing at least one form of Cp (Moffitt, 1993)

preferable model, but it has helped to organize data showing that antisocial behavior in childhood and adolescence can have noticeable effects on adult antisocial behavior. Second, a similar structure for categorizing antisocial individuals also acknowledges that some antisocial children and adolescents will persist into adulthood (DiLalla & Gottesman, 1989). Specifically, these authors suggest there are three major types of antisocial individuals: continuous (those who start early and do not desist), transitory (those whose antisocial behavior is limited to the normative period of adolescence), and late bloomers, (those whose antisocial behavior does not emerge until after adolescence). These theories are predicated on observations that set a high threshold for antisocial behavior (e.g., number offenders arrested for homicide, forcible rape, robbery, aggravated assault, burglary, larceny, and auto theft). The current study expands the scope of previous investigations by using more sensitive measures with the ability to detect counterproductive behavior even if it is not severe enough to qualify as a symptom of antisocial personality disorder. Third, evidence has accumulated over the past decade to support the framework recently summarized by Burt, Donnellan, Iacono, and McGue (2011). They marshal evidence for dichotomizing antisocial behavior into aggressive, which tends to be more prevalent early in childhood, and rule breaking, which tends to peak in adolescence; in the process demonstrating that early deviant behavior is related to later antisocial behavior (e.g., alcohol dependence). This dichotomy mirrors the structure used for the past decade in the I/O psychology literature, which separates counterproductive behavior into interpersonal and organization-directed deviance (Robinson & Bennett, 1995).

H1a: Different domains of Cp will be positively and substantially correlated.

The second focus of this paper is to examine the relations among seemingly disparate forms of Cp to see if work Cp can be predicted by Cp in other developmental stages or domains. For I/O psychologists, knowing how Cp in work settings relates to adolescence schooling Cp, adult substance Cp (drug use and alcohol abuse), and adult non-work Cp is important for both theoretical and applied reasons. Theoretically, establishing the nature and degree of association between problematic behavioral clusters from seemingly disparate life domains can illuminate the etiology of workplace Cp, potentially leading to a more complete nomological net and enhanced understanding. Practically, organizations can utilize the information about the associations among Cp in different life domains to help design selection systems.[2] For example, biodata forms or structured interviews can be designed to assess Cp potential among job applicants by inquiring about school Cp and adult non-work Cp. Our study can provide empirical evidence to help focus pre-employment background checks on relevant forms of Cp. However, we expect Cp in different domains to be less than perfectly correlated, such that unique insights into both common and domain specific forms of Cp may be possible as a result of our empirical investigations.

H1b: Work Cp will be substantially predictable using other forms of Cp.

The third aim of this paper is to estimate the extent to which genetic factors influence variance in Cp across multiple domains (i.e., school, adult non-work, adult illicit drug use and alcohol misuse, and work). This aim is in response to calls by previous authors that, "individual differences in organizational outcomes…when possible [should be based on] twin and familial data" (Ilies, Arvey, & Bouchard, 2006). We agree

[2] We note that certain forms of Cp (e.g., substance use) may not be used in selection settings in the United States due to restrictions codified in the Americans with Disabilities Act.

with such recommendations, understanding the value of going beyond investigating relations among groups of behaviors to examine if the likelihood of engaging in Cp is significantly influenced by genetic factors. Significant influence from genetic factors would indicate that differences between individuals in the likelihood of engaging in Cp are associated with differences in their genetic material (i.e., individual differences in the trait are associated with individual differences in the genotype). It may be important to stress that "behavioral genetic research seldom finds evidence that more than half of the variance for complex behavioral traits is due to genetic differences among individuals" and as a result, these studies offer evidence for the importance of environmental influences (Plomin & Daniels, 1987, p.1; Ilies et al., 2006). It is also expected that an individual's genotype interacts with their environment throughout their life to direct intermediate biological processes and produce the individual's phenotype. In behavior genetics, this idea is expounded in reaction range theory (Turkheimer & Gottesman, 1991), which asserts that specific genotypes, when placed in a certain range of environments, are associated with a specific range of phenotypes. The correlation between genotypes and phenotypes is h, and thus h^2 (i.e., the squared correlation) estimates the proportion of phenotypic variance (across individuals) accounted for by genetic variance. In short, heritability estimates index the extent to which genetic differences explain observed, between-individual differences on a trait (Ilies, Gerhardt, & Le, 2004).

Although parents and offspring show significant correlations with regard to general antisocial behavior (even when studied at different ages, r=.30s, Hicks, Krueger, Iacono, McGue, & Patrick, 2004), it is difficult to understand if this relation is due to the

genes they share, the environment they share, or both. Genetically informative studies (e.g., twin studies) provide evidence to disentangle genetic and environmental aspects of familial transmission. For example, adopted children of antisocial, biological parents have been shown to be at elevated risk of exhibiting antisocial behavior compared to adopted children of non-antisocial, biological parents (Crowe, 1974; Cadoret & Cain, 1981). Moreover, adopted children of antisocial, biological parents are at an even greater risk when placed in homes with antisocial, adoptive parents (Mednick, Gabrielli, & Hutchings, 1984; Cadoret, Troughton, & O'Gorman, 1987). Such evidence converges with the findings of other twin and family studies that indicate significant genetic influences on/as the etiology of many forms of general Cp.

When formulating hypotheses regarding the heritability of Cp in various domains, it is critical to note the vast existing behavior genetic literature has found that, "in most domains of psychology…human behavior is influenced by genetic and biological characteristics of individuals" (Ilies et al., 2006). Even in the relatively small number of behavior genetic investigations of I/O psychology variables, sizable heritabilities are often evident (e.g., job satisfaction, Arvey, Bouchard, Segal, & Abraham, 1989; replicated in Arvey, McCall, Bouchard, Taubman, & Cavanaugh, 1994; leadership role occupancy, Arvey, Zhang, Avolio, & Krueger, 2007; vocational interests, Lykken, Bouchard, McGue, & Tellegen, 1993; job switching, McCall, Cavanaugh, Arvey, & Taubman, 1997; transformational leadership, Li, Arvey, Zhang, & Song, 2011). Similar to what has been found for other workplace measures and work-relevant variables, we expect Cp differences among individuals to display sizable heritability. In other words,

genetic influences are expected to make a substantial contribution to individual differences in Cp.

Outside of the I/O psychology literature, a multitude of other studies have reported the heritability and environmentality of externalizing behaviors, drug and alcohol abuse, and delinquency. Again consistent with Turkheimer's first law of behavior genetics that "all human behavioral traits are heritable", each of these behavioral groups appears to be significantly influenced by genetic factors (Turkheimer, 2000, p. 160). Lyons, True, and Eisen et al. (1995) present findings that show significant heritability for several antisocial traits in adolescence with genetic effects increasingly accounting for behavioral variation in adulthood (see also Carey & Goldman, 1997; Krueger, Hicks, & McGue, 2001). Using a sample of monozygotic twins reared apart, Grove, Eckert, and Heston et al. (1990) demonstrated that antisocial behavior in childhood and adulthood was linked to illicit substance use and that all three groups of behaviors were significantly heritable. In her review of more than 100 studies on the heritability and environmentality of antisocial behavior, Moffitt (2005) concluded that approximately 50% of variation in antisocial behavior is due to genetic variation (congruent with previous meta-analyses: Mason & Frick, 1994). Another noteworthy finding from Mason and Frick's (1994) meta-analysis is that the influence of genetic effects on antisocial behavior did not vary with the definition of antisocial behavior used (i.e., criminality, aggression, or antisocial personality).

H2a: Cp in different domains will display significant heritabilities.

Another goal of the current study was to better understand the types of environmental factors that influence Cp. Environmental influences are critical to examine

since many current perspectives on counterproductive work behavior (CWB) focus on organizational/environmental variables as important predictors of CWB (Roberts et al., 2007). Based on behavioral genetic research examining other psychological variables, we expect that factors causing siblings to become more similar (i.e., shared environmental factors) will have a smaller effect on Cp than unique environmental influences. Findings of negligible common environmental effects, especially in non-child samples, are so pervasive that some eminent scholars in the field have codified the notion as law (Turkheimer, 2000). This conclusion has emerged across diverse topics of study and various methodological approaches (e.g., adoption studies, family studies, etc) indicating that the results are not merely an artifact of overreliance on specific designs (Loehlin, Willerman, & Horn, 1987).

More specific to Cp, we expect shared environmental effects to be minimal due to differential heritability over developmental periods and the mix of behaviors in each domain of Cp. Differential heritability acknowledges that the heritabilities of different Cp behaviors will be different across developmental periods. In general, the pattern is often such that shared environmental effects decrease throughout adolescence and genetic effects increase, while unique environmental effects remain consistently influential. This pattern has also been observed in previous studies of general antisocial behavior (Lyons et al., 1995; Goldstein, Prescott, & Kendler, 2001; and Jacobson, Prescott, & Kendler, 2002). Having Cp domains composed of heterogeneous behaviors also increases the likelihood of finding only small effects of shared environment. Some domains may tap aggressive behaviors, often found to be significantly influenced by genetic factors, as well as non-aggressive behaviors (e.g., rule-breaking) which often show larger shared

environmental effects (Eley, Lichenstein, & Moffitt, 2003; Tackett, Krueger, Iacono, & McGue, 2005). One possible explanation for larger shared environmental effects on non-aggressive Cp is that this behavior is more normative (especially during adolescence) and therefore less deviant. On the other hand, aggressive behavior is often more severe, and severe Cp has been shown to be more genetically influenced (Mason & Frick, 1994; Farrington, 1995).

A final reason for expecting small shared environmental effects is due to the use of a reared together twin sample and ACE model. The most commonly used behavior genetic models estimate trait heritabilities due to additive genetics (A) as well as shared (C) and unique environmental (E) influences on individual differences for a trait. However, non-additive genetic effects (e.g., dominance, the interaction of alleles at the same locus, and epistasis, the interaction of different genes/loci) cannot be estimated at the same time since the information they are drawn from is confounded with shared environmental effects in twin studies (Neale & Cardon, 1992). When non-additive genetic effects influence a trait but are not modeled, other estimates in the model can be distorted. Notably, additive genetic estimates can be inflated and shared environment effects deflated (Coventry & Keller, 2005).

H2b: Cp in all domains will display greater unique environmental influences than shared environmental influences.

A fifth purpose of the current study is to examine if and how the genetic and environmental influences on Cp differ by domain. The types of environments individuals operate in can substantially alter the effects of other environmental and genetic factors. More specifically, individuals have varying levels of latitude for engaging in Cp in

different domains. For example, engaging in or refraining from substance use Cp (e.g., using illicit drugs) is likely to be the result of the individuals' choices, and more distally, their predispositions since many individuals lack the tight supervision necessary to stifle this behavior (Dick, Vicken, Purcell, Kaprio, Pulkkinen, & Rose, 2007). On the other hand, academic and work environments often have tighter monitoring, guidelines, regulations, policies and so forth, that may suppress genetic predispositions toward Cp. Such constraints on behavior by certain environments limit the amount of variance in Cp behavior that can be explained by genetic factors, possibly deflating heritability estimates. However, we are also careful to note that individuals may seek and interpret environments in ways commensurate with their personal Cp tendencies and genetic predispositions. In this research, we offer the first *comparative* investigation of differences in genetic and environmental influences on Cp in work settings with those from other life domains and developmental stages.

Research question 1: How do environmental influences on Cp differ by life domain?

Another goal of the current paper is to examine the common variance of Cp variables across domains and decompose genetic and environmental influences on that general Cp factor. Following the demonstrations of Krueger et al. (2005), who provided evidence that various forms of externalizing behavior may be related to a single latent construct and indeed lie along the same spectrum, we are interested in seeing if counterproductivity in various life domains conforms to a hierarchical structure with a general, common counterproductivity factor that relates to each of the lower, domain specific manifestations. Previous research has shown that various forms of antisocial

behavior and their etiologies appear to be related and in some cases, stem largely from a common factor (Young et al., 2000; Armstrong & Costello, 2002).

Possible mechanisms underlying relations with a common factor/etiology include poor behavioral inhibition (e.g., aggression and impulsivity) as well as executive function deficits. A primary component of behavioral disinhibition is highly heritable personality traits such as impulsiveness, which have repeatedly been found to be associated with aggression in adults (Goldman & Fishbein, 2000). Similarly, executive dysfunction has been linked to decreased inhibition of impulsive behavior and aggression (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005; Raine & Yang, 2006; Raine, 1993, 2008). Similar findings emerge from examinations of counterproductive work behavior in the I/O psychology literature (e.g., low self control, low agreeableness, low conscientiousness, and low emotional stability, Marcus & Schuler, 2004; low intelligence, Dilchert et al., 2007). Our interest is to investigate if these unifying factors/relations hold across disparate life domains (e.g., work, school, home, etc).

Research question 2: Is there a general factor of counterproductivity that spans different domains of Cp?

In sum, this research examines relations among Cp variables in different life domains. It also examines genetic and environmental influences on each domain of Cp. Our particular emphasis is on work Cp and its convergence with Cp in other life domains, comparative heritability, and predictability from non-work Cp.

Methods

Our investigation utilizes a behavior genetic design, specifically adult monozygotic (MZ) and dizygotic (DZ) twins, to investigate interrelations among the Cp domains as well as the genetic/environmental influences on Cp in each domain.

Sample The sample consisted of 227 complete pairs (139 MZ and 88 DZ) of same-sex (male), reared-together twins from the midwestern United States. The twins were part of the Minnesota Twin Parenting Project, a subset of the Minnesota Twin Registry population-based, demographically representative sample of twins identified by birth records from the Minnesota State Health Department. Life outcomes for male twins born between 1961 and 1964 were assessed in 1997 when the men were in their early thirties (average age=32.8 years old, SD=0.6), reporting on current and past behavior (Jockin, 1998). The sample was predominantly Caucasian (97.6%) and Protestant or Catholic (92.4%)[3]. Education levels varied with approximately equal numbers of individuals completing only high school, trade school, junior college, and university. Eleven additional individuals did not graduate high school and thirty-seven individuals went beyond a 4-year university (e.g., professional degree). Income also varied widely, from individuals earning less than $10,000 per year before taxes to individuals earning more than $80,000 per year before taxes (in 1997 U.S. dollars). Similarly, occupations ranged widely and were approximately normally distributed across the Hollingshead rating scale from "institutionalized" to "high executives" and "major professionals". The category of "unemployed" or "homemaker" was also substantially represented with nearly one-hundred individuals.

[3] Although our sample is exclusively male and predominantly Caucasian, previous research indicates that neither sex nor race significantly moderate genetic or environmental effects on antisocial behavior (Rhee & Waldman, 2002; Mason & Frick, 1994).

Two hour, structured phone interviews were conducted with each participant and included questions regarding past and present work environment, school, drug and alcohol use, health, family, trouble with the law, and community involvement. An additional packet of pencil-and-paper, self-report inventories assessing numerous aspects of adult and adolescent life was mailed to each participant. Zygosity was determined using a five-item questionnaire that has demonstrated 95% accuracy (Lykken, Bouchard, McGue, & Tellegen, 1990). Forty-six twins completed the assessments but had co-twins that did not participate, so the singletons were only included in individual-level analyses, such as the Cp scale intercorrelations.

Measures Cp was assessed using interview items judged to be relevant to each Cp domain, often based on explicit references to "school" or "work"[4]. School Cp was assessed using 21 items (e.g., "Were you ever suspended or expelled from grades 1 through 8?" "When you were in grades 9-12, how many days a year would you miss or skip school for unexcused absences in a typical year?"). Adult non-work Cp was assessed using 14 items (e.g., "Have you ever owed people money and not paid them back [when they expected to be paid back]?" "Have you ever received speeding or traffic violations? [i.e., not parking tickets]"). Adult alcohol misuse and illicit drug use were measured by using 43 and 20 items, respectively (e.g., "Have you ever gotten into a physical fight when drinking?" "Have you ever used amphetamines [Speed, Stimulants, Diet pills]?"). Finally, work Cp was assessed using 11 items (e.g., "With respect to your behavior at work, have you threatened co-workers?" "Have you ever been formally reprimanded by your supervisor?"). Due to heterogeneity of response options, each item was standardized

[4] Even when individuals are trying to be honest, retrospective reporting of Cp can be unreliable (Henry, Moffitt, Caspi, Langley, & Silva, 1994)

before being averaged into its respective scale (see Dilchert et al., 2007). The internal consistency reliabilities of the scales ranged from .68 to .91 (Table 1).

Table 1

Consistency of Measurement for Counterproductivity (Cp) Scales

Counterproductivity Domain	Total N	Number of Items	Reliability[1]	Average Inter-Item Correlation
School	496	21	.79	.15
Adult Non-Work	424	14	.73	.16
Adult Alcohol Misuse	466	43	.86	.13
Adult Illicit Drug Use	500	20	.91	.33
Work	495	11	.68	.16

[1] Cronbach's Alpha

Analyses Relations among Cp variables were examined at the individual level using all 500 participants. As expected, there were no reliable differences in scale intercorrelations between MZ and DZ twin samples. To examine genetic and environmental influences, we used standard behavioral genetic approaches. Briefly, by comparing the similarity of MZ twins, who have the same genome, to that of DZ twins, who share on average half of their segregating genetic material, the phenotypic variance can be partitioned into genetic, shared environment, unique environment, and measurement error components.

We aimed to estimate influences from additive genetic factors (A), environments that make family members more similar (i.e., shared environment, C), and environments that are unique to/not shared between family members (i.e., unique environment, E) as

well as measurement error (traditionally grouped with E) (see Neale & Cardon, 1992 for a detailed explanation of how twin data is used to estimate A, C, and E). In the current paper we estimate narrow heritability (i.e., additive genetic effects or those that are transmissible across generations) rather than broad heritability, which combines additive and non-additive genetic effects on a trait. Narrow-heritability is most relevant to our study of Cp since previous meta-analyses have found that non-additive genetic effects have very little impact on externalizing behavior (accounting for only 9% of the variance; Rhee & Waldman, 2002) and our design did not permit the simultaneous examination of shared environmental and non-additive genetic effects. The quantitative model we used to partition variance in Cp is given in Equation (1).

$$p^2=a^2+c^2+e^2_{unique}+e^2_{measurement\ error} \quad (1)$$

In Equation (1), p^2 represents the total observed variance in Cp (i.e., the phenotype). This term is equivalent to the more familiar variance statistic, i.e., the average squared difference between the observed scores on a variable and the mean of that variable. Under standard biometric assumptions, the expected MZ twin covariance is a^2+c^2, while the expected DZ covariance is $(½)a^2+c^2$. This formulation is sufficient for the estimation of a^2, c^2, and the combined variance due to unique environment and measurement error (i.e., $e^2_{total}=e^2_{unique}+e^2_{measurement\ error}$). We used the observed reliability estimate (Cronbach α) to decompose the latter component into portions attributable to measurement error (i.e., $e^2_{measurement\ error} = (1-\alpha)\ p^2$) and that portion that is attributable to reliable unique environmental effects (i.e., $e^2_{unique} = e^2_{total}-(1-\alpha)\ p^2$). It is worth noting that most heritability estimates in the literature do not account for attenuation due to measurement error. As a result, most heritability estimates represent the heritability of

scores on a particular instrument rather than the heritability of the construct (Ilies et al., 2006). Observed heritability estimates, therefore, often underestimate genetic influences on constructs and overestimate environmental effects (see Schmidt & Hunter, 1999).

Univariate, biometric models were fit to the data using Mx computing software (Neale, Boker, Xie, & Maes, 2002). Two standard model fit indices were used to evaluate each model: (1) chi-squared fit statistic, and (2) Akaike's Information Criterion (AIC; Akaike, 1987). Improvements in the model's fit, from adding or omitting parameters, can be assessed by changes in these criteria. Like the chi-squared statistic, AIC assesses goodness of fit based on how well the model reproduces the observed data, but it also penalizes model complexity, preferring models that capture the data both accurately and parsimoniously. Lower AIC values are associated with better fitting models (Krueger, South, Johnson, & Iacono, 2008). The same procedures were used to estimate the genetic and environmental effects on the general Cp factor.

Results

Table 2 investigates the intercorrelations between the various Cp variables. Results were examined separately for MZ and DZ twins but correlations were not significantly different in almost all cases even when 90% confidence intervals were examined (Payton, Greenstone, & Schenker, 2003). Since we also have no theoretical reason to expect MZ and DZ twins to differ, we only present combined results. The high intercorrelations between scales, even before correcting for the unreliability estimates, indicate that Cp is often not confined to one domain of an individual’s life. Thus, we found strong support for hypothesis 1a that Cp in different life domains would be positively correlated.

A factor analysis of these data revealed that a general factor of Cp explained on average 48.5% of the variance in observed relations at the scale level. This finding indicates that the Cp exhibited in different domains of one's life are diverse manifestations of the same latent trait in different behavioral domains. The relatively high correlations of work Cp with the other forms of Cp indicate that we may be able to profitably predict counterproductive work behaviors by assessing Cp in other domains of an individual's life. Nevertheless, the correlations also indicated that the various Cp domains were substantially related although still clearly distinguishable, supporting hypothesis 1b (Table 2).

Next, we examined whether or not one can use Cp in non-work domains to predict Cp in the work domain. The results are presented in Table 3. We ran three regression models to examine this question. First we predicted work Cp using manifestations of Cp from all other domains. Regression results indicated that 29% of the variance in work Cp could be predicted using the other Cp scales. Second we trimmed the model to only use contemporaneous measures to predict work Cp. Drug use and non-work Cp behaviors both continued to account for significant portions of variance in work Cp. Third, due to legal restrictions in selection settings (e.g., Americans with Disabilities Act), we investigated a model that predicted work Cp using only information from school and non-work domains. Interestingly, the results indicated that the broad, contemporaneous, non-work domain Cp scale appeared to provide most of the power in predicting work Cp, still capturing nearly a quarter of the variance.

Table 2

Intercorrelations among Counterproductivity Scales

Counterproductivity Domain	School	Adult Non-Work	Adult Alcohol Misuse	Adult Illicit Drug Use	Work
School		.71	.47	.60	.46
Adult Non-Work	.54		.70	.69	.70
Adult Alcohol Misuse	.39	.56		.55	.44
Adult Illicit Drug Use	.50	.56	.49		.58
Work	.34	.49	.34	.46	

N=497-499.
Observed correlations are presented below the diagonal. Correlations corrected for unreliability in both measures are presented above the diagonal.
95% confidence interval for observed correlations= +/- .07-.08; 95% confidence interval for corrected correlations= +/- .06-0.08

We proceeded to investigate hypotheses of the heritability and environmentality of Cp. Initial analyses of the genetically informative data examining the observed and corrected intraclass correlations within twin pairs are provided in Table 4. Given that MZ correlations are significantly larger than DZ correlations in most cases, the findings suggest that each expression of Cp in the specific domains examined (school, work, etc.) has a heritable component. Few of the traits display evidence for shared environmental effects (i.e., r_{MZ} was often significantly larger than r_{DZ} but always less than 1.0)[5]. In Table 3

Cross-Domain Regressions Predicting Work Counterproductivity

Model/Predictors	*B*	*SE B*	Adjusted R	Adjusted R Square
1. All other Cp			.539	.290
School	.025	.050		
Adult Non-Work	.320	.053		
Adult Alcohol Misuse	.024	.051		
Adult Illicit Drug Use	.205	.038		
2. Contemporaneous Cp			.539	.291
Adult Non-Work	.328	.050		
Adult Alcohol Misuse	.025	.051		
Adult Illicit Drug Use	.210	.036		
3. Non-Substance Cp			.496	.246
School	.109	.049		
Adult Non-Work	.441	.047		

[5] However, it may be the case that shared environmental experiences during childhood and adolescence serve to activate/deactivate certain genes in a compounding manner. If this were the case, twins would grow more similar over time the more genetic material they share, as is observed for many traits (Burt, 2009).

contrast, evidence for *unique* environmental effects is obvious for each domain of Cp (1-r_{MZ}). It is important to note that the corrected correlations from Table 4 provide the information used to estimate the additive genetic, shared environmental, and unique environmental effects reported in Table 5 (the measurement error component is based on the reliabilities reported in Table 1).

To more precisely assess which domains of Cp display significant heritabilities, biometric models were fit to decompose the variance into additive genetic, shared environmental, and unique environmental factors, as well as measurement error (A, C, E,

Table 4

Observed and Unreliability-Corrected Twin Correlations for Counterproductivity

Counterproductivity Domain	MZ r_{obs} (CI)	DZ r_{obs} (CI)	X^2_1 (p value)	MZ r_{cor} (CI)	DZ r_{cor} (CI)
School	.56 (.47-.65)	.23 (.10-.36)	8.29* (.004)	.71 (.63-.79)	.29 (.16-.42)
Adult Non-Work	.53 (.43-.62)	.38 (.25-.51)	1.75 (.186)	.72 (.64-.79)	.52 (.40-.64)
Adult Alcohol Misuse	.66 (.57-.74)	.43 (.31-.56)	5.39* (.020)	.76 (.69-.83)	.50 (.39-.62)
Adult Illicit Drug Use	.58 (.49-.67)	.20 (.07-.34)	10.69* (.001)	.64 (.55-.72)	.23 (.09-.36)
Work	.33 (.23-.43)	.18 (.04-.31)	1.44 (.230)	.48 (.39-.58)	.26 (.12-.39)

MZ = Monozygotic (N=139 pairs); DZ = Dizygotic (N=88 pairs); r_{obs} = observed correlation between twin pairs; r_{cor} = Unreliability corrected correlation between twin pairs ; CI= 80% Confidence Interval; * indicates significance of the difference at the $p < .05$ level

and E measurement error, respectively). We began with a model including all four factors, then fit several nested variations omitting A, omitting C, and omitting A and C, to evaluate relative fit for each Cp scale. Table 5 contains the estimates obtained from the models that best fit the data, as assessed by AIC and chi-square fit statistics. In all cases, except for alcohol abuse, a model that omitted shared environmental influences fit the data best[6]. The results support hypothesis 2a, showing that Cp in each domain appears to have significant genetic influences. Our results are also mostly consistent with hypothesis 2b, which proposed that unique environmental influences on the Cp scales would be stronger than shared environmental influences. This was true in all cases except for alcohol abuse. One final interesting finding provided in Table 5 is the surprising proportion of variance accounted for by measurement error in the models. Measurement error is often not distinguished from unique environmental influences, but as can be seen in Table 5, measurement error can significantly inflate estimates of unique environmental influences. It should be noted, however, that we were only able to estimate the magnitude of measurement error using internal consistency reliability and that this proportion will vary depending on the reliability of the measures used. However, typically test-retest reliabilities of Cp measures are not available. Drawing from longitudinal studies involving Cp measures, test-retest reliabilities are clearly lower than internal consistency reliabilities. Thus, the use of internal consistency reliabilities here provides a conservative estimate regarding the degree of variance that can be attributed to measurement error.

[6] Although shared environmental variation in adulthood was not found to significantly affect individual differences in most forms of Cp, it is still possible that shared environments during childhood and adolescence have a formative effect on individuals' developmental Cp trajectory (Van Ijzendoorn & Juffer, 2005)

Finally, I partitioned the biometric estimates in each domain according to whether they stemmed from the same genetic and environmental influences as the general factor or from idiosyncratic, specific factors that only affected single domains. Specific, or residual, effects represent factors that affect the expression of one domain of counterproductivity but not any of the other domains. These specific genetic and

Table 5

Counterproductivity Biometric Model Trait Estimates

Counterproductivity Domain	A (Additive Genetic)	C (Shared Environment)	E (Unique Environment)	Measurement Error
School	.733 (.578-.858)	-	.267 (.142-.422)	.214 (.116-.336)
Adult Non-Work	.740 (.581-.871)	-	.260 (.129-.419)	.269 (.173-.385)
Adult Alcohol Misuse	.360 (.000-.792)	.374 (.000-.704)	.267 (.172-.386)	.138 (.056-.241)
Adult Illicit Drug Use	.646 (.513-.753)	-	.354 (.247-.487)	.093 (.000-.214)
Work	.505 (.284-.697)	-	.495 (.299-.716)	.317 (.183-.468)
General Cp factor	.754 (.647-.832)	-	.246 (.169-.353)	-

Note. 95% Confidence Intervals shown in parentheses.

environmental effects serve to make Cp domains distinct. In all domains except school, general genetic influences, common across all forms of Cp exerted more influence on Cp behavior than specific genetic influences that act on each of the factors. Non-work Cp

was the most extreme example, being overwhelmingly influenced by general genetic influence, which is partially explained by the broad and inclusive nature of the variable. In contrast, unique environmental effects specific to each form of Cp appear to be more influential than unique environmental influences that affect the common Cp factor (Table 6).

Table 6

Partitioning Variance in General and Specific Counterproductivity Effects

Counterproductivity Domain	$A_{General}$	$A_{Specific}$	$C_{General}$	$C_{Specific}$	$E_{General}$	$E_{Specific}$[1]	$E_{Specific}$[2]
School	.10	.31	-	-	.31	.28	.07
Adult Non-Work	.50	.07	-	-	.17	.26	.00
Adult Alcohol Misuse	.32	.25	-	.09	.11	.23	.09
Adult Illicit Drug Use	.41	.31	-	-	.14	.14	.05
Work	.27	.20	-	-	.09	.44	.12

1. E specific in this column includes/is saturated with measurement error
2. E specific in this column is estimated without measurement error

Heritability of the latent Cp factor was estimated to be .754 and its unique environmentality was estimated to be .246. Figure 1 depicts the hierarchical model of Cp that best fit the data. It graphically portrays the general genetic and environmental effects, latent Cp factor, specific Cp factors, and specific genetic and environmental effects.

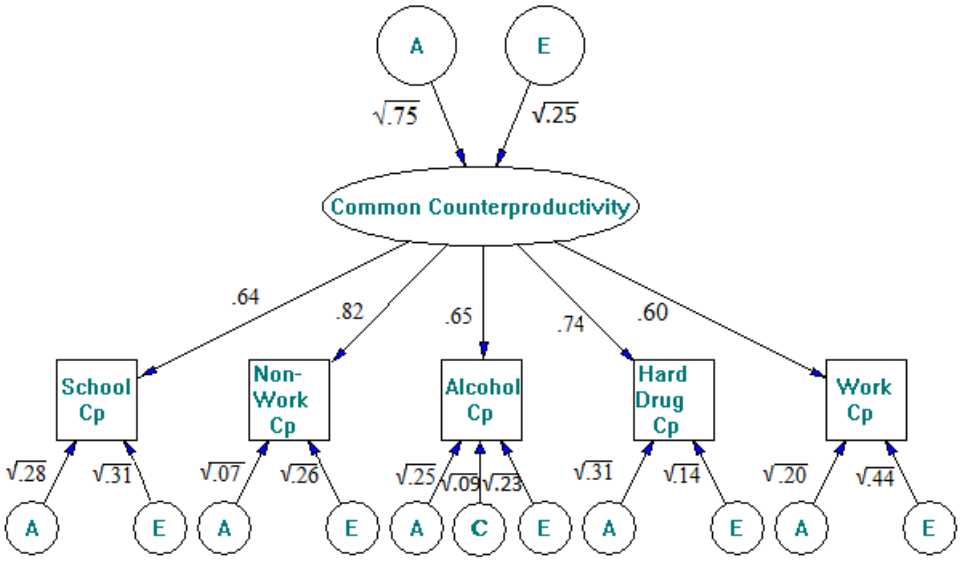
Discussion

Using adult twins' responses to interview questions, we were able to examine counterproductive behavior across time and in several life domains. We found that Cp in

various life domains and at different times in an individual's life were related and largely stemmed from a common, higher-order factor. The notion that individuals' propensity

Figure 1

Counterproductivity (Cp) Path Diagram



for Cp is expressed across diverse domains of their life is remarkable. Although there is much variance not accounted for by the common Cp factor, the evident consistency of individuals' behaviors across domains could be useful in many organizational settings (most notably, perhaps, in employee selection) for understanding and predicting behavior. These results also indicate that, when a more sensitive measure that captures behaviors below clinical or criminal thresholds, many individuals Cp does persist from adolescence to adulthood and the rank ordering of individuals may be more stable than previously thought. However, while the relations between the types of Cp were sizable, the manifestations of Cp were also diverse enough that each specific domain (except adult alcohol misuse) could add at least small increments of predictive power, for example, when trying to predict Cp at work.

Overall, the results indicated that various forms of Cp across several life domains share approximately half of their variance. The data also suggest that the etiological roots, both genetic and unique environmental, are largely shared across forms of Cp. It is important to note, however, that specific genetic and unique environmental factors do appear to be important for all forms of Cp and account for significant variance in each domain. Perhaps most relevant to the application of these results is the finding that work Cp can be predicted relatively well by a set of questions that assess *general counterproductivity in adult life* but avoid legally protected domains, such as substance use issues.

The current findings extend beyond reports of phenotypic association between different domains of Cp and attest that the likelihood of engaging in Cp across domains is influenced by genetic factors. These findings also make the study consistent with the statement that "the stable portion of an individual's nature—the behavioral continuity that makes one unique, recognizable, and predictable—owes largely to an enduring influence of genetic factors" (McGue, Bacon, & Lykken, 1993, p. 105). The latent Cp factor that emerged was highly heritable (h^2= .75), indicating that 75% of the variance in the construct of Cp could be accounted for by variance in genetic factors. However, the heritability differences across the specific Cp domains were striking. Of all Cp domains examined, adult illicit drug use evidenced the highest heritability (h^2= .59), almost twice as large as adult alcohol misuse, the lowest (h^2= .31). Notably, school, adult non-work, and adult illicit drug use Cp each appeared to be primarily influenced by genetic factors. Work Cp also appeared to be substantially influenced by genetic factors, although to a lesser degree than the other Cp scales. One explanation for this finding is that genetic

effects tend to be minimized in regulated environments (e.g., the heritability of smoking is lower in homes where the parents monitor children closely compared to the heritability estimates obtained in low-monitoring homes; Dick et al., 2007) since individuals are not able to develop or follow their natural predispositions as much as in permissive environments or in environments characterized by completely free choice.

In agreement with other authors who have conducted related research (e.g., Krueger et al., 2002), we believe that the tendency to only study counterproductive, disinhibited, or externalizing behaviors in isolated groupings, under the assumption that causal factors across domains/behaviors will be unique, may itself be counterproductive. Krueger and colleagues (2002) point out that rather than separating Cp into silos of delinquency, conduct disorder, substance use, and antisocial behavior, research may instead benefit from examining what is common among all of these behavioral clusters. We observe that there are multiple domains/settings where the same types of Cp behaviors have been studied in their own silos. Most notably, the literature on counterproductive work behavior has burgeoned in I/O psychology over the last decade and a half. Conceptualizing and studying Cp at work as a manifestation of general Cp and relating it to Cp in other domains is essential research that can highlight the common and unique etiologies of counterproductivity. Counterproductive behavior is best structured hierarchically, with a general factor at the top and more specific factors and facets (e.g., domain specific Cp, specific expressions of Cp, and Cp at different developmental stages) branching off below. Such a structure embraces the general genetic and environmental influences on counterproductivity while still preserving and delineating what is unique to different expressions/domains of Cp.

To our knowledge this is the first paper to examine the heritability of counterproductive work behaviors. The absence of shared environment in our model indicates that once genetic similarity is controlled for, twins are no more alike than individuals randomly selected from the population (except in the case of adult alcohol misuse Cp). This means that variation in Cp among individuals is predominantly the result of genetic and unique environmental variation. One plausible explanation of these findings is that individuals are born with varying genetic liabilities to engage in Cp and that unique environmental factors influence the specific form and degree to which those liabilities are expressed.

Even though job-related traits and behaviors have significant genetic components, it is important to note that this does not imply they are genetically determined or immutable. In almost all instances, it is likely that genetic factors are responsive to post-conception environmental effects and interventions (e.g., dietary supplementation to reduce the antisocial behavior of prisoners; Gesch, Hammond, Hampson, Eves, & Crowder, 2002; Raine, 2008). Estimates of heritability presented here reflect differences in the similarity of monozygotic and dizygotic twin pairs, and they are affected by factors other than how important genes are in leading to a certain behavior. In some cases, changing the environment (or population being studied) will alter heritability estimates. Changes to the environment can increase the environmentality of a trait in two scenarios: (1) when the current environments individuals are exposed to do not interact with or affect the trait or (2) when trait relevant components are weak or rare (Arvey & Bouchard, 1994, p. 61). For example, if all individuals in a population are raised in similar environments (e.g., are not abused, are closely monitored by parents, receive

twelve years of education, etc) then the amount of variance in behaviors that can be explained by environmental factors is constrained by the limited range of environmental variance. (Note that the same restriction of range can also affect the amount of variance attributable to genetic factors if all individuals in the sample have similar genetic profiles). Similarly, factors that raise/lower all individual's scores to a similar degree (e.g., secular trends) will only slightly disturb the rank-order of individuals on a trait and hence not be captured in measures of environmental/genetic variability (i.e., environmentality or heritability). Additionally, it should be kept in mind that heritability estimates, by necessity, provide estimates of how much variance in a behavior or trait is not due to genetic factors (i.e., the environmentality of the trait), which can be useful for focusing intervention programs to where they may be most effective (e.g., non-shared environmental factors). Nevertheless, examining the heritabilities of specific behavioral traits does address a primary goal of the behavioral sciences: to understand the causes and correlates of behavior (Arvey & Bouchard, 1994).

Given the increasingly clear importance of genetic factors in work-related behaviors, we believe that research in the I/O field could benefit from utilizing behavior genetic research methods more widely. It is likely that the current (i.e., phenotypic) methods of examining work contexts or phenotypic individual differences/indicators will only explain incomplete portions of the most interesting criteria. Gaining a more complete perspective, we believe, will require increasingly sophisticated investigation of the roots of behavior and the interactions between genetic predispositions, work environments, and rearing environments.

The current paper provides evidence to support previous phenotypic and genetic findings regarding Cp/externalizing/behavioral disinhibition while also extending understanding to the domain of work. We report that Cp in various domains including work is substantially, although imperfectly, related. We also demonstrate that expressions of Cp in non-work domains can be used to significantly predict Cp in the work domain. Building on the relations demonstrated at the correlational level, we fit a hierarchical model, which supported the notion of a common Cp latent factor that accounted for almost half of the variance among the specific forms of Cp. Applying behavior genetic analytic techniques, we observed that the common Cp factor was substantially heritable and that the specific Cp indicators were less heritable. In almost all cases, we did not find evidence to indicate the importance of environmental factors shared between twins. Most relevant to organizations, we found that work Cp was moderately heritable and that the genetic influences on this group of behaviors were equally due to genetic factors specific to work Cp and genetic factors associated with the common Cp latent trait.

The results of this study contribute to the counterproductive work behavior literature by providing the first direct evidence that work Cp can be legally predicted by Cp in other domains of an employee/applicant's life. Previous studies in the I/O literature have examined Cp factors that are not usable in organizational settings due to legal considerations (e.g., diagnoses of conduct disorder) and have examined severe forms of Cp (e.g., criminal convictions). Our study builds upon the previous research by examining less clinical/extreme forms of Cp and reporting results for an older sample that also has genetically informative data (cf. Roberts et al., 2007). This study also contributes to the individual differences literature by extending previous findings to show that Cp in

the domain of work appears to be part of a larger externalizing construct/spectrum. Finally, we have provided evidence that Cp appears to be substantially heritable both as a general latent construct and in more specific manifestations (e.g., work Cp).

Conclusion

Our hope is that better understanding the genetic and environmental sources of employee behaviors can inform the decisions of I/O psychologists and organizations. Recognizing the consistency of Cp across different life domains and developmental stages, as well as the roles of genetic and unique environment factors is a step toward expanding this understanding. Deepening understanding of genetic and environmental sources of human behavior should lead to a more nuanced understanding of how employees perceive, react to, and feel about their jobs, how jobs and other daily events influence their decisions, and how organizations can be designed to work in harmony with human nature (Nicholson, 1998). Examining person-situation interactions across time and situations has the potential to reveal whether genetic effects on CWB are stronger in organizations with close surveillance, or such effects are stronger when organizations give employees greater autonomy. These types of investigations may also illuminate the sources of consistency and variability in behavioral tendencies at work (Ilies et al., 2006). We also hope this research will motivate I/O psychologists to examine gene-environment interactions to better understand and manage counterproductive work behaviors.

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