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A Systematic Review of Genetic Influences on Coping

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Abstract

Coping refers to the way that an individual manages stress. Coping strategies vary; for example, problem-focused coping is directed at reducing or removing a stressor, while emotion-focused coping is directed more at managing reactions that accompany the stressor. How individuals cope with stress can impact their health, but the physiological effects of coping are not well understood. The field of genetics provides tools that could help illuminate the physiology of coping. This review of the literature was conducted to determine what is currently known about the phenotype of coping from a genetic perspective. PubMed, HubMed, PsychInfo, Medline, Scopus, and Google Scholar databases were used to conduct the search, and reference lists were reviewed to identify additional publications. Only studies that measured coping style or a coping domain specifically, were written in English language, and were human-subject focused were included in the review. We identified 19 studies that met these criteria, and 2 types of genetic studies emerged for the review: heritability (n = 9) and candidate gene association (n = 10) studies. Heritability estimates of .68–.76 support a nonadditive genetic component to coping. Replication of association was found for the serotonin transporter and adrenergic receptor beta 2 genes. In addition to finding evidence supporting a role for genetic variability with coping phenotype, it is worth noting that the review revealed a lack of consistency in instruments used to phenotype coping across studies.

Keywords

coping style; heritability; genetics; candidate gene

Folkman and Lazarus's transactional theory of stress and coping (Folkman & Lazarus, 1980; Lazarus, 2006, 2007) defines coping as the attempt to manage problems caused by stressful events appraised as threatening, harmful, challenging, or beyond one's personal resources at that time. Stressful life events may be negative and damaging and are often followed by emotional, cognitive, and behavioral changes (Baum, 1999). Coping with these events can be done actively or passively and is an attempt to manage, master, tolerate, reduce, or

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minimize internal and external alterations of the person–environment relationship (Lazarus, 1993, 2000, 2006, 2007).

People may respond to stressful life events with problem-focused or emotion-focused coping or a combination of both. Problem-focused coping, an active coping style, involves dealing with problems head-on to resolve them as soon as possible. In contrast, emotion-focused coping, a passive coping style, can involve drinking, eating, or smoking to avoid problems (Armeli, Conner, Covault, Tennen, & Kranzler, 2008; Kim et al., 2010; Folkman & Lazarus, 1980; Mackie, Conrod, Rijsdijk, & Eley, 2011; Malan et al., 2006). Usually, people will use problem-focused coping when they consider situations to be controllable and emotion-focused coping when they appraise situations to be uncontrollable (Folkman & Lazarus, 1980). Depending on the stressful events and the stakes involved, a person's coping style can vary or change over time (Folkman & Lazarus, 1980; Lazarus, 2006, 2007).

Lazarus, Lazarus, Another way to define coping styles is to look at whether a person chooses to engage the problem by actively seeking a solution or to disengage from the problem by simply ignoring it (Folkman & Lazarus, 1980; Lazarus, 2006, 2007). An individual who adopts an engagement coping (EC) style (management of stressors) directly engages in handling adverse situations, using mechanisms such as seeking advice from family, friends, or clergy and appropriately expressing feelings, thoughts, and emotions. Individuals with adequate stress management skills practice healthy behaviors such as limiting alcohol consumption, living smoke free, and maintaining a physically active lifestyle. By contrast, an individual who practices a disengagement coping (DC) style (emotion regulation) seeks to escape adverse situations by wishing problems would go away, drinking and smoking to cope, or being sedentary. In addition to the unhealthy lifestyle behaviors DC may trigger, psychological disengagement from stressors may, itself, lead to chronic illness (Lazarus & Folkman, 1984; Malan et al., 2006).

Given the links between coping and health-impacting behaviors, stress and health, and coping and stress, it is likely that insight into the physiological underpinnings of coping could point to potential interventions for health conditions impacted by stress. Additionally, a better understanding of the physiology behind variation in coping style may lead to the development of more effective interventions for improvement in coping style. The field of genomics provides tools that allow the physiology of a phenotype to be explored. Our purpose in conducting the present literature review was to bring together the published literature that addresses coping phenotype and genetics, assess those studies to extract key findings, summarize the state of knowledge of coping genetics, and identify potential opportunities for future research.

Data Collection Methods

We conducted searches in PubMed, HubMed, PsychInfo, Medline, Scopus, and Google Scholar databases and reviewed reference lists from the retrieved articles to identify additional publications. Our Boolean searches comprised the following key word combinations: *coping* AND *polymorphism; coping* AND *genetic; coping* AND *gene; coping* AND *heritability; coping* AND *genomic*. We did not limit our searches by date.

We excluded review articles, duplicate articles, and articles that did not address human subjects from further review, although we did use the reference lists from the review articles to identify additional publications. Articles we selected for review met the following additional criteria: (1) study published in English; (2) study used an instrument to measure coping, coping style, or a domain of coping as a phenotype (studies in which phenotypes related to coping, such as anxiety and depression, were measured but where coping, itself, was not measured were excluded); and (3) the phenotype of coping was analyzed. Data we extracted from the reviewed articles were related to study design, instrument used to phenotype coping, coping domain/coping style assessed in the study, subject information, and key findings from the investigation.

Results

We identified 850 studies using our key word searches. Of these, 19 met our criteria; we excluded the remaining 831 because they did not measure coping as a phenotype, were review articles, or were nonhuman studies. The 19 studies that we reviewed fell into two categories: 9 address the heritability of coping, while 10 focus on candidate genes to explore within the context of coping phenotypes.

Phenotyping Data

The instruments used to phenotype coping, which are listed in Tables 1 and 2, were not consistent among studies. Across the 19 studies we reviewed, researchers used 17 different instruments. Reported Cronbach's α for these instruments ranged from .34 to .92, and the number of items per instrument ranged from 5 to 114.

Heritability Studies of Coping

The nine studies we found in our review that address heritability of coping phenotypes, which are summarized in Table 1, all used twin pairs as subjects. All of these studies used self-report instruments for measuring coping style. Sample sizes ranged from 827 (Kendler, Kessler, Heath, Neal, & Eaves, 1991) to 74 twin pairs (Mellins, Gatz, & Baker, 1996). Therefore, sample sizes were not deemed to be a limitation in the heritability studies.

Heritability estimates across the studies varied widely, a phenomenon most likely due to the fact that methods of phenotyping coping varied across all of the studies. When we focused on independent studies that evaluated heritability of similar phenotypes and had consistent findings, we found that the strongest heritability estimates were for nonadditive genetic factors (.68–.76), indicating that interactions among genes may be important to the phenotype of coping.

In addition, we found that two independent studies had consistent results for the heritability of John Henryism (JH; .34–.35), a form of coping that involves attempts to actively cope with stress regardless of insurmountable odds (James, 1994; James, Hartnett, & Kalsbeek, 1983; James, Keenan, Strogatz, Browning, & Garrett, 1992). JH is often used to characterize coping in Black Americans. Wang, Trivedi, Treiber, and Snieder (2005) examined heritability of perceived stressful life events, anger expression, and JH in 213 Black American and 306 White American twin pairs. They reported that genetic variability

accounted for 34% of the variance for JH and anger control and 47% of the variance in perceived stressful life events. In another study, Whitfield et al. (2006) found that 35% of the variance in JH was due to genetic variability in 180 Black American same-sex twin pairs from non-shared environments.

Candidate Gene Case–Control Association Studies of Coping

We identified 10 independent candidate gene case–control association studies in the literature and have summarized these studies in Table 2. All of the studies used self-report measurements for coping style but only five of the studies included a definition of coping. Studies had relatively small sample sizes ranging from 114 to 450. Candidate genes selected for investigation with coping phenotype had either a central nervous system function or a role in susceptibility to risk factors for cardiovascular disease. Multiple studies investigated the serotonin transporter (*SLC64A;* five studies to date) and the adrenergic receptor beta 2 (*ADRB2*; two studies to date) genes. In addition, we found one study investigating each of the following: brain-derived neurotrophic factor, angiotensin-converting enzyme, monoamine oxidase A, oxytocin receptor, and the dopamine receptor 2 (*DRD2*). Investigators noted significant associations for each of these genes with at least one domain of coping in at least one study.

Both *SLC64A* (Wilhelm et al., 2007) and *ADRB2* (Busjahn, Faulhaber, Freier, & Luft, 1999; Busjahn et al., 2002) were associated with more negative or disengagement-type coping styles. Of the five studies that examined the influence of *SLC64A* on coping behaviors, four found significant associations between the gene and the coping phenotypes, while one found no association. In a study exploring alcohol drinking behavior in male college students, investigators found that men with the L/L genotype of the *SLC64A* promoter polymorphism (5-HTTLPR) drank alcohol as a mechanism for coping with their increased sensitivity to adverse events (Armeli et al., 2008). In contrast, Van der Zwaluw, Kuntsche, and Engels (2011) reported that drinking alcohol as a coping mechanism was significantly associated with the *DRD2* ($p < .01$) in adolescents but not with *SLC6A4*. The authors did, however, find that *SLC6A4* contributed to increased vulnerability during life problems and was related to emotional appraisals of fear, sadness, and joy among individuals with S/S and S/L genotypes (s-carriers). In another study, s-carriers had a decreased ability to cope with stress compared to noncarriers ($p < .05$; Szily, Bowen, Unoka, Simon, & Kéri, 2008). Wilhelm et al. (2007) examined coping strategies and the serotonin transporter polymorphism 5-HTTLPR in 170 students of European ancestry and found that participants with the S/S genotype had fewer coping strategies compared to participants with the L/L genotype.

ADRB2 is the only other gene researchers have investigated in more than one candidate gene study involving coping phenotypes. In both of the studies exploring this link, researchers found significant associations. In the first of these studies, Poole, Snieder, Davis, and Treiber (2006) investigated variation in *ADRB2* along with the influence of race, body mass index (BMI), and anger expression coping strategies on blood pressure (BP). They found two three-way interactions: The first involved BMI, haplotype, and race for resting systolic BP (SBP) and indicated that White men had elevated BP and BMI. The second included haplotype, race, and anger-in for resting SBP and indicated that anger-in presented in Black

men (n = 110) but not in White men (n = 124). In the second study, Busjahn et al. (2002) found an association between *ADRB2* and active coping, expressed as a quantitative trait, along with defense, emotional, and active coping factors.

Discussion

There is a dearth of published studies addressing coping as a phenotype within the context of genetic studies. However, the data evaluated across the 19 studies that we reviewed support a role for genetic variation to explain coping phenotypes. The combined findings of the heritability studies indicate that nonadditive genetic factors very likely play a role in coping. Future research on the genomics of coping should integrate this insight to inform the way in which genetic data are analyzed in the context of the coping phenotype. Additionally, findings of two independent heritability studies suggest that, if JH is used as the phenotype for coping, genetic variation will account for approximately one third of the variability in coping variability, particularly among African Americans. This insight will be valuable for analytical processes where having a reliable heritability estimate for a phenotype can substantially strengthen a study.

Candidate gene association studies also support the contribution of genetic variability to the phenotype of coping, particularly in genes associated with central nervous system function and risk factors for cardiovascular disease. Although investigators found associations between each of the candidate genes under investigation and the coping phenotype in at least one study, *SLC64A* and *ADRB2* were associated with the phenotype in multiple studies. A number of previous studies (Golimbet, Volel', Dolzhikov, & Isaeva, 2012; Haenisch et al., 2012; Markus, 2013; Mendes et al., 2013; Starr, Hammen, Brennan, & Najman, 2012) have demonstrated an association between the S allele of the 5-HTTLPR polymorphism of *SLC64A* and depression. It may be that in future larger, well-designed studies, researchers will find that this allele both links depression and coping phenotypes and provides a biological explanation for the linkage. Previous research has also revealed links between *ADRB2* and both resting BP and regulation of BP during stressful situations (Busjahn et al., 2002; Li et al., 2001). These findings are interesting, given the known links between stress and risk factors for cardiovascular disease. In another study, Dimsdale, Mills, Patterson, Ziegler, and Dillon (1994) studied coping with chronic life stress, hypertension, and lymphocyte ADRB2 receptors, characterized by density, in 25 men (Black n = 3, White n = 22). They found that men with higher levels of life stress had lower receptor density ($p < .005$) and that coping style and life stress explained 50% of the variance in density. This study provides a potential mechanistic link between the *ADRB2* gene and coping, whereby variation in the gene may play a role in coping phenotype via influence on the density of ADRB2 receptors.

We found adequate justification in the studies we reviewed regarding selection of the candidate genes. However, based on our review, we do recommend that future studies involve large sample sizes and consistently phenotyped subjects and that investigators use a nonparametric genome-wide approach to gain insight into the genomics of coping.

One very important finding of our review is that there is no consensus regarding instruments for phenotype coping. Two initiatives are underway to address consensus of phenotyping instrumentation in general: the Patient Reported Outcomes Measurement Information System (PROMIS, www.nihpromis.org/default) and the Consensus Measures for Phenotypes and Exposures (PhenX, https://www.phenx.orgwww.phenx.org). Currently there are no instruments within PROMIS to address coping. However, PhenX identifies one such instrument: the Coping Response Inventory (CRI), a self-reported instrument with 48 items that has versions for both youths and adults (Moos, 1993). It is interesting to note that none of the reviewed studies utilized the CRI to phenotype coping.

Consistency in phenotype characterization is paramount both within a single genetic study and when trying to compare two or more genetic studies. A consensus regarding instruments for phenotype coping is a necessity if the science of the genetics of coping is to advance. We thus recommend that the coping research community establish a consensus instrument(s) for phenotype coping and that the instrument(s) be made readily available through PROMIS and PhenX.

Conclusion

Given the sparseness of the data available in the literature on the genetics of coping, we encourage investigators who explore coping within the context of their studies to consider adding a genetic component to their program of research. Such researchers could collect and bank biological samples across studies until they have a critical mass for evaluation or for participation in a multiproject effort. The ability to link genetic data with coping phenotype as well as with health-related phenotypes that may be impacted by stress and/or coping provides an opportunity to unravel the physiologic underpinnings of coping and, perhaps, to develop biologically based interventions to improve coping skills. Further, such research may help to explain the biological links between coping and health conditions that are impacted by stress.

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Table 1

Heritability Studies Examining Genetic Influences on Coping.

Study	Coping Domain/Style	Measure	Study Population	Key Findings
Busjahn, Faulhaber, Freier, and Luft, 1999	Family oriented, social, enhancement/ social, coping social	SVF	212 German M/F twin pairs	19 coping scales: additive genetic factors (h^2 = .10–.43), nonadditive genetic factors (h^2 = .12– .68).
Jang, Thordarson, Stein, Cohan, and Taylor, 2007	Emotion oriented, task oriented, social diversion, denial	CISS	171 British Columbian twin pairs	Task oriented, emotion oriented, and social diversion were moderately heritable (h^2 = .17–.20); distraction influenced solely by environment.
Kato and Pedersen, 2005	Problem solving, turning to others, avoidance, neuroticism, extraversion, openness to experience	BMCS	446 Swedish M/F twin pairs	Problem solving (h^2 = .30–.38), turning to others (h^2 = .21–.37), avoidance (h^2 = .15–.34).
Kendler, Kessler, Heath, Neal, and Eaves, 1991	Turning to others, problem solving, denial	WOCC	827 French F twin pairs	Turning to others (h^2 = .30), problem solving (h^2 = .31), and denial (h^2 = .19).
Kozak, Strelau, and Miles, 2005	Task oriented, emotion oriented, social diversion, distraction	CISS	612 Polish M/F twin pairs	Emotion oriented (h^2 = .35), task oriented (h^2 = .34), distraction (h^2 = .33), social diversion (h^2 = .39).
Mackie, Conrod, Rijsdijk, and Eley, 2011	Family oriented, social, enhancement/ social, coping/social	SUQ	711 White M/F twin pairs from the UK	Additive effect: enhancement/social motives class (h^2 = .28 and .20, respectively), social motives class (h^2 = .66). Nonadditive effects (h^2 = .76) for coping/social motives class.
Mellins, Gatz, & Baker, 1996	Distraction, use of parents/peers, problem solve, self-soothe	CPCQ	74 White, Hispanic, Black, Asian, and mixed M/F twin pairs	Distraction (h^2 = .99), use of parents (h^2 = .53), use of peers (h^2 = . 18), problem solve (h^2 = .00), problem focused (h^2 = .57), emotion focused (h^2 = .00).
Wang, Trivedi, Treiber, and Snieder, 2005	John Henryism, anger expression, perceived stressful life events	AES, JHACS	519 White/Black American M/F twin pairs	Heritability estimates: anger-in (h^2 = .18), anger-out (h^2 = .10), anger control (h^2 = .34), JH (h^2 = .34)
Whitfield et al., 2006	John Henryism	JHACS	180 Black American M/F twin pairs	JH heritability estimates: JH (h^2 = .35).

Note. AES = Anger Expression Scale; BMCS = Billings & Moos Coping Scale; CISS = Coping Inventory for Stressful Situations; CPCQ = Children's Perceived Coping Questionnaire; F = female; JH = John Henryism; JHACS = John Henry Active Coping Scale; M = male; SUQ = Substance Use Questionnaire; SVF = Stressverarbeitungsfragebogen; UK = United Kingdom; WOCC = Ways of Coping Checklist.

Table 2

Candidate Gene Case–Control Association Studies Examining Genetic Influences on Coping.

Study	Coping Domain/Style	Gene	Coping Measure	Study Population	Key Significant Findings
Armeli, Conner, Covault, Tennen, & Kranzler, 2008	Drinking to cope/enhance	*SLC64A*	MAUS	360 White American subjects	*SLC64A* 5-HTTLPR promoter polymorphism was significantly related with drinking to cope and negative life events ($p < .01$). L/L men more likely to drink to cope than S/S men ($p = .005$).
Busjahn et al., 2002	Active coping, emotional coping	*ADRB2*	SVF	166 German twin pairs	Association was found between the *ADRB2* gene and the active coping style ($\chi^2/df = 5.38$, $a^2 = 0.21$, $p = .02$).
Caldwell et al., 2013	Emotion-focused coping, problem-focused coping	*BDNF*	SCOPE	124 subjects of American, Arab, South/Southeast Asian, Latin American, Aboriginal, and mixed descent	Problem-focused coping did not differ based on *BDNF* genotype. Emotion-focused coping varied as a function of *BDNF* genotype, such that individuals with at least one Met allele endorsed more emotion-focused coping than those with a Val/Val genotype, $F(1, 119) = 4.70$, $p < .05$, $G^2 = 0.04$.
Cicchetti, Rogosch, & Sturge-Apple, 2007	Self-coping, seeking others, avoidance	*SLC64A, MAOA*	ACOPE	339 Caucasian, African American, Hispanic & other racial/ethnic groups	In high-*MAOA*-activity genotype groups, significant association between self-coping and fewer depressive symptoms ($p < .05$). No associations for *SLC64A* with coping noted.
Heck et al., 2009	Coping styles	*ACE*	SVF	194 German subjects	*ACE* gene intronic SNP (rs8066276) was significantly associated with positive coping style distraction (adjusted $p = .009$; $f = 0.10$).
Kim et al., 2010	Emotional support seeking	*OXTR*	ND	274 Korean and Korean American subjects	Korean Americans reported more emotional support–seeking than Koreans ($p = .001$) for AA genotype compared to GG/AG genotypes ($p = .048$).
Poole, Snieder, Davis, & Treiber, 2006	Anger-in/anger-out coping strategies	*ADRB2*	SAES	450 White American and Black American subjects	Haplotype Gly16/Glu27, BMI, and race for resting SBP interaction showed high BP and high BMI in White men ($n = 124$). Haplotype Gly16/Glu27, race, and anger-in interaction indicated higher anger-in carriers had higher resting SBP. Interactions with anger-in were present in Black men ($n = 110$) only.
Szily, Bowen, Unoka, Simon, & Kéri, 2008	Emotional appraisal of fear, sadness, and joy	*SLC64A*	SAQ	114 White Hungarian subjects	*SLC64A* 5-HTTLPR promoter polymorphism s-carriers significantly associated with coping ability for fear and sadness ($p < .05$) compared to noncarriers.
Van der Zwaluw, Kuntsche, & Engels, 2011	Drinking to cope, coping motives	*DRD2 SLC64A*	DMQ-R	282 Dutch subjects	*DRD2* significantly associated with drinking to cope ($p < .01$). No significant association between *SCL64A* and drinking to cope.
Wilhelm et al., 2007	Coping with stress	*SLC64A*	ABM, CUS	127 White European subjects	*SLC64A* 5-HTTLPR promoter polymorphism was associated with using fewer problem-solving strategies ($p < .001$).

Note. BP = blood pressure; ABM = Anti-Depressive Behavior Measure; ACOPE = Adolescent-Coping Orientation for Problem Experiences; CUS = Coping under Stress; DMQ-R = Drinking Motive Questionnaire–Revised; MAUS = Motivations for Alcohol Use Scale; ND = no data; SAES = Spielberger Anger Expression Scale; SAQ = Scherer's Appraisal Questionnaire; SCOPE = Survey of Coping Profile Endorsement; SNP = single-nucleotide polymorphism; SVF = Stressverarbeitungsfragebogen; WOCQ = Ways of Coping Questionnaire; Systolic Blood Pressure (SBP).