Repressive Coping and Blood Measures of Disease Risk: Lipids and Endocrine and Immunological Responses to a Laboratory Stressor[1]

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Relations between repressive coping and a variety of health-related variables, including insulin, lipids, catecholamines, and cellular immune components, were investigated in a laboratory study of acute stress among a sample of healthy male college students ($N = 83$). Compared to nonrepressors, at baseline, repressors had fewer numbers of circulating CD4 (T-helper) cells, greater numbers of natural killer (NK) cells, lower high-density lipoprotein (HDL), a higher total/HDL cholesterol ratio, and higher fasting insulin levels. In response to an acute laboratory stressor (Stroop Color Word Conflict Test), repressors demonstrated an attenuated increase in the number of circulating NK cells compared to nonrepressors. Confounds such as physical activity, age, and smoking were unrelated to the dependent measures.

There is a growing emphasis on individual differences in the study of physiological processes and disease. The repressive coping style, a personality construct defined by low trait anxiety and high defensiveness, may represent one important individual difference carrying increased disease risk. Individuals who employ this coping style are characterized by avoidance of threatening information and failure to report negative emotions in situations where such emotions are appropriate (Bonanno & Singer, 1990; Weinberger, 1990; Weinberger, Schwartz, & Davidson, 1979). In contrast to their low reports of distress, repressors typically manifest exaggerated physiological reactivity to acute laboratory stressors (Asendorpf & Scherer, 1983; Barger, Kircher, & Croyle, 1997; Gudjonsson, 1981; Newton & Contrada, 1992; Weinberger et al., 1979). Each of these characteristics—low

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reports of distress (Derogatis, Abeloff, & Melisaratos, 1979; Gross, 1989; Temoshok, 1985), exaggerated physiological reactivity (Manuck, 1994), and the dissociation of verbal and physiological response systems (Kneier & Temoshok, 1984; Schwartz, 1983)—is hypothesized to be a marker for disease risk.

Several physiological concomitants of repressive coping add further credence to the hypothesis that these individuals are at increased health risk. Repressors have higher basal salivary cortisol levels than do low- (but not high-) anxious individuals (Brown et al., 1996), and male repressors have higher total cholesterol than do low-anxious individuals (Niaura, Herbert, McMahon, & Sommerville, 1992).

There is also evidence that repressive coping is associated with altered immune function. Indeed, when repressors are compared with nonrepressors, it has been demonstrated that repressors have lower monocyte counts (Jamner, Schwartz, & Leigh, 1988) and poorer latent control of the Epstein-Barr virus (Esterling, Antoni, Kumar, & Schneiderman, 1990), although later research has suggested that such immune differences were a result of independent effects of anxiety and defensiveness, rather than the configuration of low anxiety and high defensiveness (i.e., repressive coping; Esterling, Antoni, Kumar, & Schneiderman, 1993).

In addition to differences in basal physiological measures, it has been established that repressors demonstrate exaggerated autonomic reactivity to laboratory stress (Asendorpf & Scherer, 1983; Barger et al., 1997; Gudjonsson, 1981; Newton & Contrada, 1992; Weinberger et al., 1979). Given that interindividual differences in the magnitude of these acute physiological responses are reproducible (Manuck, 1994; Marsland, Manuck, Fazzari, Stewart, & Rabin, 1995), exaggerated reactivity may be an enduring dispositional attribute. As such, these response tendencies may be vulnerability factors moderating relationships between stress and disease. For example, reactivity may impact disease susceptibility and progression via modulation of immune parameters that accompany exposure to stress (Kiecolt-Glaser & Glaser, 1995).

Reactivity may also influence alternate processes with health implications. The heightened platelet aggregability (Malkoff, Muldoon, Zeigler, & Manuck, 1993) or increased serum cholesterol (Muldoon et al., 1995) that follows stress exposure may promote coronary heart disease. In sum, individuals with high dispositional reactivity may be at greater disease risk, and repressive copers often show exaggerated physiological reactivity. Repressors' autonomic reactivity, while not a focus of the present article, underscores the potential importance of altered lipid and immune parameters observed among repressors.

Overall, the literature suggests that physiological concomitants of repressive coping, including both baseline measures and responses to stress, may place such individuals at risk for disease. In the current study, we evaluate associations between repressive coping and a number of health parameters, including coronary

heart disease (CHD) risk factors (cholesterol, insulin), functional and enumerative immune measures, and physiological markers with health implications (plasma catecholamines: epinephrine and norepinephrine). We also report changes in blood pressure, heart rate, and cellular immune response following acute laboratory stress among repressors and nonrepressors.

Based on earlier work showing that male repressors have higher total cholesterol (Niaura et al., 1992), we expected that repressors would have a lipid profile reflective of elevated CHD risk. We assessed total cholesterol, high- and low-density lipoproteins (HDL and LDL), and also calculated the total cholesterol/HDL cholesterol ratio. Total, HDL, and LDL cholesterol are independent risk factors for CHD (Verschuren et al., 1995; Gordon et al., 1989; Avins & Browner, 1998, respectively), and the total cholesterol/HDL cholesterol ratio provides additional discriminative capability to total cholesterol and LDL cholesterol levels (Kinosian, Glick, & Garland, 1994). We also assessed insulin, another independent risk factor for CHD (Després et al., 1996). Insulin, total cholesterol, LDL cholesterol, and total cholesterol/HDL cholesterol ratios are positively associated with CHD risk; while high-density cholesterol is negatively associated. These were the primary health measures of interest, given their well-established relationship to CHD.

We also expected that repressors would show heightened sympathetic nervous system (SNS) responses (via catecholamines) to stress (e.g., Barger et al., 1997), relative to nonrepressors. Stress-related SNS reactivity was also expected to produce concomitant changes in immune parameters among repressors (cf. Bachen et al., 1995; Benschop et al., 1994). The present report represents a reanalysis of some of the data from Manuck et al. (1996) in which the effect of a family history of hypertension on cardiovascular responses was examined.

Method

Participants

A sample of 108 Caucasian undergraduate males (aged 18 to 22 years) were recruited as part of a larger study of familial and genetic influences on cardiovascular disease risk among young adults (Manuck et al., 1996).[3] Participants fasted overnight and were asked to abstain from all medications (except acetaminophen or terfenadine) for at least 3 days, from caffeine and alcohol for 12 hr, and from smoking or other tobacco use for at least 3 hr. Of the 108 participants, immune measures were available for 83.[4] All of the participants gave informed consent to

[3]In the original investigation, the relationship between familial hypertension risk and cardiovascular and catecholamine responses to mental and physical stressors was evaluated. Because familial hypertension risk was unrelated to coping style or the dependent measures, it is not discussed further.

[4]Inadequate venous access or venous collapse resulted in a reduced *N*.

participate in this investigation, which was approved by the Biomedical IRB of the University of Pittsburgh.

Laboratory Procedure

Each person participated in five experimental sessions (beginning at 8:30 a.m.), two of which are described here.[5] Upon arrival at the laboratory for the first session, participants were instrumented for cardiovascular monitoring, and an 18-gauge indwelling venous catheter was inserted into an antecubital vein. Participants then rested quietly in a seated position for 30 min. During the last 4 min of this period, 30 ml of blood was drawn to determine plasma catecholamine levels and to assess baseline immune levels. Participants then performed a series of acute laboratory stressors, including a computerized version of the Stroop Color-Word Conflict test (Manuck, Cohen, Rabin, Muldoon, & Bachen, 1991; Muldoon et al., 1992). This task was performed under time pressure and against a distractor (random test responses) generated by computer voice synthesis for 21 min.

The Stroop task can be presented continuously over a relatively long duration (i.e., 20 min) and thus presents a useful paradigm to assess acute physiological responses. This task's effects on cellular immunity and its utility as a potent laboratory stressor are well documented (e.g., Manuck et al., 1991; Marsland et al., 1995). The Stroop task is best characterized as an active coping task, where behavior is primarily under the control of appetitive reward or positive incentive motivation (Fowles, 1982, 1988). In this study, rewards were monetary (up to $20) and were provided for good performance. This active coping process is similar to behavioral challenges used in other repressive coping studies (e.g., mental arithmetic; King, Taylor, Albright, & Haskell, 1990). However, it is important to note that active coping tasks such as mental arithmetic do not always evoke exaggerated cardiovascular reactivity among repressors (e.g., Tomaka, Blascovich, & Kelsey, 1992).

Blood was drawn at minutes 5, 11, and 17 during the Stroop to assess catecholamine responses, and the values were averaged. Blood from the last draw was used for posttest immune assessments. At a later session (approximately 12 days following the initial session), blood was obtained for determination of fasting insulin, total serum, and HDL cholesterol concentration. At that time, the Spielberger State–Trait Anxiety Inventory (STAI; Spielberger, 1983) and the Marlowe-Crowne Social Desirability scale (MC; Crowne & Marlowe, 1960) were completed. The Harvard Alumni Activity Survey (Paffenbarger, Wing, &

[5]The assessments reported here occurred during the first and fourth sessions. The other sessions involved physical challenges (e.g., cold pressor) and cognitive assessments that were relevant to hypotheses involving children of hypertensives, but had no theoretical implications for the present investigation.

Hyde, 1983) was used to assess the types and duration of activities engaged in during the past week. Participants reported sport and nonsport activities that were assessed for frequency and duration to estimate total energy expenditure for the 7-day period.

Cardiovascular Measures

Baseline blood pressure (BP) readings were calculated from the average of four readings taken at 90-s intervals over the last 6 min of the initial rest period. Task BP was obtained by averaging 13 BP readings obtained at 90-s intervals throughout the Stroop task.

Heart rate was determined from a continuous, bilateral electrocardiogram (ECG). Heart rate was digitized and analyzed to obtain a mean heart rate in beats per minute (BPM) for the last 30 s of each 90-s interval occurring during baseline and task periods. We report averages of heart rate values across these recording intervals for baseline and task periods.

Personality Measures

The most common method of classifying repressors and nonrepressors requires measures of both trait anxiety and defensiveness, the latter measured by the MC scale. The MC has good internal consistency (.88) and test–retest reliability (.89; Crino, Svoboda, Rubenfeld, & White, 1983; Crowne & Marlowe, 1960). Despite its social-desirability designation, the MC does not tap a response bias (McCrae & Costa, 1983), but rather psychological defensiveness (Paulhus, 1984; Weinberger et al., 1979). The STAI was used to measure anxiety. The STAI is an internally consistent, reliable, and valid measure of anxiety (Spielberger, 1983). Because both of these scales have adequate stability, the collection of these measures at the later laboratory session was not believed to be problematic.[6] Previous investigations have used these two measures to classify repressors

[6]An anonymous reviewer wondered whether the experimental procedures may have temporarily altered responses on the personality classification scales. Neither these data nor any published data we are aware of can speak directly to this issue. However, several characteristics of the measures and setting suggest that such alterations were unlikely. First, the personality assessments were obtained in a different room and by different personnel than during the psychophysiological challenge session. Second, the anxiety measure would appear to be most sensitive to contextual influences, but the trait-oriented referents for the STAI items would tend to be insensitive to any transient emotional states. Similarly, repressors have been known to report *less* anxiety following transient stress (cf. Weinberger et al., 1979), so for them, any alteration in anxiety scores would tend not to affect their coping classification. Finally, the MC scale items (e.g., "No matter who I'm talking to, I'm always a good listener"; "I like to gossip at times") do not reflect response dimensions that appear sensitive to transient emotional elements of the personality assessment session.

and nonrepressors (Denollet, 1991; Fox, O'Boyle, Barry, & McCreary, 1989; Tomarken & Davidson, 1994).

Lipids

Serum lipid concentrations were determined by the Heinz Nutrition Lipid Laboratory of the University of Pittsburgh, which has met the accuracy and precision standards of the Centers for Disease Control and Prevention since 1982. Total cholesterol was determined enzymatically (Allain, Poon, Chan, Richmond, & Fu, 1974) by means of a bichromatic autoanalyzer. HDL cholesterol level was determined after selective precipitation by heparin/manganese chloride and removal by centrifugation of very-low-density lipoproteins and LDLs (cf. Warnick & Albers, 1978). Triglyceride levels were determined enzymatically (Bucolo & David, 1978). This involved hydrolysis of triglycerides and subsequent quantitation of glycerol content. The LDL concentration was estimated with use of Friedewald, Levy, and Frederickson's (1972) formula. We also calculated the total cholesterol/HDL ratio, which has been show to predict CHD risk better than either total cholesterol or LDL cholesterol alone (Kinosian et al., 1994).

Catecholamines

Blood samples were anticoagulated with ethylenediaminetetraacetic acid (EDTA), chilled, and immediately centrifuged at 4°C; plasma was then separated and frozen at -80°C until analysis. Epinephrine and norepinephrine were measured by high performance liquid chromatography (HPLC) with electrochemical detection (Manuck et al., 1991). Following extraction with alumina, HPLC determinations were conducted using a Phase II, reverse-phase, 3-micron column. Peak catechol heights were measured automatically by Chromatochart-PC and compared to standards tested for HPLC purity.

Immune Assays

Functional immune measures included whole-blood assessment of lymphocyte proliferative responses to phytohemagglutinin (PHA). PHA is a general T-cell mitogen that is commonly used as a measure of immune function. PHA stimulates the T-cell population to divide, and is reliably associated with decreases following stress (Herbert & Cohen, 1993).

Proliferative responses were used to establish dose-response curves at final concentrations of 2.5, 5.0, 10.0, and 20.0 mg/ml. Response was defined as the difference in counts per minute between stimulated and unstimulated samples, determined separately for each concentration. Peak mitogenic responses were found at doses of 5 mg/ml for PHA. Analyses were based on this optimal

concentration because prior comparisons involving use of a single optimal concentration versus several mitogen concentrations (as in a repeated-measures analysis) have been shown to yield similar results (Herbert, Coriell, & Cohen, 1994). In addition, circulating populations of T-cell subtypes, B cells, and natural killer (NK) cells were assessed in whole blood using dual-color fluorescence analysis with a Becton-Dickinson FACScan flow cytometer (San Jose, California). Lymphocyte subsets were analyzed using monoclonal antibodies labeled with either fluorescein or phycoerythrin to quantify CD3+ (total T), CD3+CD4+ (T-helper), CD3+ CD8+ (T-suppressor/cytotoxic), CD3-CD19+ (B), and CD3-CD16+CD56+ (NK) cells (Manuck et al., 1991). Absolute numbers of cells were calculated from a complete blood count. Pre- and posttask blood samples were assayed in the same batch on each occasion of testing.

Results

Coping Classification and Analytic Strategy

Repressors were defined as scoring below the median of anxiety (≤ 17) and above the median (≥ 15) on the MC. Earlier work has supported both the conceptual (Weinberger & Schwartz, 1990) and empirical (Barger et al., 1997) rationale for evaluating coping-style differences by comparing repressors to nonrepressors (i.e., a typological approach). To determine the suitability of such a strategy for the present study, we performed two sets of analyses. First, we tested whether high defensiveness (rather than low anxiety and high defensiveness) could explain any coping group differences. No significant main effects of defensiveness were observed for any of the dependent measures.[7]

Once defensiveness was eliminated as an explanation for coping group differences, we then evaluated in greater detail whether repressors were different from the other coping groups. Evaluating whether one cell is different in a 2×2 design may lead the reader to expect a report of the interaction term in a factorial ANOVA. However, this interaction term, because it protects against all types of interactions, does not appropriately address the hypothesis of interest: Are repressors different from the other three coping groups? The preferred solution to evaluating this ordinal interaction involves a series of planned comparisons (Bobko, 1986). Contrast 1 tests the equality of the three nonrepressor groups for each of the dependent measures. Contrast 2 compares repressors with the average of the three nonrepressor groups. Significance for Contrast 2, in the presence of

[7]Immunological, lipid, and catecholamine responses were analyzed using a 2×2 (Anxiety: High or Low $\times$ Social Desirability: High or Low) factorial ANOVA. There were no significant main effects or interactions ($ps > .10$), with the exception of two anxiety main effects on CD8 ($p = .06$) and CD19 ($p < .05$) cells. Low-anxious individuals had significantly lower numbers of these cells than did high-anxious individuals.

Table 1

Mean Baseline Characteristics of Repressors and Nonrepressors

	Age (*SD*)	BMI (*SD*)	Smokers	Physical Activity (*SD*)	Anxiety[a]	Marlowe-Crowne[a] (*SD*)
Repressors ($n = 24$)	20.5 (1.5)	23.9 (2.8)	12.5% (3/24)	4,492 (3,077)	14.0 (2.4)	18.0 (3.0)
Nonrepressors ($n = 59$)	20.1 (1.6)	23.8 (2.9)	20.3% (12/59)	3,503 (2,374)	20.2 (4.3)	11.8 (4.4)

Note. BMI = body mass index (weight in kg/height in m^2); Smokers = proportion of current smokers; Physical activity = kcal/week; Anxiety = Spielberger Trait Anxiety Inventory (Spielberger, 1983) score; Marlowe-Crowne = Marlowe-Crowne Social Desirability scale (Crowne & Marlowe, 1960) score.

[a]Scores in this column differ at $p < .001$.

null findings for the first contrast, allows for the inference of this disordinal interaction. The first set of contrasts is a validity check to ensure that the repressor/nonrepressor contrast adequately characterizes any differences among the four coping groups (Bobko, 1986).

One-way ANOVAs (Contrast 1) reveal that the three nonrepressor groups were equivalent across lipid, immune, and catecholamine measures. Given the lack of main effects for the defensiveness measure, and the equivalence of the nonrepressor categories, it was appropriate to adopt a typological analysis (compare repressors vs. nonrepressors) to evaluate coping-style differences (Weinberger & Schwartz, 1990; see also Barger et al., 1997). The remainder of the analyses presented are comparisons of repressors to the average of the three nonrepressor groups. There were 24 repressors and 59 nonrepressors. The two repressive coping categories were comparable with respect to age, body mass index, physical activity, and smoking (all $ps > .25$; Table 1). Smoking status was a covariate in all analyses, given that several of the dependent measures are sensitive to cigarette smoking.

Baseline Measures

Immune measures. Enumerative and functional components of cellular immunity were compared among repressors and nonrepressors at baseline. Repressors had significantly fewer circulating CD4 cells, $F(1, 80) = 4.55$, $p < .05$; and significantly greater numbers of NK cells, $F(1, 79) = 4.55$, $p < .05$

(Table 2). There were no differences between repressors and nonrepressors in the circulating number of CD3, CD8, or CD19 cells (all $ps > .13$) or in baseline proliferative response to PHA ($p > .70$).

Lipids and catecholamines. Repressors had significantly lower levels of HDL, $F(1,77) = 4.13$, $p = .05$; and higher levels of insulin, $F(1, 73) = 7.19$, $p < .01$ (Table 3). Repressors' total cholesterol/HDL cholesterol ratio tended to be greater than that of nonrepressors (3.8 and 3.4, respectively), $F(1, 77) = 3.58$, $p = .06$. There were no differences in triglycerides, total cholesterol, LDL cholesterol ($ps > .20$), or basal plasma epinephrine or norepinephrine ($ps > .35$).

Cardiovascular measures. Systolic and diastolic BP values were virtually identical for both groups ($ps > .50$). Mean BP across both groups was 121/63 mmHg. Repressors' resting heart rate was higher than that of nonrepressors ($Ms = 68.7$ BPM and 65.0 BPM, respectively), $F(1, 79) = 4.39$, $p < .05$.

Responses to the Stroop Task

Task-related changes in immune components were compared between repressors and nonrepressors. For this purpose, each immunological, neuroendocrine, and cardiovascular response to the task was regressed on its baseline, and the residualized change score was calculated.

Immune measures. Between-group effects were significant only for number of NK cells, $F(1, 80) = 4.21$, $p < .05$. Following the Stroop task, repressors showed a less marked rise in the number of circulating NK cells compared to nonrepressors (Table 2). Baseline to posttask changes in other immune measures did not differ between repressors and nonrepressors ($ps > .20$).

Catecholamines. For all participants, epinephrine and norepinephrine increased as a result of the task: M epinephrine change = +11.5 pg/ml, $F(1, 78) = 18.5$, $p < .001$; mean norepinephrine change = +46.0 pg/ml, $F(1, 78) = 46.7$, $p < .001$. Between repressive groups, epinephrine change was not significant ($p > .80$). Repressors' norepinephrine increased relative to nonrepressors (adjusted mean changes +20.0 pg/ml and +0.25 pg/ml, respectively), but this change did not achieve statistical significance ($p < .20$).

Cardiovascular measures. Analysis of residualized change scores revealed no differences in heart rate. Systolic and diastolic BP change were also unrelated to coping style (all $ps > .40$).

Discussion

The present investigation compared several blood measures of disease risk, such as lipids, insulin, catecholamines, and quantitative and functional parameters of cellular immunity among repressive and nonrepressive individuals. Basal levels of a variety of health-related variables as well as cellular immune changes

Table 2

Mean (Standard Deviation) Catecholamines and Circulating Immune Cell Numbers (cell/mm^3) for Repressors and Nonrepressors at Baseline and Following the Stroop Task

	CD3		CD4		CD8		CD19		NK[a]		PHA[b]		EPI		NE	
	Pre	Post	Pre[c]	Post	Pre	Post	Pre	Post	Pre[c]	Post[d]	Pre	Post	Pre	Post	Pre	Post
Repressors (n = 24)	1,105 (294)	1,083 (273)	649 (193)	633 (180)	479 (130)	519 (143)	204 (71)	197 (65)	220 (108)	273 (94)	91.9 (4.8)	85.9 (3.8)	36 (15)	49 (18)	185 (62)	251 (93)
Nonrepressors (n = 59)	1,199 (267)	1,203 (339)	751 (197)	719 (217)	515 (148)	587 (207)	231 (81)	223 (80)	168 (99)	265 (162)	88.4 (4.3)	84.6 (9.2)	40 (25)	51 (23)	206 (106)	245 (101)

Note. All values are adjusted for smoking. NK = natural killer cells; PHA = lymphocyte response to phytohemagglutinin; EPI = epinephrine; NE = norepinephrine.

[a]There were only 23 observations for NK cells among repressors. [b]These values are times 10^4. [c]Scores in this column differ at $p < .05$. [d]The magnitude of repressors' increase in NK cells was less than that of nonrepressors, $F(1, 80) = 4.2$, $p < .05$.

Table 3

Mean (Standard Deviation) Lipid, Insulin, and Triglyceride Measures for Repressors and Nonrepressors at Baseline

	CHOL[a]	HDL[b]	Total/HDL ratio[c]	LDL	Insulin[b] (mU/l)	TRG	HR	SBP	DBP
Repressors	153 (25.1)	41.1 (6.1)	3.8 (0.8)	93.8 (21.1)	11.2 (4.9)	91 (56.3)	68.7 (7.9)	122 (8.2)	63.8 (8.0)
Nonrepressors	149 (26.1)	45.3 (8.9)	3.4 (0.9)	86.3 (24.6)	8.6 (3.5)	84 (47.4)	65.0 (8.0)	121 (8.2)	62.5 (8.3)

Note. All values are adjusted for smoking. CHOL = total serum cholesterol; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TRG = triglycerides; HR = heart rate in beats per minute; SBP = systolic blood pressure; DBP = diastolic blood pressure.

[a]All cholesterol and triglyceride measures were obtained following a 12-hr fast. [b]Scores in this column differ at $p < .05$. [c]Scores in this column differ at $p = .06$.

to a laboratory stressor were examined. In this sample of healthy college-aged males, individuals who employed a repressive coping style had lower levels of HDL cholesterol, higher insulin and a higher total cholesterol/HDL ratio, fewer circulating T-helper (CD4) cells, and higher numbers of NK cells than their nonrepressive counterparts. In addition, when compared with nonrepressive individuals, repressors demonstrated an attenuated increase in numbers of circulating NK cells following acute laboratory stress. Thus, the present study replicated a pattern of basal immune alterations (Jamner et al., 1988) among repressors and extended these observations to laboratory stress.

Our observation that repressors have greater CHD risk (via lipids and insulin) is comparable to earlier work (Niaura et al., 1992). However, the risk in Niaura et al. was attributable to elevated total cholesterol, while we found a rather different constellation of risk-factor differences between repressors and nonrepressors. This may be because of a relatively older sample ($M = 30$ years) or may be a function of the smaller number of males in the Niaura et al. study ($n = 39$; 12 repressors). Future research is needed to evaluate the consistency and pattern of CHD risk factors among repressors.

Repressors' lipid and insulin characteristics observed in the present study are indicative of increased CHD risk. Repressors had lower HDL cholesterol levels and a higher total cholesterol/HDL ratio, both of which are associated with increased CHD risk (Gordon et al., 1989; Kinosian et al., 1994; O'Keefe, 1995). CHD risk is positively and continuously associated with total cholesterol/HDL

ratios, but both repressors and nonrepressors would be classified similarly using the risk strata described by Kinosian et al. Thus, the precise clinical implications of the cholesterol ratio differences observed in the present study are difficult to quantify. However, it is important to note that including the total cholesterol/HDL ratio substantially improves CHD risk prediction relative to total cholesterol or LDL cholesterol levels alone (Kinosian et al., 1989).

HDL cholesterol levels are somewhat simpler to interpret in this context. Each 1 mg/dl increase in HDL cholesterol is associated with a 2% reduction in CHD risk (Gordon et al., 1989), so repressors in the current study would have roughly an 8% greater CHD risk than would nonrepressors. Modest elevations in fasting serum insulin, as noted in repressive males in this sample, are widely felt to reflect insulin resistance (Reaven, Lithell, & Landsberg, 1996). Insulin resistance, in turn, is associated with dyslipidemia and early cardiovascular disease, and often portends development of Type II diabetes mellitus. Elevated insulin is also independently associated with increased risk of coronary artery disease (Després et al., 1996). Thus, several metabolic risk factors for heart disease are elevated for repressors in this sample.

Blood pressure has a continuous positive association with CHD risk, but elevated resting BP has been observed inconsistently among repressors (King et al., 1990; Melamed, 1996; but not Newton & Contrada, 1992). Moreover, patterns of increased BP are not consistent, with studies showing elevations in either systolic (King et al., 1990) or diastolic BP (Melamed, 1996). If elevated BP is indeed a characteristic of repressors, it may present only in relatively older individuals (King et al., 1990; Melamed, 1996).

Given the apparent elevation of CHD risk among repressors, one might expect that repressors are more common within CHD patient populations. Two investigations of male cardiac patients do not support this hypothesis (Denollet, 1991; Denollet & DePotter, 1992). However, a recent report found that repressive coping is associated prospectively with survival among cardiac patients (88% male), controlling for exercise tolerance and disease severity (Denollet, 1999). Thus, there is some evidence for greater cardiovascular disease risk among young male repressors via higher cholesterol, and older repressors via elevated BP, and a potential link between repressive coping and cardiac mortality among clinical cardiovascular patients.

Changes in BP and heart rate among coping groups are worthy of further scrutiny. In the current study, no coping-style differences in heart rate or BP reactivity were observed, apparently contradicting earlier research.[8] This lack of reactivity may reflect a limitation of the classification scheme, or a limitation of cardiovascular measures to discriminate among coping styles. Careful examination of the literature reveals that exaggerated cardiovascular reactivity among repressors is

[8]We are grateful to two anonymous reviewers for bringing this issue to our attention.

inconsistent, lending credence to the latter explanation. For BP, one study found increased reactivity (King et al., 1990), while another did not (Newton & Contrada, 1992). Similarly for heart rate, three studies failed to find increased reactivity among repressors (Barger et al., 1997; Tomaka et al., 1992; Weinberger et al., 1979); another study showed that repressors, high-anxious, and defensive high-anxious groups were similarly reactive, and more so than low-anxious individuals (Asendorpf & Scherer, 1983); while only one study found that repressors were more heart-rate reactive than were other coping groups (Newton & Contrada, 1992). Thus, heart rate and BP have relatively poor discrimination with regard to repressive coping, and "cardiovascularly reactive" is an imprecise characterization of physiological changes reported in the literature.

There are several potential explanations for this inconsistency. Heart rate is determined by both parasympathetic and sympathetic branches of the autonomic nervous system, and these competing influences may render heart rate change less precise across studies. In addition, unmeasured variability in the experimental social context may also contribute to the unreliability of autonomic measures (Cacioppo, Rourke, Marshall-Goodell, Tassinary, & Baron, 1990). The autonomic measure that consistently identifies repressors as most reactive appears to be electrodermal activity (Barger et al., 1997; Weinberger et al., 1979; see also Gudjonsson, 1981). Using spontaneous electrodermal responses, repressors are the most reactive, regardless of whether there are two (Weinberger et al., 1979) or three (Barger et al., 1997) other coping groups for comparison. Thus, apparent unreliability in reactivity among repressors may also be a result of the choice of physiological measure.

With regard to the immune measures, we observed quantitative immune differences between repressors and nonrepressors, with the former having lower numbers of T-helper and higher numbers of NK cells in general circulation. It should be noted that despite these differences, all cell subtype numbers fell within normal ranges, and hence differences between repressors and nonrepressors may not be clinically significant in this young healthy population. However, T-helper and NK cells play a critical role in host defense against immune-related disease, and such changes may portend risk in populations that are already immunosuppressed (e.g., the elderly, HIV immunosuppressed; cf. Burcham et al., 1991). In these cases, individual differences in the magnitude of immune reactivity associated with repressive coping may be clinically relevant.

One possible explanation for immune differences between repressor categories is differences in SNS activity. It is widely accepted that the SNS modulates components of cellular immunity, including circulating numbers of NK cells, under conditions of acute stress (Bachen et al., 1995; Benschop et al., 1994). SNS activation, indexed by electrodermal activity, provides the best autonomic discrimination between repressors and nonrepressors (Barger et al., 1997; Weinberger et al., 1979). Given that the extant literature demonstrates that

increases in NK cells accompany activation of the SNS, it was expected that repressors who display exaggerated autonomic arousal following stress would show greater increases in circulating numbers of this cell subtype. Indeed, it is not readily explainable why we observed attenuated NK cell responses in this group. It is known that catecholamines are released in pulsatile fashion during stress and hence our discrete catecholamine sampling (three times) may not have reliably captured this response. It is also possible that the psychological demands of the Stroop task did not cause the same manner of SNS activity elicited by other laboratory tasks. Additional research evaluating repressors' SNS activity, task characteristics, and immunological responses to acute stress would be welcome.

Repressive coping is commonly conceptualized as active inhibition of negative affect (Brown et al., 1996; Schwartz & Kline, 1995), and this putative inhibition appears to be the most likely link between repressive coping and health. Repressive coping, conceptualized as chronic inhibition, could alter cardiovascular risk factors in the pattern observed here. Evidence from a study by Weinberger and Davidson (1994) suggests that repressors' responses to negative emotion can reasonably be characterized as chronic. They found that repressors were unresponsive to social demands for emotional disclosure, and appeared to be more invested in "maintaining a view of themselves as *not* emotionally reactive" (Weinberger & Davidson, 1994, p. 604, emphasis in original). This is congruent with repressors' phenomenological self-descriptions (Weinberger, 1990) and peer descriptions of individuals high in defensiveness (McCrae & Costa, 1983). Thus, we can plausibly conclude that repressors' emotional control occurs across situations, rather than in response to circumstance.

This chronic inhibition of emotion, with correspondent elevation in autonomic activity, suggests that metabolic factors associated with stress could be pathways through which repressive coping alters health risk. For example, stress-induced changes in cortisol (e.g., Brown et al., 1996) and fatty acids may lead to the constellation of higher insulin and low HDL observed in this sample (Brindley, McCann, Niaura, Stoney, & Suarez, 1993). This metabolic mechanism is also associated with hypertension, which has been observed among older samples of repressors (King et al., 1990). It may be that this metabolic disturbance develops into hypertension as repressors age, but studies of repressive coping using older samples are rare. One assumption of this speculation is that repressors experience regular episodes associated with negative emotion and, to date, little evidence exists regarding this important antecedent (for an exception, see Myers & Brewin, 1994). Finally, because diet and smoking can alter the metabolic pathways discussed earlier, future investigation of these relations should explicitly include these well-known moderators.

Before it can be concluded that these findings have general health implications, they need to be replicated and extended to subject samples with different demographic characteristics (e.g., females, non-Whites). In addition, because of a

relatively large number of statistical comparisons, any particular difference between repressors and nonrepressors should be considered cautiously. On the other hand, the pattern of differences, especially for the cardiovascular risk factors, has an interpretive consistency that would not be expected if the differences were a result of chance alone. Indeed, the detection of statistically significant differences among important health risk factors (such as lipids and insulin) among this group of active, healthy, young adults is notable, and we believe is worthy of additional investigation.

This study lends further credence to the idea that repressive coping is a useful personality construct to identify individuals with alterations in health-relevant indexes, despite equivocal evidence for actual disease risk. These differences are especially noteworthy because of the general good health of our sample and because repressive coping classification using a median split on the Marlowe-Crowne scale (Crowne & Marlowe, 1960) provides a less robust contrast between groups. Altered immune characteristics have now been identified among repressors in three different studies, while increased cardiovascular disease risk vis-á-vis serum cholesterol level has been noted here and elsewhere (Niaura et al., 1992). Future research should evaluate the consistency of increased CHD risk factors among repressors and potential mechanisms through which immune alterations may occur, such as cortisol and exaggerated sympathetic nervous activity. A more ambitious program would evaluate repressive coping, physiological mechanisms, and disease susceptibility in a single study. Exploring this combination of autonomic, immunological, neuroendocrine, and health measures will help disentagle these concomitants of coping with stress.

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