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Emotion Suppression and Mortality Risk Over a 12-Year Follow-up

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Abstract

Objective—Suppression of emotion has long been suspected to have a role in health, but empirical work has yielded mixed findings. We examined the association between emotion suppression and all-cause, cardiovascular, and cancer mortality over 12 years of follow-up in a nationally representative US sample.

Methods—We used the 2008 General Social Survey-National Death Index (NDI) cohort, which included an emotion suppression scale administered to 729 people in 1996. Prospective mortality follow up between 1996 and 2008 of 111 deaths (37 by cardiovascular disease, 34 by cancer) was evaluated using Cox proportional hazards models adjusted for age, gender, education, and minority race/ethnicity.

Results—The 75th vs. 25th percentile on the emotional suppression score was associated with hazard ratio (HR) of 1.35 (95% Confidence Interval [95% CI] = 1.00, 1.82; p = .049) for all-cause mortality. For cancer and cardiovascular disease mortality, the HRs were 1.70 (95% CI = 1.01, 2.88, p = 0.049) and 1.47 (95% CI = .87, 2.47, p = 0.148) respectively.

Conclusions—Emotion suppression may convey risk for earlier death, including death from cancer. Further work is needed to better understand the biopsychosocial mechanisms for this risk, as well as the nature of associations between suppression and different forms of mortality.

Keywords

Emotion; Suppression; All-cause mortality; Cancer mortality; Cardiovascular disease mortality; General Social Survey

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Background

Emotion suppression, defined as a tendency to inhibit the expression of emotion (1), has long been suspected to influence health (2), with recent meta-analytic evidence linking suppression and chronic disease supportive of this long-held notion (3). Emotion suppression involves intentionally avoiding distressing feelings by thinking of other things or holding things in, while emotion repression is defined by lack of conscious awareness of negative emotion (4, 5).

Suppression is believed to operate on health first at a behavioral level, by inducing unhealthy coping behaviors such as over-eating as substitutes for healthy emotional expression (6). Second, at a physiological level, higher levels of autonomic reactivity to stress--measured both electrodermally and through blood pressure changes--have been reported among suppressors (7). Direct correlations between suppressive defensive styles and both catecholamines and glucocorticoids have also been reported (8, 9) and are reviewed in (10) and (11). In turn, neuroendocrine dysregulation, whether induced by stress processes or habitual health-damaging behaviors, has been implicated in the progression of a number of chronic diseases, and ultimately earlier death (12).

Epidemiologic evidence for links between suppression and mortality appeared initially in a Yugoslavian cohort study conducted in the 1970 by Grossarth-Maticeck (13). Specifically, a suppression-prone personality style called “anti-emotionality” predicted 10-year all-cause and Cardiovascular Disease (CVD) mortality (13). In other work, Grossarth-Maticeck noted associations between this personality style and cancer death (13, 14). Grossarth-Maticeck’s studies were subsequently the center of controversy around data collection and analysis (15–24), although he and his collaborator Hans Eysenck vigorously defended the results (25). “Type C” personality style—also defined by a tendency to suppress emotion—was linked to poor health outcomes in the 1980s (26–28). In the 1990s “Type D” personality, which involves affective distress in conjunction with social inhibition (presumably limiting emotional disclosure to others), was linked to both CVD death and numerous other health problems (29). Other studies on the suppression of anger in particular have noted increased all-cause mortality over 17 years in a US community sample (30), 6 years in Dutch CVD patients (31), and 8.5 years in a German sample (32). Yet there may also be a strong cultural contingency of suppression effects: in a Japanese community sample, lower levels of emotion suppression were linked to worse health (33), and in Japanese cancer patients, moderate, rather than high or low suppression levels were associated with survival (34). The construct of repression conveyed a survival advantage in male veterans of the American army followed for 16 years (35), hinting at differential acculturation and gender variability in mortality risk of constructs in the suppression/repression family (6).

In short, while studies on the mortality risk of emotion suppression have been suggestive, they have been far from definitive, underscoring the need for new data in broadly diverse population samples. Our primary aim was to examine whether emotion suppression was associated with death from any cause over a 12-year follow-up in a nationally-representative US sample. We also assessed links between emotion suppression and the two leading specific causes of death in the US, CVD and cancer. Finally, we explored whether suppression of anger in particular (30), or other indicators of more specific types of suppression, were linked to mortality.

Methods

Sample and Design

The General Social Survey (GSS) is an annual study of opinions and attitudes among the US public that is conducted by the National Opinion Research Center (NORC) at the University of Chicago. The survey uses a multi-stage probability sampling of non-institutionalized adults age 18 and over, with response rates from 70% to 82% in any given year (36). Interviews are conducted in person and involve a core set of questions asked every year (note that different people are included each year, so the survey is not a repeated-measures study). Each year, additional questions are also added for a representative subset of the panel. In 1996, the GSS Emotion Suppression Scale was administered to 737 respondents. Recently, GSS data from several years (including 1996) were linked to US National Death Index (NDI) records through 2008 (36), the standard national database for determining vital status in the US.

Measures

In addition to questions about social issues, the GSS records age, gender, race/ethnicity, and years of education on the basis of interviewer observation and subject report. The GSS' Emotion Suppression Scale (37) includes six items constructed using a face- and content-validity measurement approach (38). The scale items are: 1) "I keep my emotions to myself", 2) "I'm not afraid to let people know my feelings" (reverse scored), 3) "When I'm angry I let people know" (reverse scored), 4) "I often don't tell my friends something that I think will upset them", 5) "I try to be pleasant so that others won't get upset", and 6) "When I'm anxious I try not to worry anyone else". Responses are made on a 5-point Likert scale ranging from "Strongly Disagree" to "Strongly Agree", with items scored so that higher values indicated greater suppression. Construct validity evidence arises from associations between the scale and demographic and social factors (i.e., men suppress emotions more than women) (37, 39, 40). In our sample, Bentler's composite internal consistency reliability (41) was .70. Scale scores consisted of the mean across items (reverse coding the necessary items).

Vital status through 2008 was ascertained from the National Death Index (NDI). The validity of mortality records from the NDI is typically high, with true-positives achieved from social security numbers and the additional identifiers (used in the GSS matching process) reaching 99.8% (42). Cause of death was determined by collapsing International Classification of Disease-9 records into 285 mutually exclusive categories using the Clinical Classification Software System (CCS) (36). This was used to construct 3 outcomes: 1) death from any cause; 2) cancer death, consisting of any CCS cancer category; and 3) CVD death, including CCS categories pertaining to myocardial infarction, congestive heart failure, hypertension, coronary heart disease, and stroke. It was not possible to examine finer-grained categories due to prohibitively small numbers of deaths within each category. Further details on the GSS-NDI matching are available in (36).

Analysis

We examined the associations between emotion suppression and each mortality outcome using Cox proportional hazards models. A number of views exist on model building and confounder control. Most agree that crude associations reflect an indeterminate amount of negative (the unadjusted association is too large) or positive confounding (the unadjusted association is too small) and, in this case, provide biased estimates of the association in question (43). One view holds that each potential confounder should first be screened for bivariate associations with both a) the exposure, and b) the outcome, and included in the model only if such associations are apparent (44). The difficulty with this is that non-

significant associations may exist which still confound the estimate of interest substantially. As well, the bivariate associations between a confounder, exposure, and outcome may themselves be confounded in either direction by another variable, which cannot be detected in bivariate screening. As a result, every time a covariate is added, the covariance structure of the predictors can changes, leading to data-driven searches over all possible subsets of confounders. A second alternative holds that any factors which lead to a 10% or greater change in the estimate of interest (in our case, the relative risk of emotion suppression) should be included (45). Some have pointed out that this depends on the order in which variables are entered, and that 10% changes may not place an adjusted estimate outside the 95% confidence interval of a less adjusted estimate, possibly lead to decisions based on random sampling variation (45). Yet another view, mainly used in clinical trials, holds that only factors associated with the outcome should be included because in some cases, this type of adjustment can increase the estimation efficiency (i.e., reduce the standard error) of the effect of interest (46). A fourth and final view holds that one should simply select covariates a priori, based on theory (particularly sociodemographic factors in health research) (47). Although this view too has its limitations, we prefer it in this case because there is sufficient theory and empirical results to guide a priori confounder selection. We thus controlled for the following pool of possible confounders, based on known associations with mortality risk and known (40) or potential associations with the suppression scale: age, sex, minority race, education level, and self-rated health. We did not include “mediators”—that is, variables resulting from emotional suppression, and preceding mortality on the causal pathway, since our goal was to estimate the total (i.e., direct plus indirect) mortality risk associated with suppression (47). Model diagnostics screened for violations of proportional hazards, interactions among predictors, and curvilinearity of associations (48).

Since the GSS emotion suppression measure is a continuum but defined in arbitrary Likert-scale type units, scores were scaled so that a one unit difference reflected the interquartile range, namely the difference between the 75^{th} and 25^{th} percentile of the distribution, or the interquartile range metric often used for exposure in epidemiologic research. This scaling provides an interpretable metric for “unitless scales” while keeping them continuous, i.e. not discarding information by actually categorizing into quartiles (48). Exploratory analyses also examined the mortality risk associated with response to the anger question (#3) and other individual items. Sensitivity analyses removed all deaths within the first year to exert additional control for baseline health.

Results

Table 1 shows the sample demographics, which were similar to the 1996 US population estimates from the decennial census (49). Mortality rates were in line with trends for that period reported by the Centers for Disease Control (50). The CVD death category was dominated by myocardial infarction, coronary atherosclerosis, and other or ill-defined heart disease (together, 58% of CVD deaths). Leukemia, lung, pancreatic, and colon cancer accounted for 47% of the cancer deaths. The GSS emotion suppression scale scores were roughly normally distributed, suggesting items succeeded in capturing information well over the range of suppression present in the population.

Table 2 shows the results of models for the first aim of quantifying the impact of emotion suppression on mortality risk. Emotion suppression at the 75^{th} vs. 25^{th} percentile conveyed a significant elevation in risk (HR = 1.35, or a 35% increase) of death from any cause. This was comparable to the increase in mortality risk observed for 3.1 years of life expectancy (HR = 1.35). The same difference in suppression was associated with a 70% increase in risk (HR = 1.70) of death from cancer (a 5.6 year difference in life expectancy). A non-significant elevation in risk was noted for CVD death. Removing deaths within the 1^{st} year

amplified the effect of suppression (HRs (95% CIs) = 1.42 (1.04, 1.93), p = 0.028 for all-cause mortality; 2.08 (1.16, 3.75), p = 0.013 for cancer death). Suppression risk appeared proportional over time and a weak trend suggested suppression might confer greater risk for all-cause mortality in women (p = 0.115 for an interaction with gender), but this was not present for cancer or CVD death, and no other interactions or curvilinear associations were noted.

In exploratory analyses, those reporting higher anger suppression exhibited elevations in mortality risk (HR (95% CI) for 1 Likert point increase = 1.21 (1.02–1.43), p = 0.029 for all cause; 1.44 (1.03, 2.00), p = 0.031 for cancer; 1.43 (1.06, 1.91), p = 0.018 for CVD). Cancer death risk was significantly higher among those disagreeing more with “I’m not afraid to let people know my feelings” (that is, those reporting greater suppression; HR = 1.26, 95% CI = 1.04, 1.52, p = 0.017 for 1 Likert point shift).

Conclusions

Our analysis of a US nationally representative sample, followed for 12 years for mortality by cause of death, revealed significant associations between higher levels of emotion suppression and all-cause as well as cancer-related mortality. These findings have several implications. Theoretically, suppression is presumed to promote unhealthy behaviors as a substitute for appropriate emotional expression, and possibly engender neuroendocrine dysregulation (2, 51). However, whether any such biological costs are strong enough to ultimately influence mortality risk has been less certain. Our results contribute to the weight of evidence that the effects of suppression are detectable far down the progression of lifecourse health pathways, at their final common endpoint.

Psychosomatic theory and data have also suggested that emotion suppression may be implicated in cancer death, operating either through disease onset and/or course (14, 26, 52, 53), a hypothesis with which our findings are consistent. Grossarth-Maticeck defined suppression in terms of a “rationality-antiemotionality” personality tendency related to need for control, and reported that it had a potential role in cancer onset and/or survival (54). Observational studies subsequently lent some empirical support to the idea (55–57), although meta-analytic conclusions of a negligible association (58) sparked controversy further debate over the issue (59). Similarly, trials of supportive-expressive therapy, designed to reduce suppression, have yielded both positive (52, 53) and more ambiguous findings (60, 61) with respect to cancer survival (see also (62–64)). Thus, findings both with respect to incidence and death in the population (15–23), as well as survival in those with cancer (61) have been debated. Our results concern cancer mortality in the population—that is, both onset, i.e. incidence and course of the disease.

Findings for CVD death were in a direction and of a magnitude consistent with prior work (13, 30, 32), though they did not reach statistical significance. Exploratory item analysis showed that increasing disagreement with the “When I’m angry I let people know” increased risk across all three outcomes, and increasing disagreement with “I’m not afraid to let people know my feelings” elevated cancer mortality risk. Both items make reference to others, underscoring the notion that emotional expression is fundamentally an interpersonal activity, although it can be done intrapersonally via writing exercises (65–67). Thus, these findings suggest that the capacity to reduce or relieve threatening or burdensome affects by disclosure to (and processing with) others may be related to the broad health-protective effects attached to social support (68). Persons who are reluctant to disclose their emotions to others may not elicit empathic responses from others. At the same time, disclosure is also incentivized or disincentivized by social environments, so suppression must be interpreted within a person’s particular social context and history.

Based on notions such as these, some have suggested interventions to reduce suppression. Studies of supportive-expressive therapy indicate that suppression and distress it invokes can be reduced (63, 64), while other work has shown salutary immune and neuroendocrine effects of expressive writing (65–67). Care must be exercised in deciding whether the present findings support the use of such interventions to explicitly reduce mortality. It is possible, for instance, that suppression is associated only with the onset of cancer, in which case initiating supportive-expressive therapy for those diagnosed with cancer may not be helpful. Conversely, disease prevention efforts will not be useful if suppression is implicated only in the course of cancer or other diseases, rather than its onset. However, treatments such as supportive-expressive therapy may yield improved quality of life by reducing distress regardless of whether or not they influence survival.

Conclusions must also be informed by a balanced analysis of other study strengths and limitations. The study involved a nationally representative sample, maximizing ecological validity for its target population, but there were only 34 cancer and 37 CVD deaths, pointing to the need for cautious conclusions and further replication. Nevertheless, our power was sufficient to detect modest to large associations for 2 of the 3 mortality outcomes. Future work might implement larger samples, which would also permit examination of finer-grained types of mortality and afford greater power for interactions with gender and social environmental moderators of suppression risk (6). Our item analysis was also exploratory, and intended to inform future work rather than formally test a priori hypotheses. We also examined suppression with a measure focusing on suppression of disclosure of emotion, and it is not clear to what extent findings may extend to repression. There is a clear conceptual distinction between suppression and repression, and based on that body of theory, one would not necessarily expect findings from one to reflect the other. Nevertheless, empirical overlap may occur due to similar measurement, i.e., scales that cannot completely distinguish the two processes. Suppression is also similar to, but distinct, from alexithymia which has also been linked to mortality (69, 70): the latter denotes an inability to label, verbalize or communicate one's emotional experience (71), rather than a willingness or desire to suppress it. The concept of *emotion inhibition* has also been treated virtually synonymously with suppression (6). We suspect that measures of these constructs may behave similarly in an epidemiologic context, although a conservative interpretation of our findings would restrict generalization to suppression per se. It is also unknown whether suppression is functioning as a proxy for some other psychosocial factor linked to mortality or for a health factor not captured by self-rated general health.

We also did not examine mechanisms for the association between suppression and mortality. An important future direction of research would entail a randomized trial of interventions to reduce emotional suppression to better define the causal link between emotional suppression, health, and mortality. Such a study would also permit a better understanding of the underlying behavioral and physiological (72) mechanisms linking emotional suppression to mortality. For instance, the suppression / repression family of constructs might result in persistent deleterious HPA activity, or persons with systems prone to such activity might also be prone to emotional suppression (8, 9). It is also possible that emotion suppression, particularly in extreme form, may reflect underlying psychopathology that is the true source of the mortality risk. Identification of specific mechanisms, e.g. distinguishing between intrapersonal emotional expression and interpersonal emotional expression, will facilitate design of interventional research to improve health.

Finally, we caution against interpretations of these findings—particular the cancer mortality risk--as indicative of an overly specific link between a particular psychological phenomenon, and a particular cause of death. This so-called "doctrine of specific etiology" arose from the work of Franz Alexander and undergirded earlier psychosomatic theory (13,

73), but posits a degree of specificity rarely observed, and our results are not particularly supportive of it. Similarly, generalizations to populations differing from the general US public, or beyond a 12-year follow-up span, must be resisted. Due to lower power, the study likely errors on the side of Type II error (i.e., missing an association) for associations of smaller magnitude. Study strengths included a nationally representative sample, a follow-up period of 12 years, and sufficient death rates to detect effects of a clinically meaningful size.

In conclusion, our findings suggest that emotion suppression warrants more detailed investigation as a possible mortality risk. It is a construct falling outside of many personality-based studies of longevity (74), yet as a coping or defensive process, might be considered an integral part of, rather than simply a product, of personality (1, 75). In addition to pursuing further evidence for basic mortality associations, future work can further delineate the biopsychosocial pathways through which inhibiting emotional expression leads to earlier death.

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Table 1

Descriptive Statistics: Demographics and Cause of Deaths

Variable	Mean / N	SD / %
Age in 1996	44.0	16.5
Education (Years)	13.5	2.9
Female Gender	395	54%
White	593	81%
Black	102	14%
Other Minority	34	5%
Emotion Suppression*	2.9	.6
Self-Rated Health**	2.0	.8
Death, Any Cause, by 2008	111	15%
CVD Death by 2008	37	5%
Cancer Death by 2008	34	5%

Note. N = 729.

* Mean score on scale items, ranging from 1 to 5. A 1-unit increase corresponds to 1 Likert scale point (i.e., from 1, strongly disagree to 2, disagree) increase in agreement on average to admission of emotion suppression behavior.

** Mean score on scale of 1 (excellent) 2 (good) 3 (fair) 4 (poor).

Table 2

Multivariate Survival Models

	All-Cause Mortality (N=111)			Cancer Mortality (N=34)			CVD Mortality (N=37)		
	HR	**95% CI**	**P**	**HR**	**95% CI**	**P**	**HR**	**95% CI**	**P**
Age (Decades)	1.98	[1.73, 2.26]	<.001	2.19	[1.70, 2.81]	<.001	2.36	[1.84, 3.02]	<.001
Female Gender	.78	[.54, 1.14]	.219	.64	[.32, 1.27]	.202	1.04	[.54, 2.01]	.897
Black Race/Ethnicity	1.06	[.60, 1.88]	.339	.62	[.18, 2.13]	.446	.73	[.24, 2.19]	.571
Other Race/Ethnicity	2.95	[1.03, 8.47]	.043	2.75	[.34, 22.13]	.340	3.60	[.44, 29.44]	.231
Education (Years)	1.00	[.88, 1.06]	.881	.98	[.87, 1.10]	.680	.93	[.83, 1.05]	.241
Self rated health	1.55	[1.20, 1.99]	<.001	1.56	[1.00, 2.46]	.050	1.35	[.87, 2.11]	.184
Emotion Suppression	1.35	[1.00, 1.82]	.049	1.70	[1.00, 2.88]	.049	1.47	[.87, 2.47]	.148

Note. HR = hazard ratio, 95% CI = 95% Confidence Intervals. Emotion suppression scaled by interquartile range, so HR corresponds to risk at 75^{th} vs. 25^{th} percentile of distribution. Self-rated health on 1–4 scale. #Estimate presented is from first five years of follow-up during which hazards are proportional, after which male-female (for all cause mortality) and minority-white (for CVD) risk ratios diminish then reverse. Inference for based on likelihood ratio test.