Emotional Expression and Health

Advances in theory, assessment and clinical applications

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Chapter 4

Emotion suppression and cardiovascular disease

Is hiding feelings bad for your heart?

Iris B. Mauss and James J. Gross

Theorists have long claimed that the free play of emotion is incompatible with civilization (Elias, 1978; Freud, 1961). There has been a lingering suspicion, however, that the emotion control required by civilization may come at a steep price. In the popular literature, this price has often been represented in terms of the adverse consequences emotion inhibition may have for physical health (e.g. Martin, 1998). In the scientific literature, too, there have been reports linking emotion inhibition to a variety of diseases (e.g. Friedman & Booth-Kewley, 1987; Pennebaker & Traue, 1993). For example, emotion inhibition has been implicated in asthma (Florin *et al.*, 1985; Teiramaa, 1978), cancer (Denollet, 1998; Greer & Watson, 1985; Gross, 1989; Temoshok, 1987), chronic pain disorders (Beutler *et al.*, 1986; Harrison, 1975; Udelman & Udelman, 1981) and cardiovascular diseases (Brosschot & Thayer, 1998; Haynes *et al.*, 1980; Jorgensen *et al.*, 1996; Julius *et al.*, 1986).

Despite a long history of popular and scientific interest in links between emotion inhibition and disease, the complexity of the processes involved in both emotion inhibition and disease has meant that clear conclusions have been hard to come by. Our strategy in this chapter is to focus our discussion in two ways. On the "input" side, we focus on one particularly common type of emotion inhibition, namely emotion suppression, or the inhibition of ongoing emotion-expressive behavior (Gross, 1998). On the "output" side, we focus on cardiovascular diseases. Cardiovascular diseases represent the single most important source of disability and mortality worldwide (Guyton & Hall, 1997; Murray & Lopez, 1997), and they typify the slow-developing, multifactorial disease processes that seem to be most influenced by psychosocial factors such as emotion suppression (Depue & Monroe, 1986; Sapolsky, 1998).

We begin with a selective review of the literature on emotion and cardiovascular disease. This literature suggests that intense emotional responses – whether expressed or suppressed – may play a role in the development and course of cardiovascular disease. To assess whether emotion suppression makes a unique contribution to cardiovascular functioning, we review a series of laboratory studies designed to specify the acute physiological consequences of emotion suppression. These studies show that emotion suppression leads to transient increases in

sympathetic activation of the cardiovascular system. Finally, to help bridge the gap between the long-term correlational studies discussed in the first section and the short-term experimental studies discussed in the second section, we offer several possible psychophysiological and psychosocial pathways by which emotion suppression might have a longer-term impact on cardiovascular health.

Emotion and cardiovascular disease

Efforts to link emotional factors to cardiovascular diseases date back to the earliest days of psychosomatic medicine (e.g. Alexander, 1939; Dunbar, 1935). These efforts were given new visibility and credibility when a pair of cardiologists, Friedman and Rosenman, observed that their coronary heart disease patients seemed to be unusually competitive, hard-driving, impatient, and hostile (Friedman & Rosenman, 1974). Friedman and Rosenman referred to this constellation of factors as the Type A behavior pattern. Their hypothesis that the Type A behavior pattern contributed to the development of cardiovascular disease was borne out by a large number of empirical findings, including several large prospective studies (see, for reviews, Booth-Kewley & Friedman, 1987; Cooper *et al.*, 1981; Matthews, 1988; Siegel, 1984).

Emotion experience and expression

Since it was first articulated, the Type A concept has been refined considerably. We now know that anger and hostility seem to be the core features of the Type A behavior pattern. For example, Booth-Kewley and Friedman (1987) found in their quantitative review that anger, hostility, and aggressiveness were significant predictors for coronary heart disease, with combined effect sizes larger than that of any of the other Type A components. Cross-sectional and prospective studies have confirmed that a high level of anger and hostility is a risk factor for the incidence and progression of CHD as well as hypertension (see, for review, Kubzansky & Kawachi, 2000; Matthews, 1988; Miller *et al.*, 1996; Smith, 1992). Adverse cardiovascular health outcomes have been found to be particularly pronounced for individuals who openly express their angry feelings (e.g. Harburg *et al.*, 1991; Hecker *et al.*, 1988; Siegman *et al.*, 1987).

More recently, it has become clear that in addition to anger and hostility, other negative emotions seem to be related to cardiovascular disease. Several studies have established that depression (Anda *et al.*, 1993; Barefoot, 1997; Musselman *et al.*, 1998), anxiety (Fleet & Beitman, 1998; Haines *et al.*, 1987; Kawachi *et al.*, 1994) and general emotional distress (Crisp *et al.*, 1984; Gullette *et al.*, 1997; Rosengren *et al.*, 1991) are important factors in the development and progression of coronary heart disease (for reviews, see Barefoot, 1997; Booth-Kewley & Friedman, 1987; Fielding, 1991; King, 1997; Kubzansky & Kawachi, 2000). Despite some negative findings (e.g. Hearn *et al.*, 1989; Helmer *et al.*, 1991; Leon *et al.*, 1988), a consensus has developed that the coronary-prone person experiences and expresses high levels of a variety of negative emotions. As Booth-Kewley and Friedman (1987) concluded, a person prone to coronary heart disease may be not simply a hostile person, but rather a person "with one or more negative emotions: perhaps someone who is depressed, aggressively competitive, easily frustrated, anxious, angry, or some combination" (p. 358).

Emotion suppression

One puzzle has emerged, however. While the studies we have reviewed show that the experience and *expression* of high levels of negative emotion lead to increased risk for cardiovascular disease, other studies have highlighted the role of emotion *suppression* (Brosschot & Thayer, 1998). Thus, Dembroski *et al.* (1985) found that hostility predicted severity of atherosclerosis only in patients who were also likely to suppress feelings of anger ("Anger-in"). Indeed, in several studies now, measures of the inhibition of negative emotions have been found to predict all-cause mortality as well as the incidence of coronary heart disease, hypertension, and related risk-factors (Gallacher *et al.*, 1999; Grossarth-Maticek *et al.*, 1985; Haynes *et al.*, 1980; Julius *et al.*, 1986; Julkunen, 1996; Manuck *et al.*, 1986; Schalling & Svensson, 1984). In their meta-analysis of the literature on emotion and blood pressure, Suls *et al.* (1995) found that anger suppression plays an important role in essential hypertension.

An important qualification recently has been added to this emerging suppression hypothesis, namely that the tendency to inhibit emotions *per se* might not be harmful, only the tendency to inhibit strong emotional impulses (e.g. Burns, 1995). For example, Cottington *et al.* (1986) found that workers with hypertension reported suppression *and* high levels of anger and stress. Denollet and coworkers have hypothesized that the tendency to suppress emotion-expressive behavior in social interactions might interact with the individual's level of emotional distress in determining the progression of coronary heart disease. To test this hypothesis, Denollet *et al.* divided subjects who had survived a myocardial infarction into four groups, depending on their negative affectivity (anxiety, anger, and chronic tension) and social inhibition (the tendency to inhibit the expression of emotions and distress in social interactions). The subgroup scoring high on both dimensions was labeled "Type D" (for *distressed*). It was found that Type D patients had a significantly higher death rate (27 percent) than non-Type D patients (7 percent) (Denollet *et al.*, 1996). It was pointed out that the death rate in patients scoring high on negative affectivity but low on inhibition did not differ significantly from the death rate in patients with low negative affectivity – therefore it was the *negative affectivity by social inhibition interaction* that had an adverse effect on prognosis.

In addition to having an effect on prognosis, this emotional style also seems to be involved in the *development* of risk factors for coronary heart disease. In a meta-analysis, Jorgensen and colleagues (1996) found that taken together, negative affectivity and affect inhibition were the strongest psychological predictors for the development of essential hypertension. Together, these studies suggest that it may

be the behavioral suppression of relatively high levels of negative emotions that contributes to the development and progression of cardiovascular disease.

So which is it . . . expression or suppression?

How are we to reconcile the literature which suggests that it is the *expression* of negative emotions that is pathogenic with the literature which suggests that it is the *suppression* of negative emotions that is pathogenic? If suppression is the opposite of expression, how can both be associated with cardiovascular diseases?

One possibility is that suppression and expression are not "opposites", but rather behaviors that *both* may result from the experience of intense negative emotions (Julius *et al.*, 1986; Siegman *et al.*, 1987). Indeed, researchers repeatedly have shown that Anger-in and Anger-out are independent from one another, rather than negatively correlated as lay intuition might suggest (Spielberger *et al.*, 1995; Weidner *et al.*, 1989). Individuals prone to the experience of negative emotions may try to cope with these emotions in different ways, at times inhibiting them and at times expressing them.

The natural confounding of intense negative emotions with emotion suppression in everyday life makes it difficult to assess clearly the unique contribution of emotion suppression to cardiovascular outcomes on the basis of correlational studies such as the ones reviewed in the previous section. To examine the contribution of emotion suppression over and above the contributions of emotion experience and expression, we embarked on a series of laboratory studies designed to assess the acute consequences of emotion suppression. By standardizing our emotion induction procedures, and by randomly assigning participants to either suppression or no suppression conditions, we sought to clarify whether emotion suppression *per se* had any observable impact on cardiovascular responding that might be consistent with longer-term health consequences.

The acute consequences of emotion suppression

Most of what we know about the acute effects of emotion suppression has come from the *facial feedback literature*. This literature has its origins in Darwin's (1872/1979) assertion that "the free expression by outward signs of an emotion intensifies it. On the other hand, the repression, as far as this is possible, of all outward signs softens our emotions" (p. 365). As the phrase "facial feedback" suggests, the organizing principle in this literature is that emotion-expressive behavior has feedback effects, such that increasing emotion-expressive behavior should increase the emotion, whereas decreasing emotion-expressive behavior should decrease the emotion (Buck, 1980). Many of the studies in this area have compared suppression with exaggeration (and not with "natural" expression), finding that exaggeration leads to larger responses than suppression. This comparison makes it difficult to discern whether exaggeration and suppression both increase physiological responding (with exaggeration providing the larger boost), or whether – as is typically suggested – suppression actually leads to decreased physiological responding. Unfortunately, studies that have included the critical comparison between suppression and natural responding have focused on positive emotions (such as amusement), and typically have limited themselves to examining the effects of suppression on subjective emotional experience.

Initial studies

To clarify the acute behavioral, experiential, and physiological consequences of suppressing negative emotion, in an initial pair of studies, we used a short film that showed an arm amputation to elicit disgust (Gross & Levenson, 1993). We administered specific instructions to participants who had been randomly assigned to one of two experimental conditions. Watch condition subjects received instructions to simply watch the film clip carefully. Suppression condition subjects received additional instructions to "try to behave in such a way that a person watching you would not know you were feeling anything".

Under normal circumstances, as shown in the top panel of Figure 4.1, subjects move around a bit more when they are watching a disgusting film than they do in the resting baseline. When they suppress, however, they stifle the natural increase in somatic activity associated with disgust, yielding a flatter line for the suppression subjects than for the watch subjects. In the bottom panel of Figure 4.1, we see that suppression subjects' decreased body movement is associated with a slowing of heart rate. Despite these decreases in body movement and heart rate, Figure 4.2 shows that suppression *increased* sympathetic activation. Increases in sympathetic activation are shown both by a theoretically derived composite of sympathetic activation of the cardiovascular system (created by standard scoring finger pulse amplitude, finger temperature, and pulse transit times to the finger and ear) and by skin conductance level (not depicted here).

Testing boundary conditions of emotion suppression

One puzzle is why results from the initial studies were at odds with the facial feedback literature. Why did suppressing disgust increase sympathetic activation? Might the physiological and experiential effects of emotion suppression vary by emotion?

To test the boundary conditions of the effects of emotion suppression, we examined a second negative emotion – sadness. Given widespread agreement that positive emotions serve quite different functions from negative emotions, we also examined a positive emotion, namely amusement. To rule out the possibility that our initial findings were an artifact of the particular suppression instructions we employed, we also gave subjects the same suppression instructions during an affectively neutral film, when there would presumably be no emotion-expressive behavior to suppress (Gross & Levenson, 1997).

We found that watching a sad film leads to decreases in overall body movement. Watching an amusing film, by contrast, leads to increased body movement, as

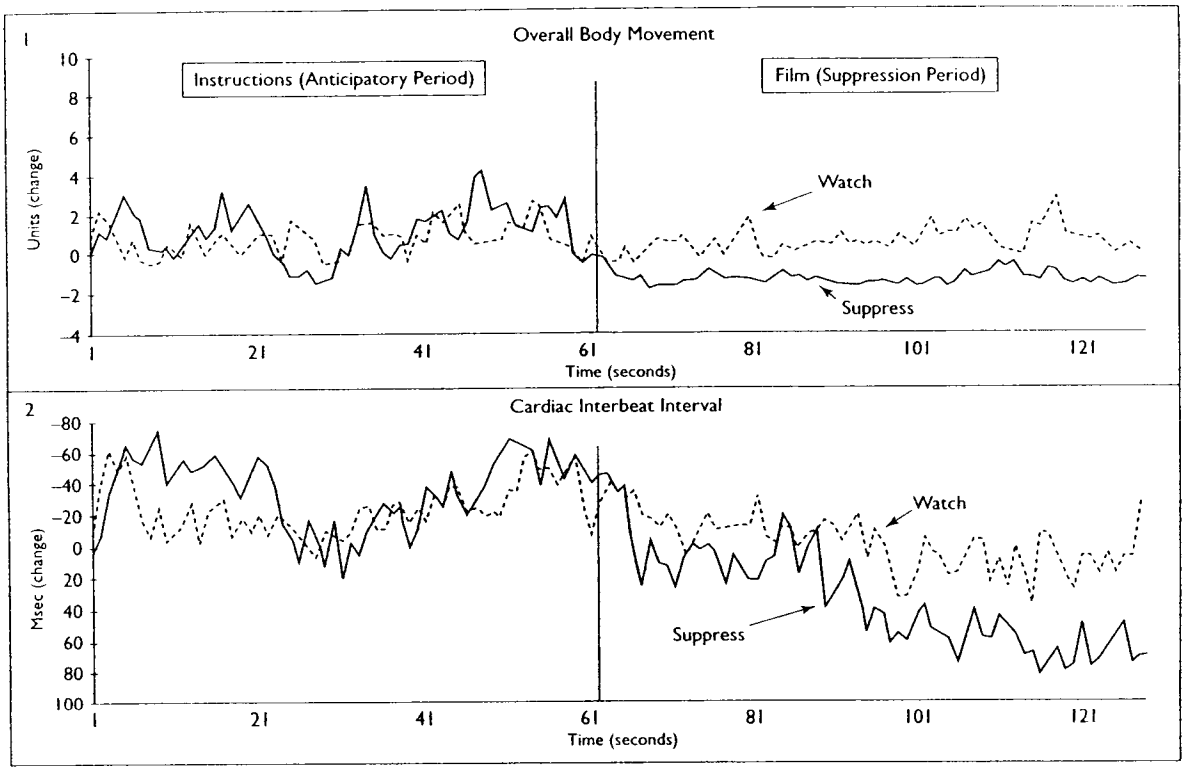

Figure 4.1 Panel 1: Somatic activity for suppression and no-suppression (Watch) participants. Panel 2: Cardiac interbeat interval for suppression and no-suppression (Watch) participants. Adapted from Gross and Levenson (1993).

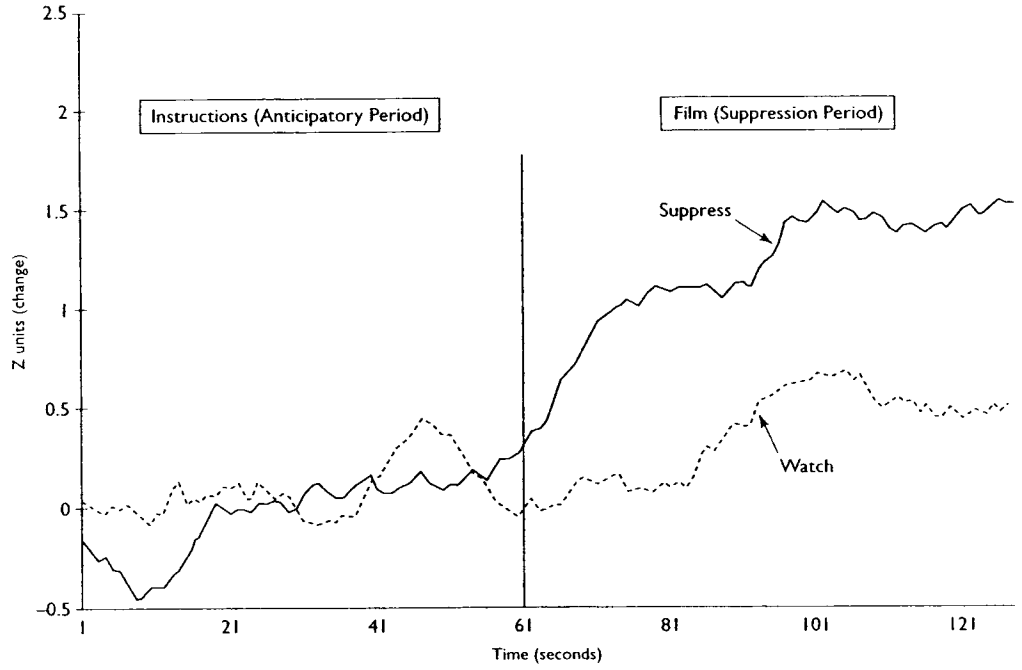

Figure 4.2 Sympathetic activation of the cardiovascular system for suppression and no-suppression (Watch) participants (adapted from Gross & Levenson, 1993).

subjects laugh and move around in their chair (Frijda, 1986). In each case, suppression decreases whatever response tendency is associated with the target emotion. Thus, suppression leads to lesser decreases in body movement during a sad film, and lesser increases in body movement during an amusing film. Heart rate generally follows somatic activity.

Despite decreased body movement and heart rate, as was the case for disgust, suppressing sadness and amusement leads to increased sympathetic activation of the cardiovascular system, including increased systolic and diastolic blood pressure, and decreased finger pulse amplitude, finger temperature, and pulse transit times. Suppressing sadness but not amusement also leads to greater electrodermal responding. Increased sympathetic activation of the cardiovascular system thus appears to be the common core to emotion suppression across emotion contexts, and suppressing either negative or positive emotions exacts a palpable physiological cost.

If these results really are the consequence of suppressing ongoing emotion-expressive behavior, suppressing non-emotional behavior during a neutral film should have no such consequences. To test this critical boundary condition, we examined subjects' responses during the neutral film. This film produced low levels of self-reported emotion and non-emotional expressive behavior such as lip-biting and yawning. As expected, suppression decreased non-emotional behavior, and no differences were found between suppress and watch subjects for any of the physiological variables. This is important, because it suggests that the physiological impact of emotion suppression grows out of the counterpoising of attempts to inhibit expression against strong impulses to express. In the absence of a stimulus that produces impulses to express, behavioral inhibition has relatively little impact on physiological responding.

Do all forms of emotion regulation have similar consequences?

Emotion regulation includes very different strategies such as thinking positive thoughts, using drugs, talking with friends, and suppressing emotional expression (e.g. Parkinson *et al.*, 1996; Thayer *et al.*, 1994). Do all these forms of emotion regulation have similar consequences? On the one hand, if the consequences of suppression are due to the effort it takes to override emotion-expressive behavior, other effortful forms of emotion regulation might have similar effects. On the other hand, different forms of emotion regulation could influence the emotion-generative process at different points, and thus have different consequences (Gross, 2001). To test this prediction, we compared emotion suppression with another form of emotion regulation, namely reappraisal, which involves re-evaluating a potentially emotionally evocative situation in order to decrease emotion.

In this study, subjects watched the same amputation film that had been shown in the initial studies. This time, subjects were randomly assigned to view this disgusting film under one of three instructional sets (Gross, 1998). In the first, subjects were asked to *think* about what they were seeing in such a way that they did not feel anything at all (reappraisal). In the second, subjects were asked to *hide* their emotional reactions (suppression). In the third, subjects simply watched the films (watch).

Results indicated that emotion suppression and reappraisal could indeed be distinguished. As observed previously, emotion suppression decreased disgust-expressive behavior, and increased sympathetic activation of the cardiovascular and electrodermal systems. Like suppression, reappraisal decreased expressive behavior. Unlike suppression, however, reappraisal had no observable consequences in terms of sympathetic activation of the cardiovascular or electrodermal systems. Whereas suppress subjects showed greater increases in sympathetic activation than watch or reappraise subjects, these latter two groups did not differ from one another. Also unlike suppression, reappraisal decreased disgust experience, whereas suppression had no effect on disgust experience. These findings show that the effects of suppression are not simply the result of *any* attempt at influencing one's emotions. How one goes about achieving an emotion regulatory goal may be as important a determinant of the affective consequences of one's efforts as the goal one is trying to achieve – not showing emotion during a social interaction may be a regulatory strategy exacting higher physiological costs than other regulatory strategies. Although much remains to be learned about the details of the physiological consequences of emotion suppression, the available evidence suggests that one core feature of emotion suppression – at least in the passive film and slide-viewing studies conducted to date – is sympathetic activation of the cardiovascular system.

Bridging the gap between acute and longer-term consequences

In the preceding sections, we have shown that (a) in the short term, emotion suppression leads to acute increases in sympathetic activation, and (b) over the longer term, individuals who suppress high levels of negative emotion seem to be at greater risk for cardiovascular disease. Together, these findings are suggestive of a causal link. However, it is far from clear how the acute effects of emotion suppression might translate into longer-term consequences that could promote cardiovascular diseases. In answering this question it is important to keep in mind the heterogeneity of cardiovascular diseases, e.g. primary and secondary hypertension, cerebrovascular disease, arrhythmias, or myocardial infarction, as well as the heterogeneity of conditions leading to cardiovascular diseases. Psychosocial factors are almost certainly involved to varying degrees in different cardiovascular diseases and might in some cases either be involved to a very limited extent or not be involved at all.

We consider two kinds of pathways. First, we consider *psychophysiological pathways*, by which emotion suppression could lead to transient increases in sympathetic activation which – if repeated many times – might precipitate a

cascade of processes that could directly influence cardiovascular health. Second, we consider *psychosocial pathways*, by which emotion suppression might significantly alter the material and emotional support that social partners provide, thereby indirectly influencing cardiovascular health. The pathways we describe are speculative, and are meant to illustrate rather than exhaust the mechanisms by which emotion suppression might contribute to cardiovascular disease. Although it seems likely that the two kinds of pathways interact (compare Jorgensen *et al.*, 1996; Myers & McClure, 1993), we describe psychophysiological and psychosocial mechanisms separately for the sake of clarity.

Psychophysiological pathways

Any one instance of heightened sympathetic responding, such as that seen in laboratory studies of emotion suppression, would seem unlikely to have deleterious consequences. However, an individual who shows an exaggerated sympathetic response of the cardiovascular system in a laboratory situation might be expected to show exaggerated sympathetic responses in many similar situations in daily life. According to the reactivity hypothesis (see Fredrikson, 1991; Krantz & Manuck, 1984), the cumulative effects of many such instances might be expected to take a toll. It is important to note that sympathetic activation is not always harmful (Dienstbier, 1989). However, if there is no time to recover (e.g. Linden *et al.*, 1997; McEwen, 1998) or if the enhanced cardiac responses are in excess of metabolic demands (e.g. Fisher, 1991; Obrist, 1983; Saab & Schneiderman, 1993), as might be the case in the context of emotion suppression, such increased sympathetic activation might lead to chronic functional and structural changes of the cardiovascular system that compromise its performance.

The available evidence seems to be generally consistent with this reactivity hypothesis. Cardiovascular hyperreactivity to different stressors appears to be involved in the development of essential hypertension (e.g. Everson *et al.*, 1996; Matthews *et al.*, 1993; Wood *et al.*, 1984; see, for review, Fredrikson, 1991) and atherosclerosis (e.g. Keys *et al.*, 1971; Manuck *et al.*, 1983), which are the two major risk factors for myocardial ischemia, infarction, and sudden cardiac death (e.g. Gillum, 1996; Guyton & Hall, 1997; Krantz & Manuck, 1984; Witteman *et al.*, 1998).

In the following sections, we describe how emotion suppression might lead to hypertension and atherosclerosis via exaggerated cardiovascular responding. Our focus is the sympathoadrenomedullary axis, although there is evidence that the adrenocortical axis (e.g. Fredrikson & Tuomisto, 1991; Henry & Stephens, 1977; Herd, 1986; Troxler *et al.*, 1977) and parasympathetic withdrawal (e.g. Brosschot & Thayer, 1998; Horsten *et al.*, 1999; Porges, 1995) also may play important roles in the development and progression of cardiovascular diseases.

The top part of Figure 4.3 shows several pathways by which the frequent incidents of sympathetic hyperreactivity associated with emotion suppression could lead to chronic hypertension. One such pathway was proposed by Folkow (1982),

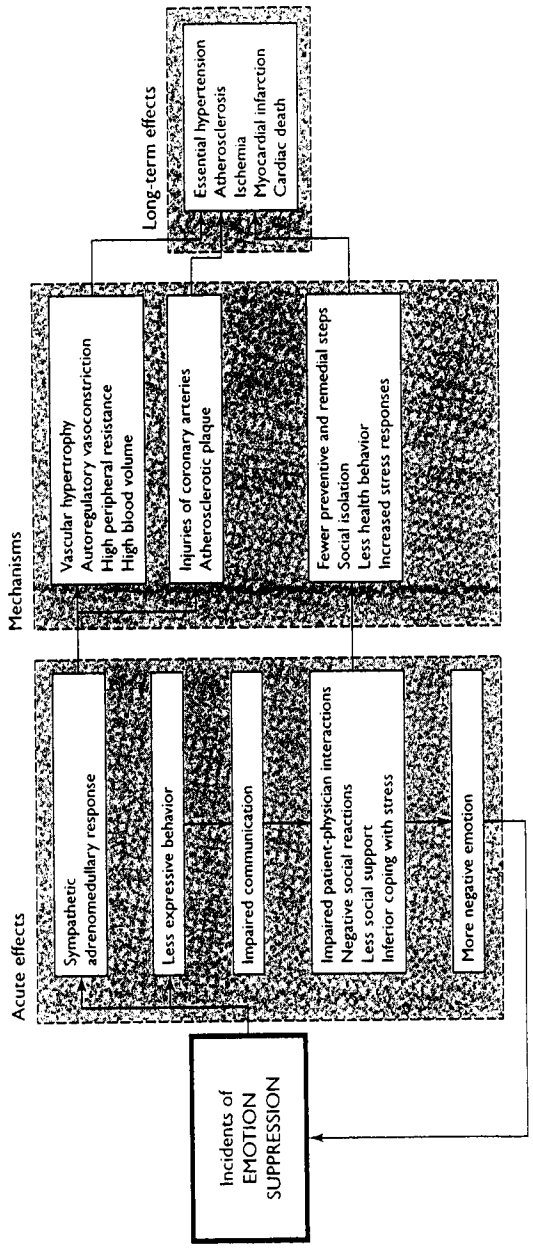

Figure 4.3 Psychophysiological and psychosocial pathways linking emotion suppression and cardiovascular diseases.

who suggested that the repeated pressor episodes exhibited by highly reactive individuals might promote smooth muscle hypertrophy. This process and a concurrent propensity for excessive vasoconstriction can lead to narrowed lumina (Folkow, 1982; Julius, 1993). Furthermore, cardiac output that is excessive relative to metabolic demand can trigger autoregulatory mechanisms, including peripheral vasoconstriction, to prevent tissue overperfusion (Obrist *et al.*, 1983; Sherwood *et al.*, 1986). Both narrowed lumina and excessive vasoconstriction can contribute to chronically heightened peripheral resistance. Moreover, high sympathetic drive might promote sodium and fluid retention through its effects on renal nerve activity. In the long run, this could lead to renal dysfunction with chronically higher blood volume (e.g. Guyton & Hall, 1997; Light *et al.*, 1983). Thus, chronically heightened sympathetic activation, with its attendant smooth muscle hypertrophy, increased peripheral resistance, and increased blood volume, might ultimately contribute to the development of chronic hypertension.

Figure 4.3 also shows pathways by which emotion suppression (and its associated increases in sympathetic activation of the cardiovascular system) could contribute to atherosclerosis. Some of these pathways involve the hemodynamic changes associated with heightened sympathetic activation. Repeated episodes of higher arterial pressure can lead, through hemodynamic forces such as sheer stress and turbulence, to micro-injuries of the coronary arteries at vulnerable points in the arterial tree (e.g. Clarkson *et al.*, 1986). Once the coronary endothelium is damaged, deposition of lipids, platelets, and fibrin (a clotting material in the blood) within the lesioned area can ensue (Guyton & Hall, 1997; Ross, 1993; Schneiderman, 1987). Atherosclerosis can then progress with chronic inflammatory cell proliferation, blood clot formation, and calcification and protrude into the lumen of the artery (Herd, 1986). The altered composition of the intima (the inner layer of the blood vessel) seems to provoke smooth muscles to move into the arterial intima and to proliferate, thereby further decreasing the size of the lumina (Herd, 1986; Ross, 1993; Schwartz *et al.*, 1981). Other possible pathways linking emotion suppression and cardiovascular disease involve the neuroendocrine components of heightened sympathetic medullary activation, in particular plasma catecholamines. Higher levels of circulating catecholamines can directly injure the intimal endothelium of the coronary arteries (Krantz & Manuck, 1984; Schneiderman, 1987), triggering atherosclerotic plaque growth. Catecholamines also induce a release of free fatty acids and lipoproteins into the blood stream, which can be atherogenic if they reach concentrations in excess of metabolic requirements (Carruthers, 1969; Henry & Stephens, 1977; Schneiderman, 1987). These factors contribute to the development of atherosclerotic plaque, which can ultimately completely occlude arteries. This development can become life-threatening, as atherosclerotic obstructive lesions and ensuing thrombosis can trigger myocardial ischemia, infarction, and cardiac death.

Psychosocial pathways

As shown in the bottom of Figure 4.3, emotion suppression also may be linked to cardiovascular disease outcomes via several psychosocial pathways. One particularly important psychosocial pathway is the patient–physician relationship. Roter and Ewart (1992) analyzed patient–physician interviews and found that patients with essential hypertension were less likely to express negative emotions than normotensive subjects. Such suppression of distress in clinical interviews might lead to a delayed detection of disease, less effective patient–physician communication, an underestimation of symptoms, and fewer preventive and remedial steps being taken by the physician to address social and emotional problems (Barsky, 1981; Roter & Ewart, 1992). This in turn could lead to decreased patient satisfaction, and an ensuing tendency of noncompliance with therapy (e.g. Haynes *et al.*, 1987; Lieberman, 1996).

In other relationships, too, emotion suppression could have costs that are relevant to cardiovascular health. Expression of emotions communicates to others a person's wishes and needs. If this communication is interrupted, because emotional expression is inhibited, others may be less accommodating. Individuals with a tendency to inhibit their negative emotions might thus, through reciprocal interactions, inadvertently create an environment provoking the experience of negative emotions (see Smith, 1992). Particularly individuals who exhibit a pattern of inhibition alternating with inappropriately strong expression of emotion could elicit negative social reactions (Davidson *et al.*, 1999). Frequent experience of negative emotions might in turn, as shown in Figure 4.3, prompt the individual to suppress these emotions, thus triggering a positive feedback loop.

In addition, emotional inexpressiveness and introversion have been related to less seeking of social support (Amirkhan *et al.*, 1995; Von Dras & Siegler, 1997). Two models have been proposed that link lessened social support to disease (Schwarzer & Leppin, 1991). On the one hand, social support serves a buffer function against other stressors such as when a person seeks emotional support from her friends after her spouse dies. If an individual has low social support, this buffer function is not afforded anymore and stressors create larger psychological and physiological stress responses (e.g. Jennison, 1992). On the other hand, low social support might have a direct, negative effect on the individual – social isolation might directly lead to heightened negative affect and poorer health-related behavior (e.g. Treiber *et al.*, 1991; Zimmerman & Conner, 1989). Additionally, the non-expression of emotions can have negative effects by preventing the beneficial effects of verbalization, which include restructuring of the emotion-eliciting event (Pennebaker, 1997). Without cognitive restructuring, there may be prolonged rumination, more frequent experience of negative emotions, and inadequate coping with subsequent events (see, for example, Greenglass, 1996; Pennebaker, 1997; Smyth, 1998), further increasing the frequency of harmful physiological responses.

Both consequences of low social support – the missing buffer function and the direct negative effects – could have a detrimental impact on various health

outcomes, including cardiovascular diseases (e.g. Adler & Matthews, 1994; Berkman, 1995; King, 1997; Smith & Pope, 1990; Uchino *et al.*, 1996). Such detrimental psychosocial effects might be especially costly *after* a cardiac event, when patients are in a vulnerable state (e.g. Berkman *et al.*, 1992; Orth-Gomér *et al.*, 1988; Ruberman *et al.*, 1984). Suppression thus might contribute both to the development of cardiovascular diseases and to their progression by impairing patient–physician interactions in particular and social relationships more generally.

Summary

The popular press has long urged that emotion suppression may be bad for our health. In this chapter, we have selectively reviewed the scientific literature on emotion and cardiovascular disease, and found that both emotion expression *and* emotion suppression seem to play a role in cardiovascular disease. To examine whether emotion suppression has any unique contribution to cardiovascular responding, we reviewed a series of laboratory studies on the acute consequences of emotion suppression. These studies showed that suppressing negative emotions such as disgust or sadness, or positive emotions such as amusement, leads to acute increases in sympathetic activation of the cardiovascular system. Although any one of these moments of increased activation is unlikely to have any long-term health impact, we have suggested psychophysiological and psychosocial pathways by which the acute effects of emotion suppression might translate into longer term threats to cardiovascular health. One important challenge for future research on emotion suppression and health will be to test these suggestions in the context of rich social interactions, with a broad range of vulnerable and non-vulnerable research participants. Such study will permit a better understanding of the complex health effects of differing patterns of emotion experience, expression, and suppression.

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