



# The role of emotional regulation in the development of hypertension

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## Abstract

The origins of psychosomatic medicine were concerned with the relationship between one's method of anger expression and resultant blood pressure change. This literature has been controversial: some theorists proposed that expression of anger will relieve psychodynamic tensions, and possibly help the person gain insight; others have posited, in contrast, that such expression will tend to promote social discord. Both pathways are thought to affect autonomic arousal, which may lead to sustained blood pressure elevation. The literature suggests that an "anger-in" style tends to characterize persons with hypertension; however, the mechanisms by which such non-expression of anger may lead to hypertension remain unclear. We have theorized that rumination about trauma-related events leads to sustained blood pressure elevations, and have supported this hypothesis in a series of empirical studies. © 2002 Elsevier Science B.V. All rights reserved.

*Keywords:* Emotions; Blood pressure; Hypertension

## 1. Overview

This article shall review the literature relating the expression of trauma-related thoughts and emotions to a physiological outcome, elevated blood pressure, and the development of hypertension. We shall describe a potential mediator of the relationship between non-

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expression of emotions and blood pressure elevations, *ruminatio*n, which may shed light on the mechanisms that underlie this association. 29  
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## 2. Emotional expression 31

The role of emotional expression has been a topic of controversy since the advent of psychosomatic medicine. Psychotherapists have historically operated on the premise that there is a therapeutic value to disclosing trauma-related thoughts and memories, e.g., Ref. [1]. However, while some early theorists proposed that the expression of emotions serves to alleviate underlying psychodynamic tensions [2,3], others have suggested that emotional expression tends to promote discord in one's self-concept and/or social relationships [4], and may cause damaging levels of physiological arousal (e.g., Refs. [5,6]). 32  
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Several pathways have been proposed to account for the therapeutic effects of expressing deep feelings. The divergent perspectives presented above pose some interesting questions. Does emotional catharsis relieve an underlying psychodynamic tension? Does it promote discord and conflict, and produce elevated autonomic arousal due to this conflict? There is also the possibility that discussing one's deepest thoughts—thoughts which may have been suppressed, or inhibited, may allow a person to engage in constructive problem-solving, to gain insight, and possibly decrease one's vulnerability to these trauma-related thoughts. 39  
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### 2.1. The non-expression of emotion and elevated blood pressure: an historical overview 47 48

Alexander formally heralded in the psychosomatic hypothesis in the 1930s, by proposing that blocking the *experience or expression* of specific emotions caused specific health difficulties [2,3]. Thus, he suggested that chronic inhibition of rage would lead to chronic blood pressure elevations. However, confusion concerning the empirical support for these conjectures has arisen due to the fuzziness surrounding the representation of “emotion inhibition” or “emotion blocking”. A close reading of Alexander's work and that of other writers of the time (e.g. Refs. [7–9]) suggests that they were referring to constructs postulated by Freud which entailed *unconsciously* inhibiting aversive emotional states. In this interpretation, the psychosomatic hypotheses propose that the inhibition of the *experience* of an emotion leads to elevated blood pressure, and, in the long term, to hypertension. This type of inhibition likely cannot be self-reported, as it presumably occurs without awareness. 49  
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On self-report scales, these persons would report relatively little anger, anxiety, or hostility, and would therefore be indistinguishable from those persons who genuinely experience infrequent or small amounts of these negative emotions (see Ref. [10] for a discussion of this topic). However, the psychodynamic model suggests that elevated blood pressure would be observed *only* in those who are unconsciously *inhibiting* painful or threatening emotions. 61  
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Within the original psychosomatic writings is also the suggestion that *knowingly* blocking or withholding a negative emotion will lead to disease states such as hypertension. In this latter case, the psychosomatic hypotheses assert that the inhibition of the 67  
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*expression* of an emotion leads to disease, and this type of inhibition is likely conscious and, therefore, self-reportable. 70  
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Although these two interpretations exist within the original psychosomatic formulations of the relation of the non-expression of emotion and high blood pressure, they are not clearly differentiated. Indeed, at certain points, it appears that Alexander [2,3] meant for terms such as emotion *experience* and emotion *expression* to be used interchangeably. This has bequeathed a legacy of confusion and ambiguity. Given the multiple meanings previously associated with the term “emotion inhibition”, the term *non-expression of emotion* will be used in this article to simply indicate that emotion is not being expressed, whether consciously or unconsciously. 72  
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### 2.2. Why focus on blood pressure and hypertension? 81

Although non-expression of emotion may affect many physiological systems and disease outcomes, there are important reasons to focus attention on blood pressure and hypertension. Hypertension is a principle risk factor for cardiovascular disease, contributing to about 10% of deaths in the United States [11]. As blood pressure increases from normal to high elevations, so too does the risk of atherosclerosis, left ventricular hypertrophy, and stroke [12]. In the U.S., the estimated age-adjusted prevalence of hypertension is 22.9% for Caucasians and 36.7% for African-American Americans [11]. The prevalence of this disease is very high, and the economic and quality of life costs are enormous. 82  
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### 2.3. The role of anger expression 92

Much of the research relating to the relationship between non-expression of emotion and blood pressure has focused on modalities of anger expression. Does keeping one’s anger in, or letting that anger emerge, tend to lower blood pressure? It is important to remember that this was the focus of the original impetus for the entire field of psychosomatic medicine—there was something highly compelling about the notion that holding one’s anger in would lead to blood pressure elevations. 93  
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Originally, anger expression was conceptualized unidimensionally: persons could be classified as either *expressing* their anger (“Anger-out”) or as *not* expressing their anger (“Anger-in”). Researchers have found that persons categorized as Anger-in and those categorized as Anger-out had different cardiovascular responses to anger-provoking situations [13]. This suggests that anger expression style has important implications for the effects of negative emotionality on physiological processes. 99  
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Spielberger et al. [14] constructed a self-report measure of anger expression based on this classification system, and found that Anger-in and Anger-out items comprised two separate and independent factors, rather than a single dimension. Spielberger’s conceptualization of anger expression into Anger-in (that is, how often angry feelings are not disclosed) and Anger-out (that is, how often angry feelings are expressed in verbally or physically aggressive behavior) has remained the predominant view of anger expression. 105  
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Despite other ambiguities in his writings, Alexander clearly hypothesized that *expressing* one’s anger would lead to lower resting blood pressure levels, and that *not* expressing 111  
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one's anger would cause one's resting blood pressure to become elevated [3]. However, in the contemporary literature, two seemingly contradictory hypotheses have been forwarded.

In the original psychosomatic hypothesis, Anger-in is conceptualized as health-damaging because it causes prolonged states of autonomic arousal, including elevated heart rate and cardiac output (the amount of blood pumped out at each heartbeat) which, over time, are hypothesized to lead to increased resting blood pressure and to coronary heart disease (CHD).

In contrast, the *Hostility/aggression* hypothesis suggests that the *outward* expression of anger is viewed as detrimental to health through heightened autonomic arousal, which results from the anger display and, which, over time, leads to an upward drift in resting blood pressure.

Many studies have examined the association of anger expression style and resting blood pressure. Suls et al. [15] published a meta-analysis of this literature and found that, for the most part, *anger-in* has been associated with *higher* resting blood pressure, and anger-out with *lower* blood pressure levels. These results tend to provide support for the psychosomatic hypothesis concerned; however, they do not address the *mechanisms* by which an anger-in style of anger expression may impact the development of hypertension.

Linden and Feuerstein [16] has proposed a Social Conflict Model, which posits that adaptive styles of anger expression lie on a continuum between aggression and passivity. The model suggests that the relation between anger expression and blood pressure is best described as a U-shaped curve: persons with extreme anger-out tendencies, as well as those exhibiting excessive anger-in behavior, are thought to generate social or intrapsychic conflict as a consequence; these persons may be at the greatest risk for developing high blood pressure. Several studies have provided support for the Social Conflict model [17,18].

Everson et al. [18] have described an anger expression style—Constructive Anger Behavior-Verbal (CAB-V)—and have shown that persons who tend to express their anger, but in a constructive and reflective manner, have lower resting blood pressure levels than those who do not. This is important because it suggests that anger-out does not necessarily involve aggressive behaviors; one may express anger in a constructive manner that may lead to a resolution of the problem and, therefore, to a lower level of autonomic arousal and blood pressure.

#### 2.4. The physiological costs of suppression emotions

Researchers have hypothesized that trauma-related thoughts may be actively avoided or suppressed [19], a process which has cognitive and emotional costs: such thoughts often do not have a chance to become integrated into an existing schema, and therefore, the problems and anger that originated with the trauma itself may remain unresolved. There also may be physiological costs. Thought suppression requires an expenditure of physiological resources: thus, it may produce a low but chronic level of activation of several physiological systems, including the cardiovascular system, hypothalamic–pituitary–adrenal system, and the immune system [1,20–23].

Several studies have considered the effects of thought inhibitions, that is, the act of deliberately *not* thinking about an object or event [19]; these researchers have suggested

that thought inhibition may produce a paradoxical effect, such that inhibited thoughts are likely to later re-emerge as thought intrusions, or ruminations. Wegner used what has now become the well-known “white bear” scenario, in which subjects are specifically instructed to *not* think about a white bear, during a cognitive task. The results of these studies showed that subjects who were instructed in this way actually reported “white bear” thoughts more, during a succeeding task period, than subjects who were told to *think* about a white bear. Although these results are provocative, they have been observed only under highly controlled laboratory conditions, and their generalizability is unclear. However, recent research by Gross [24] indicates that thought inhibition, as well as later rebounded thought rumination, may increase both autonomic activation and blood pressure.

### 2.5. The psychological life of a stressor: the role of rumination

We and others have hypothesized elsewhere that the relation between memories of a traumatic event and subsequent sustained blood pressure elevation is mediated by rumination concerning the event [25,26]. “Rumination”, in this context, refers to persistent thoughts about the traumatic event, accompanied by negative affect, which do not lead to a resolution.

Most of the research concerning physiological responses to stress as a mediator of hypertension and CHD has focused on the acute effects of the stressor on the immediate blood pressure response (i.e., “cardiovascular reactivity”) [27]. However, although the physical presence of the stressor may be brief, it may have effects on cognitive, affective, and physiological processes that endure long after the stressor itself has ended. Although such effects may persist for years in some cases, our research has focused on the short-term recovery of blood pressure following a stressor, to pre-stress levels. In a first study in this series, we found that recovery following a stressor that produced a strong negative emotion (serial subtraction with harassment and shock avoidance) led to significantly poorer blood pressure recovery than a non-emotional stressor (cold pressor, moderate physical exercise) [28].

One defining characteristic of rumination concerns the extent to which one’s attention is focused on the thoughts and emotions associated with a previously occurring event. We reasoned that one way to go about understanding the processes involved in rumination was to manipulate the attentional focus: thus, we hypothesized that the presence of *distracting* stimuli would *reduce* rumination. In a preliminary study, we found this hypothesis to be supported: following a stressful arithmetic task, subjects who were *not* provided a distraction exhibited significantly poorer recovery than subjects who were exposed to a distraction (a mild mental task). This study suggests the importance of the post-stress thoughts in the sustained blood pressure elevation following the termination of the stressor. However, it does not address this directly because post-stress cognitions were not assessed. In the next study in the series, we developed a protocol for assessing post-stress thoughts. We found that the availability of distraction (posters, magazines, toys), as predicted, reduced the likelihood of stress-related thoughts as well as hastened the blood pressure recovery. In addition, we found that trait measures of ruminative tendency was a strong predictor of post-stress blood pressure recovery. Finally, we have found that trait measures

of ruminative tendency predicted both clinic blood pressure and ambulatory blood pressure [29]. 201  
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Taken together, the results of these studies suggest that rumination plays a role in 203  
maintaining blood pressure elevations after the stressor is no longer present. The fact that a 204  
trait tendency to ruminate is associated with blood pressures taken in subjects' natural 205  
environment suggests that such thoughts may be implicated in sustained blood pressure 206  
elevations. 207

### 3. The psychological life of a stressor: the role of rumination 208

It is interesting that the very origins of psychosomatic medicine began with the 209  
hypothesis that expressing one's deepest thoughts and emotions would lower the blood 210  
pressure. It is part of the interesting story of behavioral science that we have come full 211  
circle, for the original notions, rooted in Freud's core psychoanalytic theories, fell into 212  
disfavor with the advent of disciplines such as introspectionism, Gestalt theory, and 213  
classical and operant conditioning theories. The pathways by which we speculate that 214  
non-expression of emotions may affect psychological and physiological processes have 215  
changed; thus, in this article, we have presented evidence that the continued psycho- 216  
logical representation of a traumatic stressor, i.e., *rumination*, may represent one such 217  
pathway. However, the basic intuitions that exercised so much influence over the origins 218  
of the field of psychosomatic medicine remain viable, and it is gratifying that we have 219  
not discarded them while developing the flexibility to develop new ways of testing these 220  
notions. 221

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