

Review

An historical context for behavioral models of hypertension

William Gerin^{a,b,*}, Thomas G. Pickering^b, Laura Glynn^c, Nicholas Christenfeld^d,
Amy Schwartz^b, Douglas Carroll^e, Karina Davidson^b

^aOffice of Behavioral and Social Sciences Research, NIH, Bethesda, MD, USA

^bHypertension Center/Starr Pavilion ST405, Cornell University Medical Center/The New York Hospital,

525 East 68th Street, New York, NY 10021, USA

^cUniversity of California Irvine, Irvine, CA, USA

^dUniversity of California San Diego, La Jolla, CA, USA

^eUniversity of Birmingham, Birmingham, UK

^fUniversity of Alabama, Tuscaloosa, AL, USA

Abstract

Objective: The purpose of this study is provide an historical context for current behavioral models of hypertension. **Methods:** A selective sample of the cardiovascular reactivity literature was reviewed, from 1932 to present. **Results:** In the earliest model, cardiovascular reactivity was regarded as a marker of disease risk; however, in later models, reactivity came to be viewed as a causal influence in the development of hypertension. As the models evolved, the underlying assumptions changed. Thus, the risk marker model assumed that cardiovascular responses to stress were a stable, generalized characteristic of the individual, and therefore the eliciting stimuli were

arbitrary. The later models, however, assume that the nature of the eliciting stimulus is a determinant of the cardiovascular response. We describe the increasing complexity of the four models, and contrast their underlying assumptions and the implications of these assumptions. **Conclusion:** We provide an overview of study designs and variables that should be incorporated into studies seeking to understand the ways in which cardiovascular responses to stress may influence the development of hypertension. © 2000 Elsevier Science Inc. All rights reserved.

Keywords: Cardiovascular reactivity; Blood pressure; Heart rate; Hypertension; Individual differences

Overview

Stress and other behavioral factors such as social support have been linked to a broad range of cardiovascular disease outcomes, including coronary heart disease [1,2] and hypertension [1,3]. The mechanisms, or pathways, through which these influences operate, however, remain controversial. We will discuss the evolution and characteristics of four different psychophysiological reactivity models that focus on the relationship between stress and the development of hypertension. In the earliest model, it was hypothesized that stressful events produce an increase in cardiovascular parameters such as blood pressure and heart rate, and that persons who display very large increases are at increased risk for the development of hypertension. Thus, reactivity is a property of persons that marks vulnerability to hypertension. A later model hypothesized that these exagger-

ated cardiovascular responses play a causal role in the development of hypertension [4]. One assumption common to both viewpoints is that reactivity is a property of the *person* (i.e., is unrelated to the circumstances in which the reactivity occurs).

More recent research, however, has focused on the *interplay* of person properties and situation properties to understand the causal role that reactivity may play in hypertension development [1]. Concern lies with what properties—either of persons or of situations—are pathogenic, and how they influence each other. For example, a situation may have a large effect on blood pressure reactivity only if the individual possesses certain trait characteristics. Thus, an irritating interpersonal stressor may have more pronounced effects on a highly hostile person than on one who scores relatively low on this dimension. In this model, reactivity continues to be linked causally to hypertension onset, but the link depends on the complex interactions between trait and situation characteristics.

* Corresponding author.

E-mail address: bgerin@ibm.net (W. Gerin)

Table 1
Characteristics of four reactivity models

Model	Characteristics
1	<ul style="list-style-type: none"> • Risk marker model (no consideration of possible causal role) • Focus is on physiological differences between individuals • Cardiovascular response assumed to generalize across stimuli; stimulus considered arbitrary
2	<ul style="list-style-type: none"> • Causal model • Focus is on physiological and psychological differences between individuals • Generalizability of cardiovascular responses across stimuli found to be poor; nature of stimuli no longer considered arbitrary
3	<ul style="list-style-type: none"> • Causal model • Focus is on situational differences (no consideration of individual differences in either physiology or psychology) • Nature of stimulus is major determinant of cardiovascular response
4	<ul style="list-style-type: none"> • Causal model • Focus is on physiological and psychological differences between individuals, as they may interact with the situation • Generalizability of cardiovascular responses across stimuli not assumed; nature of stimulus assumed to interact with personality characteristics to produce cardiovascular responses

This article traces the history of four reactivity models, and will show how their origins and the early research influenced thinking about the role that reactivity might play in the development of hypertension. We examine the models in roughly chronological order. Table 1 shows the characteristics of the four models.

We selectively review reactivity studies from an historical perspective to see how the four models have been applied. Table 2 gives a timeline of some of the major events and changes that occurred during the several decades of research in this area.

Basics of cardiovascular reactivity testing

Over the past half-century, hundreds of cardiovascular reactivity studies have been published. The purposes of these studies, as well as the specific techniques used, are quite varied. However, the basic procedures, laid out in the first studies conducted by Hines and Brown [5,6], have remained remarkably constant across laboratories and over time. Most studies involve the following steps:

1. Participants are instrumented with a device that measures cardiovascular outcomes, and then sit through a rest, or baseline, period (usually be-

tween 5 and 30 minutes). The purpose of this phase is to provide a within-person comparison for the effect of the stressor. Cardiovascular measurements are taken throughout, and the mean of these represents the *resting* measure (initial measurements may be discarded to allow for adaptation).

2. A stressor is then presented. The duration of presentation is quite brief, usually a few minutes. More will be said about how the stressors are chosen in a later section. Cardiovascular measurements are taken during this period; the average of these usually represents the *task* measure of the cardiovascular parameter(s). Peak value is sometimes used, rather than the mean of the entire period.
3. Occasionally, a poststress rest period is included. This period provides a way of evaluating recovery of the parameter from the stress-induced levels. Cardiovascular measurements are taken during this period, and the average of these measurements, or of a subset over a specified poststressor interval, usually represents the recovery measure.
4. A change score is usually computed in which the baseline measure is subtracted from the task level.

Table 2
Selective timeline of important events in the evolution of cardiovascular reactivity models

Period	Event
1932	Hines and Brown report their first study of cardiovascular reactivity; model 1 becomes focus of laboratory studies
1930s to 1950s	Many cardiovascular reactivity studies published, most focusing on differences in blood pressure response between normotensives and hypertensives; most of these studies use the cold pressor as the eliciting stimulus
1951	Wolff and Wolf use ballistocardiography to estimate cardiac output in response to a stressful interview
1959	Brod and colleagues use a mental arithmetic stressor to differentiate hemodynamic patterning between hypertensive patients and normotensive controls
1969	Guyton publishes his work on autoregulatory models
1970s	Test–retest reliability (stability over time) of cardiovascular reactivity addressed; impedance cardiography techniques come into use; model 2 becomes the focus of laboratory studies
1976	Obrist first publishes “active/passive coping” formulation; model 3 becomes focus of laboratory studies
1980s	Generalizability among laboratory stressors investigated; ambulatory blood pressure monitors come into use; generalizability of blood pressure responses to laboratory stressors to the natural environment investigated
1990s	Stressful social interactions come into use in the laboratory in an attempt to broaden ecological validity of the cardiovascular reactivity model; model 4 becomes focus of laboratory studies

The change score usually constitutes the measure of cardiovascular reactivity. More sophisticated representations of reactivity are also used, in which the baseline is controlled statistically, to account for individual differences in resting levels [7].

Reactivity as a marker for hypertension risk (model 1)

In 1932, Hines and Brown [6] published the first account of cardiovascular reactivity testing. The motivation for their study was to develop a simple procedure, applicable in the doctor's office, for identifying future cases of hypertension. This rationale was clearly laid out in a study by Ayman and Goldshine, published in 1938 [8]. They noted:

Many young people are certain to develop essential hypertension some day. If it were possible to apply a standard stimulus to such people, it might be possible to determine a group in which an abnormally great reaction resulted from this stimulus. In other words, it might be possible to determine in advance the future subjects of essential hypertension.

This statement clearly highlights the *marker* status that cardiovascular reactivity had been assigned by these early researchers. A marker is a variable that *predicts* a disease outcome, but does not necessarily *cause* the outcome. Such a variable may, instead, be associated with both the marker and the outcome. Thus, race is a risk marker for hypertension, with African-Americans considerably more likely to become hypertensive than whites [9]. However, race itself is not a causal factor in this relation; specific mechanisms must be sought. For example, it has been suggested that African Americans may be exposed to a disproportionate amount of chronic stressors due to racism [10].

To test their hypothesis, Hines and Brown performed what was to be the first of many cross-sectional comparisons of stress-elicited cardiovascular responses in normotensives compared with hypertensive patients [5,6]. They used the cold pressor as the stressor (cold stimulus applied to the patient's hand). They did find that hypertensive patients exhibited larger blood pressure reactions to the cold pressor than normotensive controls, thus providing evidence for the marker status of cardiovascular reactivity. They also found that hypertensives, on average, took longer than normotensives to recover to pretask levels. The results of Hines and Brown's studies [5,6] have been replicated by many other researchers [11], although not all studies have found this pattern [4].

Nature of the eliciting stimulus

There are two important implications of the risk marker model. The first is that the elicited physiological

response was judged to be a broad, general characteristic of the person; that is, if a particular person showed a very large blood pressure increase in response to the cold pressor, it was assumed that s/he would show comparably large responses to a broad range of other stimuli as well.

The second implication is derived from the first: the nature of the eliciting stimulus is therefore arbitrary. This model merely calls for a stimulus that elevates blood pressure, allowing people to be ranked with respect to their risk for hypertension.

As described earlier, Hines and Brown [5,6] used the cold pressor as the stressor in their studies, having the person place his or her hand in a bucket of ice cold water for 20–30 seconds. As an illustration of the arbitrariness with which this task was selected, the *alternative* they had considered was inhalation of carbon dioxide, which will also raise blood pressure. They chose the cold pressor mostly because it was easy to administer [6].

The cold pressor was the only stressor used for many years in reactivity studies. The first use of a "mental" (compared to a physical) stressor occurred, as far as we can determine, almost two decades after Hines and Brown's initial study [5]. Wolff and Wolf [12] estimated cardiac output by ballistocardiography (an invasive technique that provides estimates of hemodynamic patterning), in response to a stressful interview. In a similar investigation conducted a few years later (in 1957), Hejl used mental arithmetic as the stressor [13]. This study was followed up by the classic 1959 study by Brod and colleagues [14], who also used mental arithmetic as a stressor to study the hemodynamic patterning underlying the pressor responses of normotensives and hypertensives. Mental arithmetic became the most commonly used mental stressor in reactivity testing during the next 30 years [4], and probably remains so today.

The original conception of the role of cardiovascular reactivity was only as a marker of future hypertension, rather than as a causal factor in the etiology of hypertension. It is significant that reactivity, at this stage, was regarded as a *trait*; that is, a characteristic of the person that was presumably stable over time and across situations. In fact, situational influences on blood pressure variability were not considered in this model at all.

Reactivity as a trait-based contributing cause to the development of hypertension (model 2)

In the past few decades, the causal model of reactivity has been adopted by most researchers in this area and, consequently, reactivity has been embraced as the most likely mechanism through which stress adversely affects cardiovascular health. This revised formulation—that cardiovascular reactivity plays a causal role in the development of hypertension—has an important implication concerning the notion of *exposure*; that is, for reactivity to play a causal role in the mediation of the stress–

disease relation, we must assume that the exaggerated blood pressure and heart rate responses occur frequently. Manuck et al. [7], for example, noted:

... if a heightened cardiovascular responsivity to stress were to contribute directly to the development of hypertension, it must be assumed that the reactivity of the hyperresponsive person is “expressed” frequently and over protracted intervals. To the extent that the intensity of real-life stimuli (stressors) needed to evoke expressions of reactivity is common in daily life, this assumption may be valid for persons living in all but the most benign environments.

It is important to reconceptualize psychophysiological reactivity as an exposure concept rather than as an individual difference concept. This shifts the research goal from solely one of identifying persons at risk for disease to identifying persons as well as the situational and psychological exposures that *put* persons at risk.

We previously noted that reactivity was originally viewed purely as a person-based trait (in addition to its perceived status as a marker for risk). Model 2, like model 1, views reactivity as a trait dimension. However, where model 1 viewed reactivity only as a marker for disease, model 2 posits that reactivity plays a causal role in the disease process.

Various biological mechanisms that may mediate the relationship between reactive episodes and hypertension (e.g., arteriolar structural changes) have been proposed; a review of these, however, is beyond the scope of this paper. See Pickering [3] and Saab and Schneiderman [15] for brief reviews.

Trait construct of reactivity: measurement issues

It was not until the 1970s that researchers began asking the questions that must be addressed by any hypothetical trait construct, including reactivity. Is the response replicable within the person over time? Is it stable across different stimuli? And, does the stressor response generalize across situations, and to the real world?

Stability of cardiovascular responses over time

The first test of any putative trait dimension is the degree to which it remains stable within the person, over time. Clearly, to be regarded as a stable trait, or person dimension, the parameter must remain fairly constant from one occasion to the next. In a recent review, Manuck and colleagues reported that, in the studies they examined, correlations ranged from quite weak to quite strong for heart rate reactivity; the mean correlation was moderate at best ($r = 0.51$) [7]. They reported that the test–retest correlations for systolic blood pressure reactivity are weaker than those for heart rate. Diastolic pressure reactivity, they reported, is the

weakest of the three, with an average correlation of 0.34. Overall, they concluded, the evidence that reactivity is a stable characteristic over time is “mixed, and at best, moderate [for heart rate]; in the case of diastolic blood pressure, retest reliability is frankly equivocal” (p. 116). They further commented “When taken as a whole, it must be concluded that this literature provides rather less support for retest reliability than would ordinarily be expected of a robust dispositional construct” (p. 118). Employing basic measurement theory principles, Kamarck et al. [16] found that aggregating reactivity measures across three different stressors produced stronger test–retest associations (most greater than 0.70) than had previously been observed. The value of this sort of aggregation is that it will reduce the effects of nonsystematic influences (e.g., fatigue, order of presentation of the stressors, caffeine, mood) on the outcome measures. However, such a strategy will only be useful when the effects of the different stressors are themselves correlated. As such, the response to each individual stressor may be regarded as an aspect of a broader trait dimension; that is, “blood pressure reactivity to mental stress,” rather than a narrower dimension (e.g., “blood pressure reactivity to serial subtraction”).

In summary, the stability of reactivity over time was moderate at best [7]. Test–retest reliability serves as the theoretical upper limit in terms of the observed relationships with any other measure, because no other dimension will correlate more strongly with a given measure than the *same* measure assessed on a different occasion. Thus, it is crucial to demonstrate that the reactivity dimension is replicable over occasions. Kamarck and colleagues’ suggestion of aggregating across stressors [16] should increase the stability of the measure, but most researchers still tend to rely on responses to a single stressor, and therefore these measurements are only moderately reliable.

Stability of cardiovascular responses across stressors

Beginning in the mid-1980s, many researchers began to consider the question of the associations among different cardiovascular measures or across tasks. Few studies have systematically compared the association between reactivities to different tasks within participants. Parati et al. [17] found strong correlations between the responses to some tasks but not others. McKinney et al. [18] found a strong correlation between a video game and a reaction time task, for mean blood pressure. Weaker correlations were found between each of these tasks and the cold pressor. Fredrikson et al. [19] examined the correlation between change scores for four tasks: an attentional demands task; mental arithmetic; cold pressor; and isometric muscle contraction. For systolic blood pressure, only the correlation between mental arithmetic and isometric muscle contraction was significant. This was only true for normotensive

participants; for hypertensive participants, the correlation between these tasks was small and nonsignificant. Also, as with the previous studies, the smallest correlations found were between the cold pressor and the other tasks; thus, little evidence of generalizability between the tasks was found overall. Finally, Kamarck et al. [16] aggregated cardiovascular responses across laboratory stressors, on two occasions, and examined the intertask associations within each of the sessions. Three tasks were used in this study: a memory recall task; a target-shooting task; and a tracking task. The correlations among the three tasks ranged from small to fairly large for systolic blood pressure, and were uniformly moderate for diastolic pressure and heart rate change.

In summary, the evidence that reactivity is a characteristic of the person that can be generalized across a wide variety of tasks is mixed, and requires additional research.

Generalizing from the laboratory to the natural environment

We have noted that it is difficult to demonstrate associations among blood pressure and heart rate responses to a range of stimuli within the laboratory, where other influences may be controlled. However, establishing relations between reactions in the laboratory and in the real world presents an even greater challenge. For example, in the laboratory, it is usual to ensure that participants are seated during stress testing. In contrast, in the real world, stressors may be encountered while standing. This is important because posture has a substantial influence on blood pressure [3] and the underlying hemodynamics of blood pressure reactions to stress [20]. In the natural environment, each blood pressure measurement may be influenced by a number of uncontrolled stimuli, including posture (other influences include activity, mood, caffeine intake, etc. [3]). If these influences remain unaccounted for, the measurement will tend to correlate poorly with other measures (in the same way that poor test–retest reliability will attenuate correlations between measures [21]).

However, generalization from laboratory to everyday life is a crucial issue if we are to understand the role that reactivity may play in the development of hypertension. The value of observing cardiovascular reactions to a laboratory stressor lies in the ability of that stressor to elicit reactions typical of those provoked by the large number of the stressful situations the person will encounter in the real world. If reactivity is to be implicated as a cause of hypertension, it must occur with some frequency in the person's natural environment. If, however, the laboratory-based reactivity does not predict reactivity in the real world (i.e., generalizability is poor), then there is little reason to assume that the people identified as high reactors in the laboratory will be the ones who suffer more damaging reactive episodes in the

real world. For example, an individual may produce very high blood pressure responses to a cold pressor stimulus in the laboratory. If the cold pressor is not representative of the types of stressors that occur in the real world, then the blood pressure response it produces may only be reproducible in those instances in which the person finds herself immersed in ice water, an occurrence that is relatively infrequent in the lives of most people.

Beginning in the 1980s, researchers began to examine associations between blood pressure and heart rate reactivity in the laboratory and reactivity in the field [4]. In part, such investigations did not occur earlier because ambulatory monitoring techniques were not widely available. The development of reliable and robust ambulatory blood pressure monitors allowed investigators to sample participants' blood pressure while the participants went about their usual activities.

Several studies have shown that resting blood pressure levels in the laboratory predict ambulatory blood pressure levels obtained in the field (see Ironson et al. [22] for a review). However, a key assumption of the reactivity hypothesis is that additional information, over and above that provided by resting levels, may be obtained by an examination of the changes in blood pressure and heart rate that occur in response to stressful and challenging laboratory tasks. Some studies report that reactivity scores do not provide information different from that gained from resting blood pressure levels [23,24]. However, Saab and Schneiderman [15] report that systolic blood pressure reactivity change scores obtained during a structured interview added 6–7% of variance explained in ambulatory blood pressure levels over that predicted by baseline levels. Similarly, they found, in African-American subjects, that diastolic blood pressure reactivity change scores from the cold pressor task predicted an additional 5% of the variance in ambulatory blood pressure. They point out that properties of persons—in this case gender and ethnicity—need to be considered in understanding which task reactivities predict to natural environment blood pressure levels.

We conclude, as we have elsewhere [4], that the evidence supporting laboratory-to-life generalizability remains weak, and that this weakness is problematic for trait-based models of the reactivity–hypertension link.

Reactivity as a situation-based contributing cause in the development of hypertension (model 3)

Like model 2, model 3 hypothesizes that reactivity plays a causal role in the development of hypertension. However, rather than focusing on trait dimensions, which places the emphasis on characteristic responses of the individual, model 3 focuses on dimensions of the situation that tend to elicit blood pressure and heart

rate reactivity. Thus, model 3 posits that individuals who are exposed to stressful situations, and therefore exhibit exaggerated reactivity with some degree of frequency, are at greater risk for hypertension. For example, we have found that college students' blood pressure rose substantially when placed in a situation in which their performance was being evaluated by another person [25,26]. When the situation was arranged so that the other person present did not pay attention to the results of their performance, the subjects' blood pressures were not elevated much beyond the resting level.

Active and passive coping

In 1981, Obrist published a volume entitled *Cardiovascular Psychophysiology* [27]. Drawing on the earlier ideas of Guyton and associates [28], Obrist [27,29] outlined a possible mechanism whereby enhanced cardiac reactions could contribute to the development of high blood pressure and, eventually, to hypertension. He suggested that some sorts of psychological stressors demand attention and vigilance, what he called "active coping," but require little in the way of physical activity. These are events over which the person is able to exercise some degree of control, in accordance with his or her abilities and efforts. In contrast, a *passive* coping response was elicited by situations over which the person had little or no control, and thus, working to overcome the stressor was useless. Obrist hypothesized that active coping situations tend to provoke reactions of the heart that are inappropriately large for the corresponding increases of energy expenditure [29]. Carroll [1] pointed out that this pattern of reaction is in marked contrast to reactions to physical exercise, where the increase in cardiac output closely mirrors the increase in the physical demands of the exercise and the energy expenditure of the person. Carroll suggests that psychological or mental stress may elicit what has been termed "additional" cardiac output; that is, increases in cardiac output over and above what would be expected on the basis of the physical energy demands of the particular psychological stress [1]. According to Obrist's formulation, this results in overperfusion, particularly of skeletal muscle tissue. Blood is pumped to these muscles to an extent that is surplus to the muscles' requirements for energy. This, in turn, precipitates autoregulation in the form of adjustments to the circulation in order to compensate for the overperfusion. These autoregulatory adjustments take the form of increases in arterial resistance [1].

The sequence of events described by Obrist suggests that a sustained blood pressure increase can occur in the face of even the most transient psychological stressor. Blood pressure rises initially as a result of increased cardiac activity, and the elevation in blood pressure is then maintained, even when the cardiac output increase subsides, by the autoregulatory increase in resistance.

It is worth noting that this formulation anticipates a somewhat later development, by focusing on both individual predispositions *as well as* situational exposure. Thus, this theory further presumes that repeated exposures to acute psychological stress in persons who are highly responsive to it, and the relatively sustained blood pressure elevations this provokes, over time lead to a resetting of that person's blood pressure at a higher level. Such *person-situation interactions* will be discussed more fully, in the context of model 4.

Obrist's formulation has been tested by several theorists. First, whether a stressor provokes an active coping response or a passive response has been linked to differential effects on the cardiovascular system. Specifically, researchers have demonstrated that active coping stressors, which appear to be mediated by way of β -adrenergic pathways, tend to raise blood pressure via central mechanisms, that is, by increasing cardiac output [30]. Passive stressors, those over which the person has little or no control, appear to be mediated via α -adrenergic pathways, and appear to elevate blood pressure via arterial mechanisms, that is, by increasing total peripheral resistance [30]. These studies provide insight into the nature of "stress"; a particular event may be experienced as more or less stressful depending on the degree to which it is perceived as controllable; and this factor appears to manifest itself systematically in the differential adrenergic pathways through which blood pressure is maintained.

This line of research provided an important complement to the blood pressure reactivity studies by demonstrating that the task typology suggested by Obrist—active coping vs. passive—corresponded to different physiological pathways, that is, cardiac output (active coping) and peripheral resistance (passive) [27,29]. This provided strong evidence that psychological dimensions (high or low perceived control), corresponding to aspects of the environment (active coping/high control; passive/low control) might systematically influence the blood pressure [1].

By the late 1980s, other researchers also began to examine the effects of situations on blood pressure and heart rate [4,20,23]. Along these lines, for example, it was thought that some job or workplace characteristics might be rated as more stressful than others, and that workers exposed to such conditions, rather than to low-stress job conditions, were considered at greater risk for the development of hypertension. This is in marked contrast to models in which researchers focused upon trait characteristics such as type A (i.e., coronary-prone) personality [31] or anger-expression style [32].

In situational-risk models, rather than assigning participants to groups on the basis of a *person* dimension such as resting blood pressure status (i.e., normotensive vs. hypertensive), or reactivity status (high vs. low reactors), participants are randomly assigned to stress expo-

sure under different conditions. For example, several studies have reported differential effects on reactivity due to manipulations of controllability of task outcomes (a characteristic of the situation) [33,34]. In a different line of research, we and others have shown that the presence of a supportive person during exposure to stress [25,26] can affect the magnitude of the blood pressure and heart rate response, although not all studies have shown this [e.g., 35]. In these studies, person dimensions were ignored; it was the effects of exposure to some aspect of the environment that were assessed. These situational manipulations represented an important development in the reactivity model, because, unlike the person models, they directly addressed the issue of exposure. In summary, this model argues that some people are at risk of future hypertension because they are frequently exposed to situations that produce exaggerated reactivity (e.g., high-stress jobs). Thus, model 2 does not depend on the generalizability of individual differences in reactivity.

Reactivity as a person-by-situation cause of hypertension (model 4)

It is likely that many factors—person and situation—contribute, perhaps interactively, to the disease process. Some individuals are thought to be predisposed to display larger blood pressure and heart rate reactivity, operating through one or another personality dimension, such as anger expression or hostility. Such exaggerated reactions may only occur, however, when the person is exposed to circumstances that are relevant to this trait (e.g., in a frustrating situation). Thus, some researchers have moved from purely person- or situation-based models to a person-by-situation model. One possible reason for this transition is the poor generalizability from laboratory to real life discussed earlier. An example of the person-by-situation model is provided by studies in which participants were ranked on the trait dimension “cynical hostility” [36,37]. Persons who score high on this dimension are purported to have a general mistrust of others, and to view others’ motives with suspicion. Ratings of hostility derived from the Type A Structured Interview [31] and scores on the Cook–Medley Hostility Scale [38] have been found to predict subsequent cardiovascular and other cause morbidity and mortality [32]. Because of the association with heart disease, researchers have considered the relation between hostility and cardiovascular reactivity [36,37,39]. In two early studies in this area, hostility scores were not associated with cardiovascular reactivity to standard laboratory tasks [36,39]. In a later study, Suarez and colleagues [37] also found that participants high in trait hostility did not evidence greater blood pressure and heart rate responses to a laboratory stressor (an anagram task) when the task was performed alone. How-

ever, a second group of participants was exposed to the anagram task, but with a confederate who harassed participants about their performance. In contrast to the “alone” condition, during social interaction (i.e., the “harassment” condition) participants rated high on trait hostility evidenced larger blood pressure responses than low-hostile participants. Thus, it was necessary to know about characteristics of both the situation and the person in order to predict the cardiovascular response.

Another example of the usefulness of the person-by-situation interaction model was provided by Bohlin et al. [40]. They compared blood pressure reactivity between normotensive and borderline hypertensive participants (a *person* factor) in response to a mental arithmetic task. However, they manipulated a *situational* element: the degree of control the participants had over the way in which the task proceeded. In one condition, participants were given control over the pace at which arithmetic problems were presented (high control). For the other group of participants, however, the pacing was determined externally (low control). In fact, the actual pacing was matched between the two conditions, so that objectively, the two groups were exposed to identical stressors. There were no differences in blood pressure response between the two groups in the low control (externally paced) condition; however, the borderline hypertensives evidenced greater systolic responses than the normotensives in the high control condition. Again, neither the situational factor alone, nor the person factor alone, elicited effects; it was their interaction that was the key.

Model 4 was an important development because it takes into account the limited generalizability of individual differences, while preserving the predictive utility of such constructs. Thus, the situation-based model (model 3) assumes that every individual in a high-stress job is at increased risk of hypertension; the person-based models (models 1 and 2) assume that the people who show large responses to one stressor will show larger responses to all stressors. The interactive model (model 4), however, does not rest upon these assumptions, but allows one to predict increased risk for hypertension development on the basis of the person–environment fit.

Reactivity model—as it appears today

The 70 years of cardiovascular reactivity literature reveal several controversies, and many of these remain unresolved. There is little consensus, for example, on the specific physiological mechanisms involved in the development of hypertension. In addition, we and others have suggested that reactivity—the response that occurs while the stressor is present—may not be the most important aspect of the stress–hypertension relation. Recovery of prestress resting levels, we suggest,

may be an equally important dimension [41], but tends to receive relatively little attention, partially, we suspect, because it presents measurement difficulties that are less problematic when one focuses on reactivity [42].

An important issue in this area remains the generalizability of blood pressure responses observed in the laboratory (where reactivity is evaluated) to the natural environment. The available evidence suggests that this aspect of the reactivity model is not well supported [4,23,24].

For purposes of presenting past and current reactivity models, we have limited the discussion of various models to the basic cardiovascular variables—blood pressure and heart rate—which characterized the early studies and still constitute the core measures of most subsequent research. However, techniques have been developed that allow researchers to investigate the underlying hemodynamics of blood pressure reactivity. Although techniques for assessing different underlying hemodynamic activity have been in use since the 1950s (e.g., Wolff and Wolf [12]), these techniques were invasive and not available to most researchers. However, in the late 1970s [30], a noninvasive technique, impedance cardiography, provided substantial impetus for a resurgence of interest in the hemodynamics that underlie blood pressure regulation. This technique allows the calculation of the relative contributions of central mechanisms (e.g., cardiac output) and peripheral mechanisms (e.g., total peripheral resistance) to blood pressure reactions to stress.

In addition to the study of hemodynamic patterning, researchers are also developing tools that allow the examination of the role of sympathetic and parasympathetic activity in blood pressure regulation and in coronary heart disease. Information concerning these processes is inferred, to an extent, from heart rate variability [43]. Studies now indicate that reduced variability of heart rate is associated with heart disease (although its role in hypertension is as yet unknown) [43].

Currently, researchers who are also interested in the pathways through which stress may influence cardiovascular disease focus on a far broader set of measures than we have discussed in this study. Although we have focused solely on hypertension, cardiovascular reactivity is also hypothesized as influencing the development of atherosclerotic plaque, and eventually arterial rupture, which can lead to acute coronary heart disease events [3].

Summary and conclusions

Hines and Brown began, in 1932, with a simple and straightforward question: Can we devise a simple test that will predict future hypertension? [5]. Such a test still does not exist, and that reveals something about the difficulties encountered in such an endeavor. Although

some studies show a relationship between reactivity and blood pressure level, or hypertension status, the observed relationships are not strong enough to allow us to make predictions on a case-by-case basis. However, future models will incorporate a broader range of the cardiovascular process than just reactivity (i.e., cardiovascular recovery, underlying hemodynamic changes, heart rate, and possibly blood pressure variability) and these models may prove to be more powerful predictors of future disease than reactivity alone.

We have labeled reactivity models by their relative focus on person and situation properties, as we believe that future models will continue to expand and elaborate upon the interplay between these two sets of factors. Furthermore, by emphasizing the role that person and situation properties have had in the evolution of reactivity models, we will develop a better understanding of the mechanisms that link stress to the development of hypertension. The focus on the broader hemodynamic processes that underlie blood pressure regulation, as well as a slight shift in focus to include cardiovascular recovery and reactivity, may help in providing an answer to the question of why resting blood pressure levels drift upward in some, but not in others.

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