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Chronic Psychosocial Stress and Hypertension

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Abstract

Genetic and behavioral factors do not fully explain the development of hypertension, and there is increasing evidence suggesting that psychosocial factors may also play an important role. Exposure to chronic stress has been hypothesized as a risk factor for hypertension, and occupational stress, stressful aspects of the social environment, and low socioeconomic status have each been studied extensively. The study of discrimination is a more recent and rapidly growing area of investigation and may also help to explain the well-known racial disparities in hypertension. Research regarding mechanisms underlying stress effects on hypertension has largely focused on cardiovascular reactivity, but delayed recovery to the pre-stress level is increasingly being evaluated as another possible pathway. Recent findings in each of these areas are reviewed, and directions for future research are discussed.

Introduction

The etiology of hypertension remains poorly understood. Genetic and behavioral factors known to be involved leave a substantial portion of variability in outcomes unexplained, and a large body of literature has accumulated evaluating psychosocial stress as another possible risk factor. Broadly speaking, stress is conceptualized as the perception of environmental demands that are believed to exceed one's resources for adapting to the situation [1]. The intensity and duration of exposure are presumed to be important determinants of risk; effects of acute stressors on blood pressure (BP) have been demonstrated, but ongoing exposure to stress may be more plausibly linked to sustained BP elevations and hypertension incidence [2].

The effects of chronic stress in a number of domains are being investigated, including work-related stress, relationship stress, low socioeconomic status (SES), and more recently, race-related discrimination. Associations between each of these and BP outcomes have been reported, but the level of evidence varies, and many questions remain regarding the mechanisms involved, as well as vulnerability and protective factors that may be important in determining the impact of chronic stress on hypertension. Recent findings in each of these areas are reviewed, and directions for future research are discussed.

Occupational Stress

Most adults spend a substantial portion of their lives at work, so it should not be surprising that chronic job stress can have a powerful impact on health. The most widely studied model of occupational stress is the job strain model of Karasek et al. [3], which focuses on two characteristics of the work environment: job demands, or workload, and decision latitude, or the degree of control an employee has in performing his or her work. According to this

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model, the combination of high demand and low control, referred to as *high strain*, produces the most stress. High job strain has been associated with increased ambulatory BP at work, at home, and during sleep, as well as increased left ventricular mass, consistent with the anticipated effects of sustained BP elevation [4]. Overcommitment and an imbalance between effort at work and rewards received, two components of an alternative model of work stress proposed by Siegrist [5], have also been related to BP both during and outside work [6, 7].

A number of longitudinal studies have demonstrated that high job strain is associated with increases in BP and the development of hypertension [8, 9, 10•, 11], though some have failed to show a relationship [12]. Stressful work conditions, like other stressors, can fluctuate over time, and the chronicity of exposure appears to be an important factor in predicting hypertension risk. For example, increasing job strain over an 8-year follow-up period predicted hypertension incidence in 3200 young, healthy, employed participants of the Coronary Artery Risk Development in Young Adults (CARDIA) study [8]. In a study of 8395 white-collar workers in Canada, both cumulative and new exposure to job strain predicted increases in BP over 7.5 years; these effects were stronger among workers with low levels of social support at work [9].

The influence of demand and control on BP has also been demonstrated using an ecological momentary assessment approach, in which subjects' experiences are measured in real time in the natural environment. In a substudy of the Pittsburgh Healthy Heart Project, 340 healthy adults rated various aspects of their daily experiences on an electronic diary every 45 minutes for 6 days, while also wearing ambulatory BP monitors [13]. Ratings of task demand, decisional control, social conflict, negative affect, and arousal were each associated with within-person BP fluctuations, but only demand and control experiences showed significant between-person associations with mean ambulatory BP. These effects were independent of posture, activity, substance use, and resting clinic BP.

In general, effects of job strain on BP tend to be stronger among men than among women [11]. The reasons for this difference are not clear, but one possible explanation is that the specific aspects of work that are stressful for men and women may differ. For example, in a 20-year follow up of the Alameda County Study, job insecurity, unemployment, and low self-reported job performance each independently predicted hypertension incidence among men, whereas low-status work predicted hypertension among women [14]. The interactive effects of job strain and stress in other domains of life may also differ for men and women. In the Double Exposure Study, the impact of high job strain on ambulatory BP change in women was buffered by high marital cohesion (systolic BP decrease of 4.3 mm Hg over 1 year) and exacerbated by low cohesion (systolic BP increase of 3.4 mm Hg) [10•]. The effects of job strain on BP were not modified by marital stress among men in this study.

Social Isolation and Marital Stress

Social relationships are important sources of emotional and practical support and can buffer the negative physical and psychological effects of stress. The lack of supportive relationships not only leaves one without these resources, but can itself be a major source of stress. Social isolation, defined in terms of the size and composition of the social network (eg, marital status, number of close friends and relatives, religious or other group affiliations) has been prospectively associated with cardiovascular disease and all-cause mortality [15]. Isolation has also been associated with delayed post-stress BP recovery [16], which is related to hypertension risk. Though structural support is certainly important, the perception of social isolation may occur even in the context of large social networks. Loneliness, which reflects a discrepancy between one's actual and desired level of social

connectedness, has been associated with cardiovascular function and reactivity to laboratory stress [17, 18], though its influence on hypertension risk has not yet been evaluated.

Of the various elements of the social network, marriage is often the central relationship in people's lives. Married individuals tend to experience better health outcomes than those who are single [19], and other sources of support do not fully compensate for the effects of being single [20•]. On the other hand, relationships can be a source of conflict, and the stress associated with unhappy or strained marriages has been associated with negative cardiovascular effects. For example, acute episodes of marital conflict have been shown to produce BP elevations in laboratory studies. Nealey-Moore et al. [21] studied cardiovascular reactivity to positive, neutral, and negative interactions in 114 young couples. Compared with positive and neutral discussions, negative discussions produced larger increases in BP, heart rate, and cardiac output, and larger decreases in peripheral resistance and pre-ejection period. Significant differences were observed in both men and women while preparing for the tasks, speaking, and listening to their spouse's comments. These investigators demonstrated similar results among 300 middle-aged and older couples who participated in marital disagreement and collaborative problem-solving tasks and also found that the cardiovascular effects of negative interaction persisted into a post-task recovery period [22].

Results of studies conducted in the natural environment are consistent with the laboratory research findings. Grewen et al. [23] found that men and women reporting high relationship quality had lower ambulatory BPs at home and at work, compared with those reporting intermediate or low relationship quality, as well as those without a partner. In a study of mildly hypertensive subjects who were living with a significant other for at least 6 months, the effect of marital quality on BP was modified by the amount of spousal contact [24]. Specifically, greater contact with one's spouse was associated with increases in 24-hour ambulatory BP over 3 years among those reporting low marital quality, and with decreased BP among those reporting high marital quality. Low marital quality, but not spousal contact, also predicted 3-year increases in left ventricular mass. In general, marital stress appears to affect women more strongly than job stress [25] and tends to have a greater negative impact on women than on men [26], though sex differences are not always observed [21].

Low Socioeconomic Status

Numerous indices of SES have been studied, the most common being educational attainment, occupational status, and income; others include social class, social status, and neighborhood characteristics. Although these are generally correlated, each provides unique information and may affect various health outcomes differently. Epidemiologic studies consistently demonstrate graded associations between SES and risk of hypertension, cardiovascular disease, and mortality [27, 28, 29•, 30]. Low SES has also been related to BP patterns that are related to hypertension, including reduced nocturnal BP dipping [31, 32] and delayed BP recovery following laboratory stress [16]. Hypothesized mechanisms of the association between low SES and hypertension include a poorer health behavior profile and greater exposure to stress, as well as the availability of fewer resources with which to cope with stress. In an analysis of the Work Site Blood Pressure Study, Landsbergis et al. [33] found that associations between high job strain and ambulatory BP at work were stronger among participants with low SES versus those with high SES, suggesting that low SES may exacerbate the effects of other chronic stressors.

SES can be highly variable over time, particularly with regard to income, and it appears that BP may be sensitive to these fluctuations. Matthews et al. [34] evaluated changes in several socioeconomic indicators as predictors of incident hypertension in 10-year follow-up of CARDIA study participants. There was a trend for an association between decline in income

and incident hypertension, and difficulties paying for basics at baseline and during the follow-up period each predicted hypertension incidence, independent of standard covariates. Earning a new educational degree, on the other hand, did not significantly affect risk. A smaller study of 160 adults found that improvement in financial strain, defined as difficulty paying bills, replacing needed items, and providing for one's family, was associated with reduced ambulatory BP 3 years later [35].

Neighborhood characteristics reflect another aspect of SES that may influence health. A cross-sectional analysis of 2612 participants from 495 neighborhoods included in the Multi-Ethnic Study of Atherosclerosis (MESA) found that poorer conditions regarding walking environment, availability of healthy foods, safety, and social cohesion were each associated with a greater likelihood of hypertension [36]. Traditional socioeconomic indicators (education, income) were strongly related to neighborhood conditions, but did not account for these effects when included in the analysis. Adjusting for race, however, reduced or eliminated associations between neighborhood conditions and hypertension. Although this suggests that neighborhood may be a proxy for race, others have found that adjusting for socioeconomic factors reduces race differences in BP outcomes [32]. The fact that race and SES are so closely intertwined makes it difficult to determine the extent to which each has independent effects on hypertension, and the answer to this question may depend on the specific socioeconomic indices and outcomes being examined.

Racial Discrimination

African Americans living in the United States suffer from disproportionately higher rates of hypertension than whites, and the reasons for this disparity remain poorly understood. In addition to socioeconomic differences, exposure to racial discrimination has been proposed as a contributing factor [37, 38•]. Discrimination includes negative attitudes and beliefs about members of ethnic or racial minority groups, as well as differential treatment. It encompasses both explicit and more subtle behavior such as unfair treatment, stigmatization, social exclusion, and overt acts of verbal or physical aggression, and can occur at the interpersonal, institutional, and cultural levels [39].

A number of studies have shown that African Americans demonstrate elevated cardiovascular reactivity to race-related laboratory stressors compared with neutral tasks and non–race-related stressors [38•]. Various tasks have been used, ranging from speech tasks requiring subjects to respond to shoplifting accusations, to recall of personally relevant experiences of discrimination, to exposure to audiotapes and videotapes of blatantly racist scenarios. When confronted with more ambiguous situations in which race is not explicitly mentioned, there is evidence that reactivity is influenced by the degree to which the subject makes attributions of race-related discrimination [40, 41], which has been linked to past exposure to racism [42]. Reactivity to these tasks has also been moderated by gender, SES, personality, social support, and coping styles, though not always in a consistent pattern [38•, 39, 43].

Outside the laboratory setting, self-reported exposure to racism has been associated with higher daytime and nocturnal ambulatory BP, as well as reduced nocturnal BP dipping [44, 45]. Although exposure to racial discrimination was not related to hypertension in the Metro Atlanta Heart Disease Study, there was a significant cross-sectional relationship with high stress due to experiences of racism [46]. The only prospective study of racism and hypertension to date is the Black Women's Health Study, in which a large cohort of US black women were followed for up to 4 years [47]. The prevalence of exposure to racism in this sample was high: more than 70% of women reported at least one instance of institutional racism (unfair treatment at work, in housing, or by the police), and almost half

reported at least monthly exposure to interpersonal racism (eg, receiving poorer service than others, others acting as though she is dishonest or not intelligent). Overall, there was no significant relationship between exposure to racism and hypertension incidence, but there was a positive association with interpersonal racism among women born outside the United States. Additional prospective studies are clearly needed to better understand the long-term health effects of racial discrimination in both men and women.

Mechanisms Underlying the Stress-Hypertension Relationship

The impact of stress on the development of hypertension is believed to involve a sympathetic nervous system response, in which release of catecholamines leads to increased heart rate, cardiac output, and BP. Sympathetic responses to acute stress are well documented, but the process by which stress contributes to sustained BP elevation over time is not well understood. It may be repeated activation of this system, failure to return to resting levels following stressful events, failure to habituate to repeated stressors of the same type, or some combination that is responsible for the development of hypertension [48].

In investigating mechanisms of the stress-hypertension relationship, researchers have tended to focus on the physiological changes that occur during exposure to a stressor. The cardiovascular reactivity hypothesis proposes that individuals who exhibit exaggerated cardiovascular responses to acute stressors are at greater risk for hypertension and cardiovascular disease than those who show lesser reactivity. More recent versions of the hypothesis incorporate the degree of stress exposure and the role of genetic vulnerability [49]. Prospective studies of normotensive and borderline hypertensive subjects have shown that cardiovascular reactivity predicts the development of stable hypertension up to 36 years later, using various laboratory stressors and controlling for traditional risk factors [50], though several studies have failed to demonstrate this association [12, 50]. A 20-year follow-up of the Air Traffic Controllers Health Change Study found that BP reactivity to job stress (ie, the number of planes a controller was responsible for at a given time) measured over 5 hours predicted hypertension incidence, controlling for baseline BP and other covariates [51]. Demonstrating this relationship in the context of real-life stress represents an important contribution to the reactivity literature, which is dominated by studies of laboratory stress.

Recent research suggests that the return of BP to the pre-stress resting level may also have a role in the pathogenesis of hypertension. Prospective studies have demonstrated that poor BP recovery following mental stress predicts future BP levels, in some cases more strongly than reactivity [52]. Thus, the physiological responses that allow the body to maintain homeostasis when confronted with a stressor can become damaging when they persist after the need for activation has passed. Rumination, a type of perseverative cognition characterized by repetitive, intrusive, negative thoughts, has been proposed as a mechanism that may delay BP recovery [52, 53]. Stressful events often lead to negative affect, including depression, anxiety, and anger, and to negative thoughts about the events, which, according to this hypothesis, can sustain physiological arousal. Stress-related emotions and thoughts are not limited to those occasions when a stressor is actually present; a substantial amount of time may also be spent in anticipation of future stress and dealing with past stress. For example, Cropley et al. [54] found that teachers reporting high job strain engaged in more ruminative thoughts about work in the evening than teachers reporting low job strain, even after controlling for time spent working at home. If rumination does in fact sustain stress-related physiological arousal, this may help to explain the observed carryover effects of job strain on BP outside the work setting.

Several elements of a link between rumination and BP have been documented. Thinking about a marital conflict has been shown to produce cardiovascular arousal among women reporting marital distress [55]. Glynn et al. [56] found that when subjects returned to the laboratory after a stress session and were directed to simply think about the prior session, BP increased significantly, whether they returned 30 minutes or 1 week later. The same investigators also demonstrated that post-stress BP recovery was delayed only during emotional (vs nonemotional) stressors, that a brief post-stress distraction task accelerated BP recovery, and that during a "directed rumination" period, BP drifted upward [57]. These findings are consistent with several other studies that have demonstrated effects of rumination on delayed cardiovascular recovery following anger-provoking laboratory stressors and have shown the ability of distraction to speed recovery, presumably by interrupting the ruminative process [53, 58].

Rumination is qualitatively distinct from other cognitive coping strategies such as problem-solving or reappraisal, in that it involves an unproductive focus on the causes and consequences of a negative mood. There is evidence supporting the notion that the dysfunctional style of thinking that characterizes rumination—not just the process of thinking about stress—is what influences mood and BP. In a study of female undergraduates, Ray et al. [59] found that subjects directed to ruminate following an anger recall task reported feeling angrier and exhibited greater sympathetic activation than those who were directed to reappraise the event ("try to see this event from the perspective of an impartial observer"). Similarly, self-focused cognitive processing of stressful events has been shown to generate and maintain negative mood [60] and BP elevation [61] to a greater extent than distanced, reflective processing.

Much of the work to date regarding effects of rumination on BP has used laboratory stressors; though this approach is useful and important, the artificiality of the situation limits the generalizability of results to the real world. Response to laboratory stressors may be a poor indicator of the degree to which people ruminate about real-life stressors. Further, in the laboratory, the participant is "forced" to confront the stressor being studied, whereas in the field, the person may use various strategies (eg, distraction) to avoid such engagement. Thus, an important next step in evaluating rumination as a possible mechanism by which stress contributes to hypertension is to observe the effects of naturally occurring stressors on ruminative thoughts and BP outside the laboratory.

Conclusions

Overall, there is growing empirical support for the hypothesis that exposure to chronic psychosocial stress contributes to the development of hypertension. Additional prospective studies are needed before a causal relationship is established, particularly in the case of discrimination. Evidence of sex and race differences highlights the importance of identifying the types of stressors that are most harmful for various groups, as well as factors that may exacerbate or buffer the effects of stress. Laboratory studies have provided convincing preliminary evidence that thinking about stressful events, in addition to experiencing them directly, can delay BP recovery. Further research is needed to determine whether rumination prolongs cardiovascular responses to real-life stressors and is prospectively related to sustained BP elevations.

A recent meta-analysis of various stress reduction interventions including biofeedback, progressive muscle relaxation, stress management training, and transcendental meditation, found that only transcendental meditation was associated with significant BP reductions [62]. Meditation has been shown to reduce rumination to a greater degree than somatic relaxation training [63], which may help to explain its greater effectiveness in reducing BP.

Thus, targeting factors that influence the impact of stress on BP, such as coping responses, may be a useful intervention strategy, particularly when exposure to stress cannot be avoided or reduced. If future research supports the role of rumination as one of the mechanisms by which exposure to stress increases risk for hypertension, an important next step will be to examine whether modifying ruminative tendencies does in fact improve BP recovery following stressful events and reduce the risk of hypertension.

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