and avoided wilting even though it had a greater leaf area.

Water use efficiency, defined as milligrams of CO_2 fixed per gram of water transpired, increased dramatically with increasing CO_2 concentrations for the species tested (Fig. 1). Although plants grown at high CO_2 levels were larger, they used available soil water at a much lower rate because of stomatal closure. For corn, water use efficiency was improved in CO_2-enriched atmospheres through lower transpiration rates. For soybeans and sweetgum, enhancement of photosynthesis also contributed to this improvement.

The leaves of soybeans, pine, and sweetgum thickened steadily as CO_2 levels rose. At 910 ppm CO_2, leaf thickness in these three species was 131, 110, and 121 percent of the control values, respectively. Thickness increased in all of the cell layers of pine and sweetgum leaves. In soybeans the greatest effect was the appearance of a well-developed third layer of palisade cells. As CO_2 levels increased for soybeans, more nodes, earlier anthesis, and less floral abscission were observed. Germinability of soybean seeds was not affected, however. Leaf thickness, node number, and reproduction in corn were not significantly altered by the CO_2 treatments.

These findings suggest positive growth responses to CO_2 enrichment for the agronomic and forest species studied. Further studies are needed to improve the experimental system and to provide additional data on the effect of CO_2 enrichment on vegetation.

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Psychological Stress Induces Sodium and Fluid Retention in Men at High Risk for Hypertension

Abstract. *Exposure to competitive mental tasks significantly reduced the urinary sodium and fluid excreted by young men with one or two hypertensive parents or with borderline hypertension. In this high-risk group, the degree of retention was directly related to the magnitude of heart rate increase during stress, suggesting common mediation by way of the sympathetic nervous system. Thus, psychological stress appears to induce changes in renal excretory functions that may play a critical role in long-term blood pressure regulation.*

Psychological stress has long been hypothesized to be one of the set of interacting elements that may influence the development of primary hypertension in humans, but its role remains unclear (*1*). Certain authorities contend that a critical factor in the establishment of hypertension is the failure of the kidneys to maintain blood pressure (BP) within normal limits by excreting sufficient salt and water (*2*). Abnormal retention of sodium may contribute to hypertension in other ways as well (*3*). Studies in the dog (*4*) and rat (*5*) show that psychological stress reduces renal excretion of sodium and fluid. This stress-induced retention is greater among young spontaneously hypertensive rats than among rats with no genetic predisposition for hypertension (*5*). Thus, such retention may be one mechanism whereby stress contributes to the pathogenic process, particularly in individuals with genetic or acquired predispositions.

In humans, evidence that psychological stress may induce sodium and fluid retention is primarily indirect. Reports that stressful mental tasks increase plasma renin activity and decrease renal blood flow in certain individuals, particularly those with one or two hypertensive parents and those showing high heart rate responses to stress, suggest but do not demonstrate that excretion of sodium and fluid may be reduced (*6–8*). High heart rate response in humans reflects enhanced sympathetic nervous system activity during competitive and other mental tasks (*7*); in animals, stress-induced sodium retention is sympathetically mediated (*4, 5*). The purpose of the present study was to determine whether exposing young men to competitive mental stress reduces their excretion of sodium and fluid, and whether any observed reductions are greater among those with known risk factors for hypertension [for example, parental or borderline hypertension (*9, 10*)] or those demonstrating high heart rate responses to stress.

Forty male college students (18 to 22 years old) participated in the study, 24 of them being subjected to the "stress" and 16 to the "nonstress" condition. All had resting diastolic pressures less than 90 mmHg and no clinical signs of any cardiovascular or renal disorder. During the 5-hour experiments, high rates of fluid excretion were established and maintained by requiring the subjects to drink 1 liter of water during the first hour and 200 ml every 30 minutes thereafter. Urine collections were obtained by voluntary voiding every 60 minutes; the subjects were reminded each time to empty their bladders completely. Samples from each collection were assayed in duplicate for sodium concentration (Beckman Electrolyte 2 Analyzer) and osmolality (Model 3DII Digimatic Osmometer, Advanced Instruments). Sodium concentration was multiplied by fluid excretion rate to derive sodium excretion rate. Cardiovascular measures were sampled during each of the last 3 hours. Each sample included 5 minutes of heart rate, scored from the electrocardiogram (EKG) in beats per minute, and four to six BP determinations obtained by means of a remotely controlled inflation cuff with a Narco Korotkoff Sounds microphone fixed on the left brachial artery. The EKG, cuff pressure, and Korotkoff sounds were recorded on a Beckman Dynograph located in an adjacent room.

The subjects were separated into high risk (HR) and low risk (LR) groups depending on the presence or absence of borderline systolic hypertension (*11*) or parental history of hypertension (*12*). In the nonstress condition, nine LR and

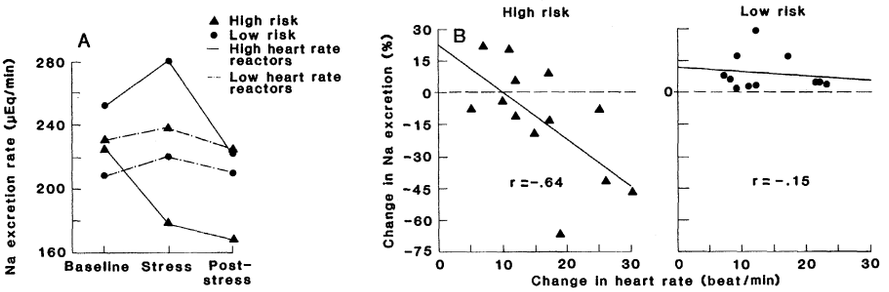

Fig. 1. (A) Actual rates of sodium excretion during baseline, stress, and post-stress periods. (B) The relation between heart rate increases during stress and changes in sodium excretion for HR (N = 13) and LR (N = 11) subjects. Lines of regression (solid lines) and Pearson correlation coefficients (r) are indicated. Dashed lines separate subjects showing increases in excretion during stress from those showing decreases in excretion. Note that none of the LR subjects show a decrease in sodium excretion whereas 9 of 13 HR subjects do show decreases.

seven HR subjects (including two with borderline hypertension and five with parental hypertension) rested or read while seated in a quiet room throughout the experiment. By hours 3, 4, and 5, both their fluid excretion [8.5 ± 0.5 (mean ± standard error), 8.7 ± 0.4, and 8.0 ± 0.5 ml/min, respectively] and sodium excretion had stabilized at high and uniform rates (220 ± 9, 209 ± 12, and 210 ± 13 μEq/min, respectively). Repeated measures analyses of variance (Bimed Program BMDP2V) verified that no significant changes had occurred over these 3 hours in sodium or fluid excretion rates or in BP levels for either HR or LR subjects (all probabilities > .40), although heart rates had decreased slightly over time (63 ± 3, 58 ± 2, and 57 ± 2 beats per minute, respectively, Greenhouse-Geiser P < .01).

On the basis of this stability, for the stress condition, hour 3 was designated as the baseline, hour 4 as the stress period, and hour 5 as the post-stress period. The procedure was identical to that used in the nonstress condition except that during hour 4 the subjects were exposed in pairs to competitive tasks in which the subject who recognized a target stimulus and pressed a telegraph key faster than his competitor won small money incentives. To maintain competitive effort, we initiated new variations of the task every 6 minutes. The simplest task required the subjects to respond whenever a clearly audible tone occurred, while the most complex required them to listen to tape-recorded arithmetic problems and respond only when the solution presented on the tape was incorrect. To minimize motor activity, the subjects were instructed to move only their right index fingers and to keep the rest of their bodies relaxed and still. During this hour, heart rate was monitored continuously except when new instructions were being read, and 22 to 34 BP determinations were made for each subject. By averaging these data, we obtained mean heart rate, systolic blood pressure (SBP), and diastolic blood pressure (DBP) for the entire stress period.

In the stress condition, 11 LR and 13 HR individuals (one with borderline hypertension, nine with parental hypertension, and three with both risk factors) were tested. During the baseline period, no significant differences between HR and LR subjects were evident for heart rate (64 ± 3 compared with 66 ± 3 beats per minute), SBP (132 ± 3 compared with 129 ± 3 mmHg), DBP (82 ± 2 compared with 83 ± 2 mmHg), sodium (235 ± 29 compared with 229 ± 20 μEq/min), or fluid excretion (9.6 ± 0.3 compared with 9.7 ± 0.5 ml/min).

To assess the effects of stress, we further divided these groups into high and low heart rate reactors according to whether their mean heart rates during the competitive tasks exceeded baseline rates by more or less than the median, 13 beats per minute. Substantial stress-induced reductions in sodium and fluid excretion were shown only by HR subjects who were high heart rate reactors to stress (−27 and −35 percent from baseline periods, respectively) (Table 1 and Fig. 1A). These reductions persisted into the post-stress period (−28 and −29 percent from baseline periods, respectively). All other groups showed slight increases in sodium excretion and no consistent changes in fluid excretion during stress.

Figure 1B depicts each subject's change in sodium excretion during stress in relation to his concomitant increase in heart rate. Among HR subjects, greater decreases in sodium excretion were consistently associated with greater increases in heart rate (r = −.64, P < .05). No significant relation between sodium excretion and heart rate response was evident for LR subjects (r = −.15). Changes in fluid excretion and heart rate during stress were similarly related for HR (r = −.55, P < .05) but not for LR subjects (r = −.13).

For most of the subjects showing sodium and fluid retention, the present evidence suggests that stress alters either the glomerular filtration rate or the tubular reabsorption of sodium or both. In 12 of the 13 HR subjects, changes in sodium and fluid excretion were highly correlated (r = +.89) and urine osmolality remained essentially unchanged, indicating no selective reabsorption of water such as may result from increased circulating vasopressin (*13*). Only one subject showed a possible vasopressin effect, since he reduced fluid excretion much more than sodium excretion (−66 percent as opposed to −13 percent), which

Table 1. Cardiovascular and renal excretory responses during mental stress. The data show mean heart rates and blood pressures (± standard error) during stress and mean percentage changes from baseline periods for sodium and fluid excretion rates.

Group	Heart rate* (beats per minute)	Blood pressure (mmHg)		Change in excretion (%)	
		SBP*	DBP*	Sodium†	Fluid†
Low risk					
Low heart rate reactors (N = 6)	74 ± 3	143 ± 5	91 ± 3	+6 ± 2	−9 ± 13
High heart rate reactors (N = 5)	87 ± 6	159 ± 5	93 ± 2	+12 ± 5	−5 ± 15
High risk					
Low heart rate reactors (N = 6)	69 ± 3	142 ± 4	87 ± 2	+4 ± 6	+4 ± 6
High heart rate reactors (N = 7)	88 ± 4	159 ± 7	93 ± 3	−27 ± 10	−35 ± 11

*On the basis of analyses of variance indicating a main effect of heart rate reactivity, high heart rate reactors showed significantly higher heart rates (P < .001) and SBP (P < .05) and a trend toward higher DBP (.05 < P < .10) than low heart rate reactors across both risk groups. Results of these analyses were the same with or without adjustment for slight baseline differences using analyses of covariance. †On the basis of analyses of variance indicating an interaction between the risk group and heart rate reactivity factors and on subsequent mean comparisons with the use of Scheffés statistic, HR subjects who were high heart rate reactors showed significantly greater decreases in sodium excretion (P < .01) and fluid excretion (P < .05) than the other three subgroups combined.

led to a dramatic increase in urine osmolality (+148 percent). Another humoral factor, aldosterone, is also unlikely to be a mediator of the observed sodium retention because of the rapid onset of this response and the fact that potassium excretion did not increase (*14*) but instead decreased in parallel with sodium excretion (−38 percent from a baseline of 58 μEq/min for the high heart rate reactors in the HR group).

Since heart rate responses to this type of task reflect sympathetic activity (*7*), the relation between heart rate response and decreased sodium excretion obtained for HR subjects suggests a common mediation by the sympathetic nervous system. This interpretation is reinforced by the results of studies in the dog (*4*) and the spontaneously hypertensive rat (*5*) demonstrating that stress-induced sodium retention is abolished by infusion of propranolol or by surgical destruction of the renal sympathetic nerves. However, the lack of sodium retention shown by the LR subjects who were high heart rate reactors indicates that cardiovascular and renal sympathetic responses do not always show parallel changes.

Sodium retention mediated by the renal nerves has been linked to hypertension development in the spontaneously hypertensive rat; renal denervation reduces this sodium retention and delays the pathogenic process (*15*). Similarly, long-term increases in BP have been shown to develop in dogs exposed to daily shock-avoidance combined with saline infusion, although neither the stressful task alone nor the saline alone led to any change in baseline BP (*16*). The data for spontaneously hypertensive rats indicate that the tendency to retain sodium has a genetic basis and contributes to hypertension development. The fact that all but one of the men who showed acute sodium retention in the present study had a hypertensive parent leads to the speculation that the tendency to retain sodium during stress may be familial in man as well, and that it may reflect a predisposition to develop hypertension.

Future investigations of human renal excretory responses with the use of sympathetic antagonists such as propranolol should provide more direct evidence of the role of sympathetic activity in mediating stress-induced sodium retention. Ideally, such studies should also assess possible changes in glomerular filtration rate, renal blood flow, plasma renin activity, and vasopressin. Other investigations are also necessary to evaluate the possible relationship between such retention and development of hypertension. These studies could include additional research with animal models during chronic exposure to stress, and also the lengthier but more definitive long-term follow-up investigations in man.

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11. Borderline hypertension was defined by a mean SBP of 140 mmHg or greater, based on eight readings intermittently above and below 140 obtained with the subject seated during a rest period lasting at least 1 hour (*10*).
12. Information on parental normotension or hypertension was obtained by contacting the parents of the subjects. Of the 80 parents, 76 (95 percent) reported that they had been medically examined during the previous year, and all those reporting hypertension described appropriate diet control or drug treatment. Only two subjects, including one in the stress condition, had two hypertensive parents.
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Conditioned Cues Elicit Feeding in Sated Rats: A Role for Learning in Meal Initiation

Abstract. *Pavlovian conditioning was used to teach rats an association between an arbitrary external cue and food. Presentation of the conditioned cue elicited feeding by sated animals. The meal constituted approximately 20 percent of daily intake, and it was compensated for by a reduction of subsequent intake.*

Several hypotheses regarding the physiological mechanisms underlying the initiation of feeding share the assertion that organisms begin to feed in response to a state of energy depletion. The hypotheses differ in the specific physiological events identified as reflecting the state of energy balance and assumed, therefore, to be instrumental in meal initiation (*1*). Recent analyses of food intake control systems indicate, however, that organisms eat not only as a result of energy demands but also in response to other factors not directly associated with energy depletion. Some recent theoretical treatments of human feeding behavior stress that learned social and cognitive factors are critical in determining when an organism will eat and sometimes dominate regulatory signals linked to the state of energy balance (*2*). Little convincing experimental evidence exists, however.

I have tested the hypothesis that external cues that, through a process of Pavlovian conditioning, have become associated with feeding determine when a

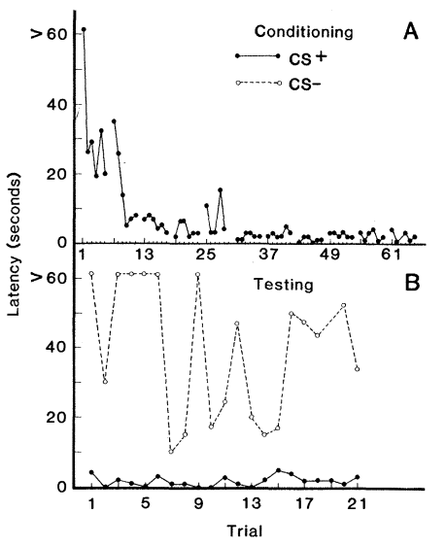

Fig. 1. Group median latency to initiate feeding after the delivery of liquid diet into the food cup (A) during conditioning and (B) during testing.