with retreat of one; and (iv) intense fighting resulting in injury to, or death of, one combatant.

A fight may be initiated by either individual by confronting the other with outspread front tarsi. The challenged male either backs away or responds by raising and spreading apart his forelegs and extruding his cheliceral stylets.

The combatants circle and rush each other, flailing their forelegs and jousting with their extruded stylets. There is much pushing and grappling. Mites often use their palpal glands to apply strands of silk to the mouthparts and legs of the opponent. An individual may be so badly entangled that his movements are impeded and he retreats to clean himself, thus ending the encounter, at least temporarily.

Rarely, one male punctures the other's integument with his stylets. Males injured in this way are crippled and death usually results. Although very few agonistic encounters of the fourth type were observed in progress, dead deflated males were not unusual in the vicinity of guarded females.

Size is important in fighting. We selected 20 decisive fights and compared winners and losers with regard to size of three length characters (Table 1). On the basis of these criteria, winning males are consistently larger.

A successful combatant returns to the preferred position on top of the quiescent female. The male in possession of the female at her ecdysis will have the first opportunity to mate with her. If a resident male is unsuccessful in driving away intruders he is either replaced or he becomes one of two or more co-guarders at the female's ecdysis and competes for access to the female when she emerges. The reproductive advantage of solitary guarding was measured. Seventy active albino (a recessive trait) female deutonymphs were placed alone on leaf disks. After the females became quiescent (at 6 hours), a single male, alternately albino and wild type, was introduced on the disk (at 12 hours). Later (at 24 hours), a second male of the opposite genotype was placed on each disk. The relationship between the males and the quiescent female was checked every half-hour until the female's emergence, and all instances of guarding and co-guarding were recorded. After ecdysis and mating, the females were left to oviposit and the males were removed. The progeny were reared, enabling us to determine their sex and phenotype. Albino daughters indicated a mating with an albino male; wild-type daughters resulted from a wild-type father. Males who guarded alone during the final half-hour before ecdysis were almost always successful in mating (93 percent). Males who occupied the preferred position on top of the female but tolerated co-guarders had a reduced frequency of mating success (63 percent) (*10*).

It is likely that, within a spider mite colony, male aggression does not generate any specific dominance order but rather gives rise to a random series of confrontations. Guarding pharate females represents a form of ephemeral territoriality; the procurement and retention of such a territory is to a large extent dependent on fighting ability.

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11. We thank J. F. Downhower and G. W. Wharton for helpful criticism. The Sambucus strain was obtained from W. Helle, Laboratory of Applied Entomology, Amsterdam. Supported by training grant 5 T01 AI00216 from the National Institute of Allergy and Infectious Diseases.

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Genetic Predisposition and Stress-Induced Hypertension

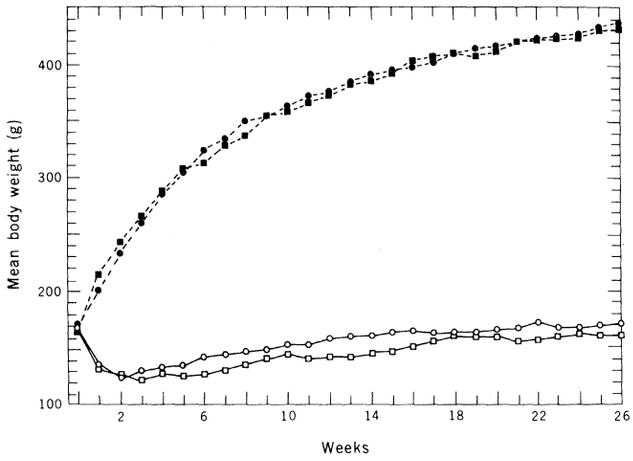
Abstract. *When chronically exposed to an approach-avoidance conflict, rats with a genetic susceptibility to hypertension showed persistent elevations in systolic blood pressure, but rats with a genetic resistance to hypertension did not. Hence, psychic stress is selectively efficacious in producing hypertensive effects depending on genetic predisposition of the animal.*

The etiologic significance of psychic stress in hypertension is still controversial. However, despite some conflicting results and methodological difficulties, animal experiments indicate that appropriate psychic stress can result in sustained but usually moderate increases in blood pressure with associated anatomical pathological changes (*1*). A neglected factor in previous research is the genetic predisposition of the experimental animals. The absence of reported attempts to investigate the interaction of psychic stress and genetic predisposition is surprising, especially since most stress studies have been done on rats, the same animal used in developing genetic models for hypertension.

Dahl *et al.* (*2*) developed two strains of rats in this laboratory by selective inbreeding of Sprague-Dawley rats according to their blood pressure response to salt ingestion. The Dahl hypertension-sensitive strain (S) rapidly and predictably develops severe fatal hypertension upon excess salt ingestion whereas the Dahl hypertension-resistant strain (R) responds mildly or not at all. Using this rat model Dahl's group found that other putative hypertensinogenic stimuli were also selectively efficacious depending on genetic predisposition (*2*). Unlike the spontaneously hypertensive rat (*3*), S strain rats remain normotensive when not exposed to a specific hypertensinogenic stimulus.

Despite their genetic predisposition, when S rats were exposed to aversive Pavlovian conditioning procedures—that is, electric shocks were administered independent of the subjects' behavior—no hypertension resulted (*4*). However, exposure of S rats to an aversive operant conditioning schedule involving a food-shock conflict did result in persistent, occasionally severe elevations in blood pressure. The food-shock conflict also resulted in severe food deprivation and frequent electric shocks. When S rats were exposed to identical amounts and patterns of either food deprivation or electric shocks or both but independent of their behavior, they did not exhibit blood pressure elevations as persistent or severe as those of the conflict-exposed rats (*5*). This indicates that exposure to conflict is more hypertensinogenic than simple exposure to aversive events.

In the experiment reported here, both S and R rats were exposed to the food-shock conflict in order to determine whether the effects of this type of stress depend on genetic predisposition. Beginning approximately 3 weeks after weaning, male rats were exposed either 5 or 7



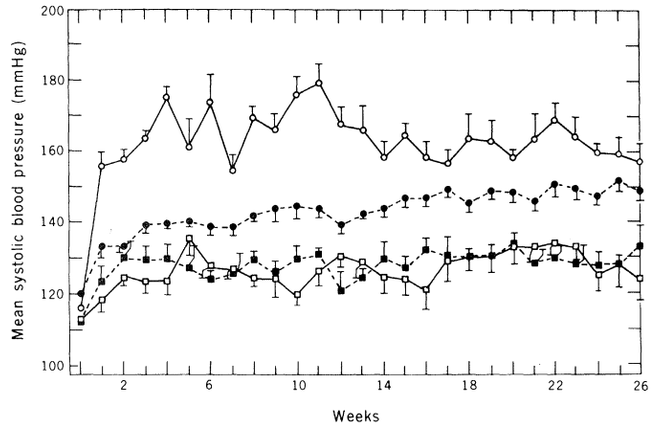


Fig. 1 (left). Mean body weights of S rats (open circles) and R rats (open squares) exposed to the food-shock conflict and S (closed circles) and R (closed squares) free-feeding controls. Fig. 2 (right). Mean systolic blood pressure of S rats (open circles) and R rats (open squares) exposed to the food-shock conflict and S (closed circles) and R (closed squares) free-feeding controls. Vertical lines indicate standard errors.

days each week for 6 hours to the following regimen. They were required to press a lever in order to obtain a 45-mg food pellet (*6*) under a 50-second variable interval schedule, but pressing the lever resulted also in brief electrification (1.0 ma) of the grid floor of the cage under a variable-ratio schedule (*7*). Since S rats develop hypertension in response to salt, specially prepared food pellets containing less than 0.4 percent sodium chloride were used throughout the experiment.

Because food was generally not otherwise available, the rat had to obtain it by means of the lever-press response. Both food and shock were presented under variable random schedules, which prevented the rat from determining whether any particular response would result in food, shock, neither, or both. The subjects were exposed to this regimen for 26 weeks while age-matched controls were maintained with free access to food without shock in the same room. Each week each subject and control was weighed and had its systolic blood pressure measured by tail plethysmography under light ether anesthesia by the method of Friedman and Freed (*8*).

Exposure to the food-shock conflict resulted in dramatically low lever-press response rates (*9*), manifested in extremely low body weights (Fig. 1). The mean body weights of S and R rats exposed to conflict did not differ significantly during the entire experiment. Hence, since food intake and, therefore, body weight depended on lever pressing, it is reasonable to assume that rats of the two strains found the conflict equally aversive. Likewise, vocalizations, ambulation in the experimental cage, and reactivity to handling during the experiment were the same in both strains.

In contrast to these behavioral similarities, the two strains showed dramatically different systolic blood pressure responses (Fig. 2). An analysis of variance revealed highly significant main effects of strain, treatment, and weeks of the experiment ($P < .001$). The strain-treatment interaction was also highly significant ($P < .001$). Appropriate pairwise comparisons indicated that throughout the experiment, despite considerable variability, the mean blood pressure of S rats exposed to conflict was always significantly higher than that of S controls ($P < .01$) whereas R rats exposed to conflict were never significantly different from R controls.

It is not clear at this juncture how these results might be incorporated into the current body of knowledge concerning the relation between stress and hypertension. Either increased neurogenic vasoconstrictor activity or increased vascular responsiveness to normal vasoconstrictor influences are believed to be responsible for the initiation of hypertension. In this regard, S rats do exhibit greater vascular reactivity to pressor agents such as norepinephrine and angiotensin (*10*).

Psychic stress, like other hypertensinogenic stimuli, appears to be selectively efficacious depending on genetic predisposition. This may help explain why the role of stress in hypertension has been such an enigma. The biochemical and physiologic mechanisms involved in the differential stress-induced blood pressure elevations remain to be determined. However, despite their genetic susceptibility, the S rats exposed to stress showed blood pressure elevations less severe than those shown by even unselected rats exposed to physiologic insults such as excess sodium ingestion or the Goldblatt clamp. Hence, although the present results indicate that psychic stress can, in certain predisposed organisms, cause prolonged elevations in blood pressure, there is still insufficient evidence to allow attributing to stress a primary etiologic role in essential hypertension.

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