Hypertension and Death from Consumption of Processed Baby Foods by Rats[1] (34700)

LEWIS K. DAHL, MARTHA HEINE, GEORGE LEITL, AND LORRAINE TASSINARI
Medical Research Center, Brookhaven National Laboratory, Upton, New York 11973

In 1963 we reported (1) that experimental hypertension had been induced in a few female rats fed commercial baby foods. Analyses by ourselves and the U.S. Department of Agriculture (2) indicated that such processed baby foods contained concentrations of salt (NaCl), added by the processors, greatly in excess of the meats and vegetables from which they derived. Viewed in the light of earlier correlations between chronic salt ingestion and hypertension [for review see (3)] this pilot experiment appeared relevant to man: it suggested that a high intake of salt in infancy could enhance the risk of developing hypertension. Since current feeding practices in the U.S. promote the chronic consumption of amounts of NaCl well in excess of the metabolic requirements of infants (4–7), our concern increased when we found that young rats developed permanent, even fatal, hypertension after brief exposures to excessive dietary salt (8). The increasing awareness of delayed injury from chronic exposure to "innocuous" environmental agents stimulated us to confirm and extend our pilot experiment.

Materials and Methods. The protocol of the current study was identical with the original one (1) except that more animals of both sexes were observed for longer times. Forty weanling rats from our hypertension-prone strain (9) were distributed randomly between test and control series. The 25 test rats (14 male, 11 female) were fed only baby foods consisting of processed meats and vegetables purchased on the open market. The 15 controls (7 male, 8 female) were littermates of the test animals and ate our special chow pellets containing 0.4% NaCl (9). Both groups received their respective diets and tap water *ad libitum*. Blood pressures were measured at 2- to 4-week intervals by a standard tail plethysmograph method (8). Our analyses of the baby foods confirmed the earlier findings, namely about 150–500 mg %, as sodium.

Results. The results are summarized in Fig. 1. In brief, these baby foods induced hypertension which was fatal in about half of the rats. By the end of the first month, the average blood pressure in the group eating the baby foods was significantly higher than that of the controls, 130.6 vs. 119.0 mm Hg, respectively, ($p < 0.0002$). The difference increased thereafter ($p < 0.00001$). The maximum difference in means emerged after 6 months at which time the pressures averaged 190.2 versus 138.2 mm Hg for test and control groups, respectively. The slight decline in average pressures in the test group at 7 and 8 months (180.0 and 186.0 mm, respectively), was due to the intercurrent deaths of those with the highest pressures. The experiment was terminated after 8 months because all of the 25 rats on processed baby foods had developed hypertension and about half were dead. The first test animal died between the fourth and fifth months followed by 11 more during the next 2 months; another 2 were seriously ill at the end of the study. In contrast, among their littermates serving as controls, most pressures at 8 months were clustered around 140 mm Hg and the group average was 141.4 mm Hg. One control animal had developed a chronic respiratory infection but the other 14 appeared in good health.

Body weights of the test group were significantly less ($p < 0.05$) during the first 2 and 3 months for both females and males but not

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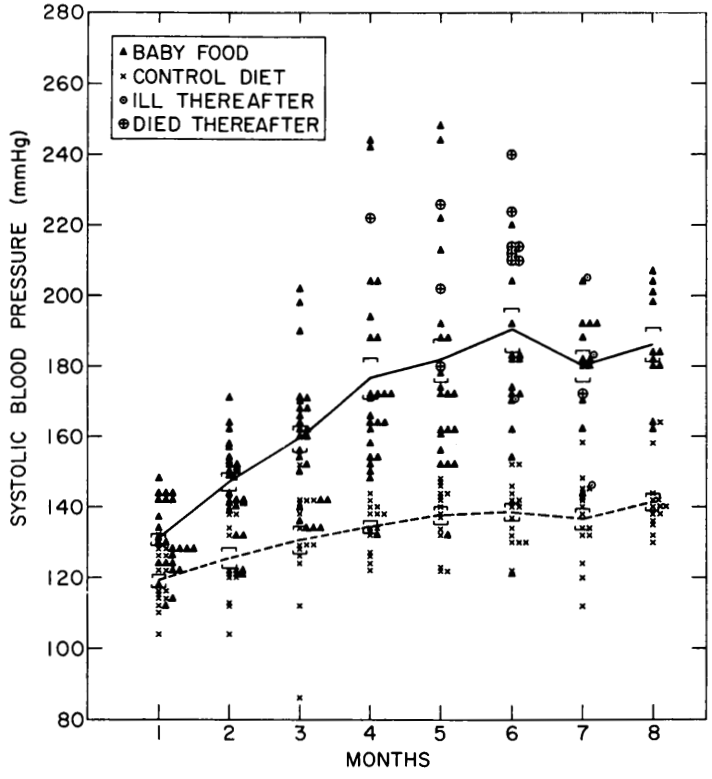

FIG. 1. Effect of processed baby foods on blood pressure of hypertension-prone rats. The mean blood pressures were computed without regard to sex because there were no consistent differences between the responses of the two sexes. Horizontal brackets above and below mean values indicate ± 1 SE. The average pressures of the test group were significantly higher ($p < 0.0002$–0.0001) than those of the control group at every period. Blood pressure shown for animals that died or became ill are the last recorded when animal appeared in good health.

thereafter. This may have been due to a lower average food consumption by the test rats in which case their NaCl intake was correspondingly decreased during this same period.

Discussion. Of necessity, the control diet differed by more than one variable from the experimental one: processed baby foods without added salt were not being manufactured at the time this study was initiated. Even today they are not readily available. We did not make our own baby foods with NaCl as the sole variable, since we wanted to test the baby foods *actually eaten* by American infants. It is conceivable, therefore, that differences in the diet other than the NaCl content were responsible for the serious effects observed in these animals. In similar experiments where both groups received rat chow with NaCl as the only variable, however, the results were equivalent to those reported here. Whatever the explanation, the fact remains that all of the test rats became hypertensive and half of them died. Consumption of processed baby foods caused hypertension and death. This is in sharp contrast to approximately 10,000 rats from this same strain studied during the last 9 years. In our studies on these hypertension-prone rats, we have sometimes observed "spontaneous" development of mild hypertension even on a low salt intake. This was true in the present experiment, for example, in which 2 of the 15 controls had final systolic pressures of 158 and 164 mm Hg, respectively. We do not ordinarily observe our animals after 12 to 15 months of age because such older animals develop murine pneumonia and serve as foci

for respiratory infections in the colony generally. During such a period of observation, however, the mild elevations described seem not to affect health. In any event, we have never observed anything approaching the incidence and severity of the disease displayed by these rats eating baby foods. It seems most probable that the effects were, in fact, due to the high NaCl content of the processed foods but we are now investigating NaCl as the single variable in baby foods prepared by ourselves.

Since the NaCl requirements of the normal infant are met by milk (5, 7, 10) the addition of salt to baby foods is irrelevant to metabolic needs. There is no evidence that the infant is born with a craving for salt. Salt is no longer a necessary preservative for these foods. The initial effect of a chronically high salt intake begun in infancy may be no more than to induce a salt appetite. There is considerable evidence that salt appetite is acquired and that it readily adapts to customary salt intakes however varied (7, 11). This appetite will be satisfied, or enhanced, when the child graduates to processed adult foods which, except for fruits, also generally contain high concentrations of added NaCl (2). Indeed, the addition of NaCl to processed foods is now so ubiquitous that it is difficult to obtain a diet low in salt even when required by prescription. An intake greatly in excess of normal metabolic requirements is thus virtually unavoidable in the western world.

If the primary effect of a chronic high salt intake is merely the induction of a salt appetite, a secondary effect may be the induction of hypertension. The fulminating hypertension displayed by our rats is rarely found before the third or fourth decades of human life but the strong genetic predisposition for hypertension must be equally rare in man. In genetically predisposed persons, our data suggest that a high salt intake, even if confined solely to infancy, might initiate hypertension either then or later. The possibility that evidence of injury from salt might remain covert for years is by no means unique. The appearance of thyroid cancer in adolescence from thymic radiation received in infancy; of blood dyscrasias long after exposure to benzene or radiation; of Parkinson's disease decades after encephalitis; of cancer and emphysema after many years of cigarette smoking provide strong precedents for the possibility that manifestations of injury from salt could be similarly delayed.

As indicated earlier, it is most unlikely that the high salt intake will, in fact, be confined to infancy. It is far more likely that salt will be ingested continually throughout life in large amounts by eating processed foods, supplemented both in the kitchen by the cook and at table by the consumer. We have suggested elsewhere that hypertension may develop thereafter depending on the genetic predilection of the specific individual combined with the intensity and duration of the salt intake (9, 12).

Summary. Some processed baby foods were lethal to hypertension-prone rats. Among 25 rats from a genetically hypertension-prone strain fed solely on such baby foods, all developed significant hypertension (averaging 180–190 mm Hg in the last 3 months of observation), 12 died, and 2 others became seriously ill during the 8 months of study. In contrast, the 15 control rats maintained on a low sodium chow were all alive and their average pressure at 8 months was 141.4 mm Hg. Considerable evidence suggests that the difference in response of test and control groups was due to the high NaCl content added to the processed baby foods. This added NaCl is unnecessary for the health of infants. It may contribute to the later development of hypertension in genetically predisposed individuals.

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