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THE DEADLY ROLE OF SALT IN KIDNEY DISEASE

A VOYAGE TO THE HOUYHNHNMS

"I was at first at a great loss for salt, but custom soon reconciled the want of it; and I am confident that the frequent use of salt among us is an effect of luxury, and was first introduced only as a provocative to drink, except where it is necessary for preserving of flesh in long voyages, or in places remote from great markets. For we observe no animal to be fond of it but man. As to myself, when I left this country it was a great while before I could endure the taste of it in anything that I ate."—
Jonathan Swift: Gulliver's Travels, Part 4, chapter 2 (published in 1726).

A paper by Dr. Kempner appeared in the January, 1965, issue of The Journal of the American Medical Association, where he reported experiments with rats in which kidney disease had been produced by injecting under the skin of the animals a few drops of a chemical substance called aminonucleoside of puromycin. The experiment was made to find out what effects small amounts of sodium salt (table salt) had in kidney disease:

Twenty rats were not given any injection and, therefore, had no kidney disease (control group).
Forty rats received the injections and developed kidney disease.
All 60 rats were placed on a diet of nothing but plain rice and vitamins. Of these 60 rats, 10 "control" rats and 20 rats with kidney disease received as drinking fluid only water (Group I), whereas the other 10 "control" rats and the remaining 20 rats with kidney disease received as drinking fluid water to which the same concentration of salt (.4 gm. of sodium in 100 cc.) had been added as that present in commercial tomato juice (Group II).

In 2-3 weeks after the injections, the results were evident: Of the 20 rats in Group I on rice with salt-free water, 2 had died and 18 were alive. Of the 20 rats in Group II on rice with some salt in the drinking fluid, 16 had died and only 4 remained alive. All the "control" animals remained alive and it did not matter whether they got salt or no salt in their drinking fluid. However, of the rats with kidney disease eight times as many animals died when the fluid contained salt. In other words, when the animals have kidney disease (which can easily be produced experimentally), the

mortality rate when salt is present is 800% of that when no salt is present.

Similar experimental set-ups have been used by Dr. Kempner to test various foods. From the patient's practical point of view, the most interesting is probably a comparison between two types of rice which were sold by the same company. Both had a very low salt content; in 100 gm. of Type A there was only .03 gm. of sodium, but the Type B contained even less than .004 gm. The experimental set-up was similar to the one outlined above:

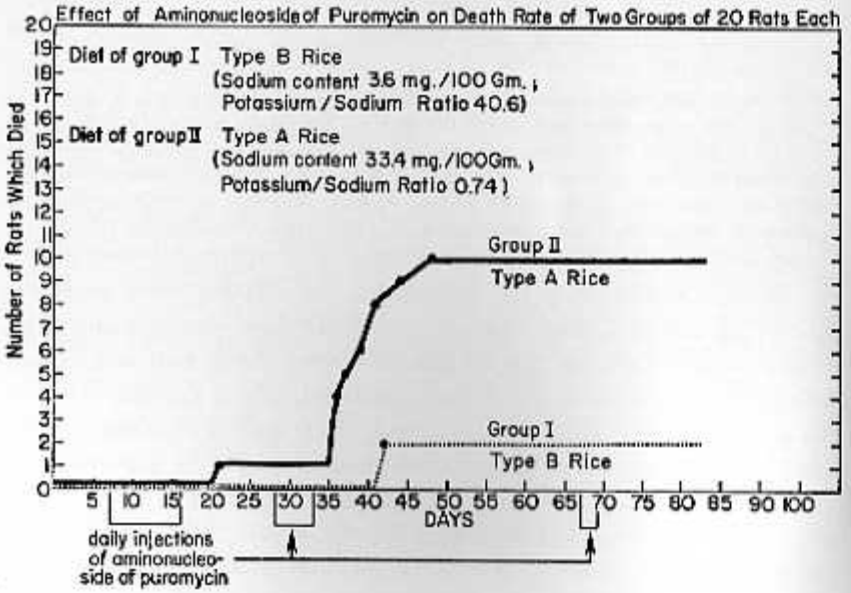
Twenty rats were not given any injection and, therefore, had no kidney disease (control group)

Forty rats were given the injections and developed kidney disease.

All 60 rats received plain water as drinking fluid and vitamins.

Of these 60 rats, 10 "control" rats and 20 rats with kidney disease were placed on a diet of Type B rice (Group I), whereas the other 10 "control" rats and the remaining 20 rats with kidney disease were given Type A rice (Group II).

In 3-4 weeks after the injections, the results were evident: Of the 20 rats in Group I, 2 had died and 18 were alive. Of the 20 rats in Group II, 10 had died and only 10 remained alive. All the "controls" remained alive and it did not matter what kind of rice they ate. However, of the rats with kidney disease, five times



as many died when they ate the rice which contained a very small amount of salt as when they ate the rice which contained almost no salt.

Only after having found out what deleterious effects even the smallest amounts of salt might have, can one realize what a big role minute differences in salt content may play in the struggle of the diseased kidney cells for survival.