not in equilibrium with the intracellular metabolite pool.

Free asparagine has been demonstrated in mammalian tissues (*5*) and plasma (*6*), and it appears to be a regular constituent of proteins (*7*). Ohno has recently shown (*8*) that lysozyme has 12 asparaginyl residues and only one aspartyl residue. Despite the wide occurrence of asparagine, little is known of its biosynthesis. Mardashev and Lestrovaya (*9*), on the basis of experiments with rat liver slices, proposed a transamidation between glutamine and aspartic acid yielding asparagine and glutamic acid, but there is as yet no unequivocal evidence for the proposed reaction. It should be emphasized that the results described in this report offer no clue to the mechanism of transfer of the amide group or to the nature of possible intermediates. They do, however, render it unlikely that asparagine is formed in this system by the direct amidation of aspartic acid by ammonia (*10*).

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Acquisition of Resistance to Osteolathyrism during Adaptation to Cold

"Crossed resistance" is a condition in which exposure to one agent induces resistance to another agent (*1*). Analysis has shown that, in most instances, this phenomenon is due to the fact that the stress of exposure to a noxious agent results in a discharge of glucocorticoids which, in their turn, inhibit responsiveness to other agents. It has been shown, for example, that through this mechanism, exposure to stressors (cold, muscular exercise, trauma, and infections) can inhibit the lung edema normally produced by adrenaline, the anaphylactoid reaction usually elicited by egg white or dextran, and many other types of inflammatory responses (*2*).

Since the thyroid also participates in certain systemic adaptive reactions, it seemed of interest to determine whether an increased endogenous secretion of thyroid hormone could likewise produce a phenomenon of "crossed resistance."

Experimental osteolathyrism is a disease characterized by excessive proliferation and degeneration of bone and junction-cartilage tissue (not to be confused with the clinical lathyrism, which affects the nervous tissue selectively). This skeletal disease, which is usually induced in the rat by treatment with *Lathyrus odoratus* or aminoacetonitrile (AAN), can readily be prevented by thyroxin (*3*). It is well known, furthermore, that exposure to cold augments the secretion of thyroid hormone. Could this stimulation of the thyroid during adaptation to a low temperature afford protection against intoxication with aminoacetonitrile?

Thirty female Sprague-Dawley rats with a mean initial body weight of 97 g (range 90 to 107 g) were subdivided into three equal groups. Group I was kept at room temperature throughout the experiment. Group II was kept at 0°C during treatment with aminoacetonitrile and group III was kept at 0°C for 10 days before and during aminoacetonitrile treatment. Aminoacetonitrile hydrosulfate was administered to all three groups, by stomach tube, at the daily dose level of 12 mg (6 mg in 0.2 ml of water twice daily). The experiment was terminated after 16 days of aminoacetonitrile treatment. At autopsy the skeleton was examined macroscopically, and one femur of each animal was fixed and simultaneously decalcified in Susa solution for the subsequent histologic examination of paraffin-imbedded sections stained with hematoxylin-eosin.

Mere macroscopic inspection of the bones sufficed to show that all the control animals (group I) had developed severe osteolathyrism, with multiple exostoses at tendon-insertion sites and excessive periosteal bone formation. Only traces of such changes were seen in group II, and none were seen in group III (Fig. 1). Histologic examination of the bones merely confirmed the macroscopic findings.

The histologic structure of the thyroid was essentially normal in group I, while in the two groups exposed to cold, the thyroid showed cellular hypertrophy and hyperplasia.

Since thyroidectomized rats do not withstand exposure to cold, it was impossible to verify the importance of the thyroid gland in the development of this kind of "crossed resistance" by control experiments on thyroidectomized animals. However, we know that small doses of thyroxin can inhibit osteolathyrism, and exposure to cold did, in fact, cause thyroid stimulation under our experimental conditions. Therefore, it appears justifiable to conclude that the resistance to osteolathyrism, which develops during adaptation to cold, is probably the result of an increased secretion of thyroid hormone.

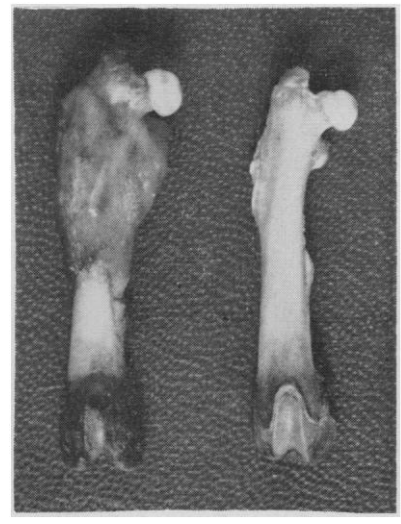

Fig. 1. Femur of AAN-treated rat kept at room temperature (group I) (left) and femur of a rat which had been kept in a refrigerated room during AAN administration (group II) (right). There is intense bone proliferation (especially in the upper two-thirds of the femur) and widening of the junction-cartilage line (in the distal extremity of the bone) under the influence of AAN at room temperature. These changes are inhibited in the rat that had been kept in the cold.

These experiments furnish us with still another example of an experimental disease whose development is decisively influenced by a hormone. They show, furthermore, that the amount and type of hormone normally secreted by the thyroid during adaptation to cold suffices to induce resistance against a severe experimental malady (*4*).

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Thorium Content of Stone Meteorites

The abundance and distribution of thorium in many terrestrial rocks and in meteorites have not been well defined, owing to the difficulty of detecting the small amounts of thorium involved, compounded with the problem of contamination by extraneous thorium at such low