RAYED ANIMALS

Rat. No.	Init. weight February 2	Fin. weight June 17	Xerophthalmia after
27 ♂	35 grams	72	128 days
28 ♂	29 grams	70	128 days
30 ♂	32 grams	80	.....
31 ♀	40 grams	88	.....
34 ♀	36 grams	80	140 days
36 ♀	59 grams	110	.....

It can be seen from the table that there was no difference as regard the rate of growth and incidence of xerophthalmia as the result of treatment with ultra violet light. We could not tell whether the earlier development of xerophthalmia in the males is of any significance or only purely accidental. A histological examination of the ribs of both series of rats was undertaken with the view to detecting rickets. The results were negative.

We are indebted to Dr. Hess for placing at our disposal the sources of light mentioned. We also thank the pathological department of the College of Physicians and Surgeons for help extended to us.

40 (2000)

The influence of light and darkness upon the development of xerophthalmia in the rat.

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The preventive influence of direct sunlight and of radiation with the mercury vapor quartz lamp upon the development of experimental rickets in rats has been demonstrated in experiments reported in previous studies. A logical further step was to determine whether or not direct sunlight and radiation with the mercury vapor quartz lamp would also prevent the development of xerophthalmia in rats fed diets which, under ordinary

conditions of roomlight, would lead to the development of both rickets and xerophthalmia and of xerophthalmia alone. If this information could be ascertained it would be a valuable contribution to the study of xerophthalmia and by analogy would suggest either the unity or the divisibility of the factors contained in cod liver oil, which prevent and cure both xerophthalmia and rickets. It was desired also to determine whether or not different combinations or groups of light rays—as for example, direct sunlight, quartz lamp radiations and roomlight—have the same or similar influence upon rats fed xerophthalmia producing diets. It was conceivable also that complete absence of light rays might have a different effect upon experimental animals than that produced by radiations showing either a complete solar spectrum (direct sunlight) or spectra considerably different from sunlight (quartz lamp radiation and roomlight).

On October 22, 1921, fifteen young albino rats were placed on a diet low in fat-soluble A and phosphorus (3127). Previous experience had shown that young rats on this diet would develop rickets and xerophthalmia. The animals were divided into three groups of five each. One group was to be kept in a laboratory room screened with ordinary window glass; a second group was to be kept in total darkness, excepting for the rays from a red electric light bulb such as is used in photographic dark rooms[1]; the third group was to be kept in ordinary roomlight, but was to be radiated with a mercury vapor quartz lamp. On November 15, 1921 another experiment was started using twenty-six young albino rats. These animals were placed on a diet low in fat-soluble A and poor in proteins of good quality (3392). Previous experiments had shown that this diet produces xerophthalmia in young rats. These animals were divided into groups and subjected to the same experimental conditions as detailed above. In these preliminary experiments it was found that all of the animals failed to grow, became emaciated and developed xerophthalmia. On the doubly deficient diet only the rats treated with the violet rays failed to develop rickets.

It was decided to repeat these preliminary experiments under conditions more favorable for study, and in addition to expose rats to the effect of direct sunlight. Accordingly, on April

[1] This bulb was lighted only when the animals were being fed or inspected.

1, 1922, twelve young rats were placed on the doubly deficient diet (3127); they were divided into four groups (each animal in a separate cage). The first three groups were subjected to the same conditions of light and darkness as heretofore described in the report of the preliminary experiments; the fourth group was exposed to direct sunlight. The daily period of exposure to sunlight averaged four hours and to ultra violet radiation thirty minutes. On April 20, 1922 a parallel experiment was begun, using eighteen young rats and the diet producing xerophthalmia only (3392).

Diet 3392 because of the inadequacy of its proteins both in quantity and quality produces xerophthalmia in rats more quickly than the doubly deficient diet 3127.

The results of these experiments and the conclusions which they warrant may be summarized as follows:

On both diets the rats in the roomlight, darkness and violet lamp groups developed xerophthalmia early, ceased to grow, became markedly emaciated and died. On the doubly deficient diet (3127) only the animals subjected to violet lamp radiation and sunlight exposure failed to develop rickets.

Radiation with the mercury vapor quartz lamp for thirty to sixty minutes daily does not prevent the development of xerophthalmia but promotes bodily vigor to a very limited degree.

The rats exposed to direct sunlight (with one exception) never developed xerophthalmia until the animals in the other groups had developed the disease and died. In some of these "sunlight" rats xerophthalmia developed late and the animals died; in others the disease was manifested late also but in recurring mild attacks; in still others it occurred in a mild form from which there was spontaneous recovery. Two of the rats never developed xerophthalmia at all; one of these animals and one in which there was spontaneous recovery were still living and free from xerophthalmia when the experiment was terminated, five months after it was begun. None of the animals had rickets.

In the "sunlight" animals which developed xerophthalmia and in all the rats in the other groups the disease was more severe in the animals on diet 3392.

The rats exposed to sunlight made an initial gain in weight which in the animals on the rickets-xerophthalmia producing diet

(3127) was maintained and in those on the diet producing xerophthalmia alone (3392) never entirely lost.

Exposure to direct sunlight protects rats from xerophthalmia to a limited degree, dependent in part at least on the extent of the dietary deficiency in fat-soluble A and in other factors, particularly proteins of good quality and upon the duration and constancy of the exposure. In some animals the disease develops regardless of sunlight; in others it is on the surface or just submerged; while in a few it never develops at all.

In this paper when the expression "exposure to direct sunlight" is used, it is inclusive of all that is ordinarily indissolubly associated with sunlight exposures. Sunlight itself, the effect of changes in the atmosphere by solar radiation, temperature, out-of-door air, these and other factors unknown to us acting singly or in combination may be responsible for the results usually attributed simply to "sunlight."[2] These experiments bring out above all else that exposure to direct sunlight and out-of-door air promotes in a very wonderful way the health, bodily vigor and longevity of animals which otherwise are unable to adapt themselves to markedly adverse environmental conditions.

Sunlight probably exerts no specific anti-xerophthalmic influence but acts by raising the level of the cellular activity of the organism to a point where the progress of the disease is held in check or allowed to advance very slowly and with relatively little disturbance.

Previous experiments have shown that sunlight contains an equivalent of the unknown factor in cod liver oil which promotes the normal formation of bone and in that sense may be spoken of as anti-rachitic. The experiments herein reported indicate that sunlight does not contain at all or only to a very slight degree the equivalent of the anti-xerophthalmic factor in cod liver oil—fat-soluble A.

By analogy, therefore, these experiments suggest that there are in cod liver oil two distinct factors—one preventive and curative of rickets, and the other preventive and curative of xerophthalmia. Sunlight can compensate for the absence of the one but not completely of the other.

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[2] Experiments are now in progress which seem to indicate that out-of-door air does have a favorable influence upon rats fed a rickets-producing diet.