Light Is Essential for Bone Deposition. Sunlight Prevents Rickets in Rats. The Work of A. M. Pappenheimer

Experimental Rickets in Rats. III. The Prevention of Rickets in Rats by Exposure to Sunlight


A. M. Pappenheimer was a distinguished pathologist at Columbia University College of Physicians and Surgeons. As described in the introduction to this Journal of Biological Chemistry (JBC) Classic, it had been shown that exposure of infants to sunlight eliminated the symptoms of rickets. Rickets was a very common disease in the 17th century, particularly in countries like England where the climate often provided several months without sunlight. Rickets is characterized by deficiencies in calcification of bone and cartilage in children, which results in abnormalities of skeletal development. The adult form of the disease is called osteomalacia.

Earlier, Pappenheimer had discovered that a diet high in calcium and low in phosphorus could cause rickets in rats (1). The study reprinted here exploits this animal model for a controlled examination of the effects of diet and sunlight on bone development in rats. It was shown that rats fed exclusively a diet of flour, calcium lactate, sodium chloride, and ferric citrate developed bone lesions characteristic of rickets in human infants. However, if the rats were exposed to sunlight for 15 or 30 min each day, there were no symptoms of rickets like those in the control rats housed entirely in the dark. It was also shown that supplementation of the diet with potassium phosphate could, in part, overcome the effects of the “richitic” diet. This study illustrated the necessity to treat light as an experimental variable. As the authors point out, “It seems probable that some of the irregularities and lack of conformity observed by investigators in this field may be attributed to keeping the experimental animals under dissimilar intensities of light.”

It is now also known that the effect of sunlight, UV light specifically, is to catalyze the non-enzymatic conversion of 7-dehydrocholesterol, an intermediate in cholesterol biosynthesis, to “provitamin D₃” which spontaneously isomerizes to vitamin D₃, cholecalciferol. Cholecalciferol is subsequently hydroxylated to form calcitriol, the active form of the hormone. So, vitamin D is not really a vitamin at all, because there is no dietary requirement as long exposure to sunlight is sufficient to initiate the conversion of 7-dehydrocholesterol to vitamin D₃. The active form of the hormone regulates transcription with products necessary for calcium absorption by intestinal cells and calcium uptake by bone-forming osteoclasts.

Because of the seriousness of rickets and because of seasonally inadequate sunlight in many geographical regions, most individuals obtain vitamin D from food or supplements like the children’s favorite, cod liver oil. Today, most vitamin D in the Western diet comes from fortified foods such as milk.

In experiments that paralleled those of Pappenheimer and his colleagues, E. V. McCollum discovered vitamin D (2) as reported in the paper featured in a previous installment of JBC Classics (3).
The biochemical relationships between vitamin D, the effects of sunlight, and bone metabolism were not made until many years later. It was shown in a series of papers that sunlight caused the conversion of 7-dehydrocholesterol to provitamin D$_3$ (4–8).

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REFERENCES

3. JBC Classics: McCollum, E. V. et al. (1917) J. Biol. Chem. 31, 229–253; (1922) J. Biol. Chem. 53, 293–312; (1925) J. Biol. Chem. 63, 553–562 (http://www.jbc.org/cgi/content/full/277/19/e8)

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1 Hector F. Deluca, Professor and Chairman of the Biochemistry Dept. at the University of Wisconsin, kindly provided the references for the UV-catalyzed conversion of 7-dehydrocholesterol to vitamin D$_3$. 