Vitamin D is essential for the mineralization of bones in both animals and humans, through its actions on bone, intestine, and kidney by promoting calcium homeostasis (1). Vitamin D is formed in skin exposed to solar irradiation, but alternative forms of the vitamin can also be obtained from food (2). Skin and dietary precursors are activated through specific metabolic reactions in the liver and kidneys (3).

The efficiency of vitamin D synthesis in the skin depends on the intensity of ultraviolet light from solar irradiation and is affected by several factors; latitude and seasonality being the most important (1). The efficiency of synthesis increases with shorter distance to the equator. As the angle of the incident sunlight becomes smaller at higher latitudes or in winter, the efficiency of vitamin D synthesis decreases. This decrease is particularly pronounced in high latitude regions (1, 5).

DEFICIENCY

Low solar irradiation impairing skin synthesis, low availability of dietary precursors and/or disorders in the metabolic pathways can cause vitamin D deficiency (5, 6). This deficiency produces abnormal mineralization in juveniles (rickets) and adults (osteomalacia) and is typically evidenced in juveniles by a thickening of endocortical junctions, bowing of large bones and swelling of joints. Juveniles and adults may also have softened bones that are more susceptible to fractures (5, 6).

Descriptions of vitamin D deficiency in domestic herbivores have been typically related to extreme latitudes with high availability of green grasses. Examples are New Zealand, South Australia, and the United Kingdom (4, 6). In humans, the problem is also common in high latitude regions (1, 5).

It is apparent from these observations that many of the factors that regulate the supply of vitamin D are strongly influenced by climate and can be affected by changes that may occur in relation to global warming. There is a high degree of uncertainty in predictions, particularly in relation to the secondary effects of temperature and atmospheric circulation patterns such as precipitation or soil moisture. Some consensus exists for a seasonal increase in rainfall at high latitudes (7, 8). What would be the consequences of these changes on vitamin D status in animal and human populations?

VITAMIN D AND PERIODONTAL DISEASE: AN ONGOING PROJECT AND HYPOTHETICAL EXAMPLE

This question arose when a skull of a huemul (Hippocamelus bisulcus), an endangered deer from the southern Andes, was sent in 1990 to our laboratory with strong periodontal lesions. A research project is now attempting to identify the prevalence and pathogenesis of periodontal disease in wild mammals. One of the main objectives is to evaluate whether vitamin D deficiency can, at least in part, be responsible for the disease and, if this is the case, to consider the potential climate-change related consequences. But, what relationships are there between vitamin D and periodontal problems?

Periodontal diseaseconstantly occurs in relation to global warming. There is a high degree of uncertainty in predictions, particularly in relation to the secondary effects of temperature and atmospheric circulation patterns such as precipitation or soil moisture. Some consensus exists for a seasonal increase in rainfall at high latitudes (7, 8). What would be the consequences of these changes on vitamin D status in animal and human populations?

References and notes

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dystrophy, a disorder present in soire phases of this deficiency (5, 6), causes severe softening of maxillary structure with dental loosen- ing and loss, in all domestic species (6). Thus, vitamin D deficiency could be playing a role in the pathogenesis of periodontal disease, not only because it can produce the predisposing lesions but also because both occur in similar geographical locations; regions at high latitudes with an abundance of green vegetation.

Clinical and pathological findings charac-
teristic of periodontal disease were recently described in sheep in southern Argentina, at about latitude 42°S (20).

A review of periodontal disease in wild herbivores shows that the majority of the descriptions were reported at high latitudes: reindeer (Rangifer tarandus) in South Georgia (latitude about 54°S) (21); caribou (Rangifer tarandus) in northwestern Alaska (22) and northern Canada (23); Dall’s sheep (Ovis dalli dalli) in northwestern Canada (23); chamois (Rupicapra rupicapra L.) (25) and feral goats (Capra hircus L.) in New Zealand (26); guanaco (Lama guanicoe) (Latitude 42°S and 55°S) (27, 28) and huemul (Hippocamelus bisulcus) (Latitude 43°S) (29) in southern Argentina.

Periodontal disease, mainly described in herbivores, has also been detected in other mammals such as pinnipeds. It was diagnosed in the Alcadians (latitude 54°N) in historic sea lions (Enypnaptias jubatus), and sea otters (Enydyra lutris) (30). A lep- noroad seal (Hydrurga leptonyx), from the South Atlantic Ocean, was found on the Argentinean coast in 1928 bearing an osteomyelitis of both tarsus articulations caused by a joint disease (31).

Although the studies of these wildlife dis-
cases were based mainly on bone analyses, the particular distribution in high latitude regions and the complex hormonal and mineral interactions that regulate bone homeostasis, suggest that vitamin D deficiency might have played an important role in the pathogenesis. A causal relationship with periodontal disease would determine that environmental changes affecting vitamin D could be responsible for the historical prevalence of the problem.

An interesting study of periodontal lesions in grey seals (Halichoerus grypus) (32) and harbor seals (Phoca vitulina) (33) from the Baltic Sea, indicated that the problem has been present at least since the 19th century. The authors detected an increased prevalence of skull lesions collected after 1960 in grey seals (32), and since the turn of the last century in harbor seals (33). Organochlorine pollutants, were suggested to contribute to the increased prevalence, because of their suspected effects in producing high levels of glucocorticoids (hyperadrenocorticism); compounds that can produce bone porosity (32, 33). Vitamin D deficiency may also have played a role in the increased incidence of lesions because of climate change, which could have occurred in the last decades, but also for the fact that glucocorticoids can diminish the number of vitamin D receptors in bone and intestinal mucosal cells (1). Even when fish—particularly the liver—have high vitamin D concentra-
tions, hepatic tissue can also accumulate high concentrations of calcium as in several

marine species such as oysters, crabs and mussels (34). Cadmium was another pollut-
detected in seals, with increasing concentra-
tions with age (35). Cadmium chronic toxicity is characterized by renal damage and osteomalacia in humans, as was described above after an episode of chronic ingestion of highly contaminated rice in Japan (34).

In addition, decreasing levels of vitamin D in seals have been observed in cadmium-induced renal damage; evidence that vitamin D could be seriously involved in the pathogenesis of cadmium-induced osteomalacia (36). Thus, it may also have played a role in the skull lesions of seals.

CONCLUSION

The causal relationship between vitamin D and periodontal disease in mammals is a hypothesis that needs to be investigated, and which poses some major questions. Different factors could be altering vitamin D availability: smog, through the decrease in solar irra-
thtage; air pollution, through atmospheric metabolic dysfunctions; and climate change through the reduction of sunshine hours and other indirect effects. Thus, these environ-
mental changes could be altering the epide-
miology of vitamin D deficiency.

References and Notes

1. Fraser, D.R. 1988. Calcium-regulating hormones: Vita-