Darkness, Water, Osteoporosis

By Raymond Peat

(Ray Peat's Newsletter)

Around 1973, there was an interesting observation, in which eyes that had been burned by ultraviolet rays were treated with red or infrared light, and the pain was relieved. Since I had been interested in solvated electrons, I wondered if this medical observation might involve a mechanism similar to the "bleaching" of trapped electrons in crystals or glass which has been colored by exposure to x-rays or gamma rays. Szent-Gyorgyi used to demonstrate that structured materials, e.g., ice, can stabilize excited states of electrons. He was curious about the existence of pigments inside organism, and suggested that they related to matabolic electrons, since he assumed that external light didn't reach these tissues.

In December, I drove in sunny weather all the way to Houston, and thought about light. Having read that the leaves of deciduous trees go into a negative protein balance (work by K. Thimann, U.C. Santa Cruz), as they age in the short days of autumn, I realized that the mechanisms of aging that I have discussed in other newsletters could be general for all living things. Knowing that plants have "photo-respiration," that is, the respiratory production of usable energy during stimulation by light, I wondered if animals might have something similar, accounting for winter sickness, dark-stress, and aging itself.

Knowing that photosynthesis uses orange-red light (though other plant processes are stimulated by other colors), I wondered if that frequency also stimulated photo-respiration. Anyone who has held their hand over a bright light knows that red light does penetrate tissue. Pineal researchers planted a photoelectric cell in a sheep's brain, near the pineal gland, to see whether significant amounts of light reached that organ directly, and the device clearly indicated when the animal moved from shade to sunlight. So, when I got home I was thinking about how to design an experiment to test the direct effect of red light on tissue respiration, especially in the brain.

I had been home for a couple of days, when a journal arrived in the mail, describing exactly the sort of experiment I needed to test the idea. Rats had a bright red laser beam shined on their heads for 15 minutes, and then the respiratory enzymes of the Krebs cycle was studied. The changes were consistent with enhanced respiration. (A. T. Pikulev, et al., Radiobiology 24(1):29-34, 1984.) (The red laser has been used for more than ten years to promote healing -- references will be provided on request.)

So my present thought is that darkness does in some way "poison" respiration, and that light enhances it, possibly by the indirect mechanism of disposing of electrons that have leaked out of the usable-energy pathway.

WHEN RESPIRATION IS blocked, tissue takes up water. A simple illustration of this is the cornea -- when contact lenses were very impermeable to oxygen, they caused severe corneal swelling. I think estrogen works by the same mechanism (and this explains why so many toxic substances are "estrogenic"). There was a doctrine that cells can move water only by moving sodium, so medical textbooks say that estrogen causes sodium retention; but it occurred to someone to actually measure the effects of estrogen on sodium and water, and they found that estrogen causes the fluids to become hypotonic, with a lower than normal sodium concentration.

Estrogen (like darkness and stress) promotes prolactin secretion. A previous newsletter discussed the experiments in which sodium suppressed prolactin secretion, while taking water without sodium stimulated it. So it seems likely that hypotonic (or sodium deficient) tissue fluids might be a common factor in the various situations which increase prolactin secretion.

In the salmon which swim up the Columbia river to spawn, the fresh water, which is very sodium deficient compared to the ocean, does stimulate the secretion of prolactin, and the prolactin is involved in their adaptation to the fresh water. The adaptation is very costly, though, because these fish undergo very rapid aging. Their bones are transformed in ways very similar to the bones of other animals in extreme old age: for example, the back humps and the jaw juts forward. (Since the fish vertebrae don't bear weight as those of a biped do, we shouldn't attribute the bending of the human spine in old age to "compression.")

Some recent studies have investigated prolactin's involvement in calcium disposition, and its contribution of osteoporosis. (J. A. Schlechte, et al., *J. Clin. Endocrinol. Metab.* 56:1120-1123, 1983; growth hormone is closely associated with prolactin, and I mentioned in Nutrition for Women that there was some basis for thinking that estrogen might make osteoporosis worse, acting through the growth hormone. The editor of the *Yearbook of Endocrinology* (1984, 273) says "Consider, if hyperprolactinemia leads to osteoporosis, and the administration of estrogens to postmenopausal women leads to hyperprolactinemia...")

Since it is established that excess prolactin does contribute to osteoporosis in humans, it seems reasonable to suppose that the dramatic increase in prolactin secretion when these animals enter fresh water would be the cause for the equally dramatic skeletal changes in the fish.

It is already known that excess calcium damages respiration and that calcium is associated with structural disruption of the mitochondria, and that tissues tend to calcify in aging. Inside cells, calcification begins in the mitochondria; outside cells, for example in blood vessel walls, the "ground substance" (a fine-textured component of connective tissue) can become calcified.

I think rational therapy for osteoporosis would include thyroid, Vitamin A, bright light, and either pregnenolone or a mixture of progesterone and DHEA, with a diet rich in protein and all the salts: calcium, magnesium, sodium, and potassium.

Hypotonic fluids are disruptive to life processes and structures. Even tissues which are not very cellular, such as cornea, joint cartilage, and the various "basement membranes" (which are rich in ground substance) can be damaged.

I suspect that we -- fish and humans -- have a simple way to protect ourselves from "hypotonicity," that is, from a deficiency of salts in our fluids. (There have been various observations in which one salt can substitute for another, at least to the extent of preventing an immediate disaster.) We carry with us a huge reservoir of minerals, our skeleton. In osmotic stress, it is better to lose bone material (and teeth) than to experience hypotonic disruption.

Undoubtedly the liberated calcium participates in adaptation in ways besides preventing swelling of the connective tissues, but I plan to examine the metabolic effects of calcium in future newsletters. (The bones also provide a large reservoir of sodium.)

Both prolactin and cortisone are secreted during stress (and in darkness), and both mobilize calcium. If calcium is so harmful to our energy producing system, why do the stress hormones dose us with it? I think this is largely explained by the generality of hypotonicity in stress, and by the convenience of the skeleton as a mineral reservoir. Selye illuminated estrogen by comparing it with the initial shock phase of the stress reaction (before adaptation begins), but the value of the comparison probably goes the other way, too: the essential element in stress is the inadequacy of energy to meet a challenge, and when energy is insufficient, water is taken up. (And some water is produced in the cell when oxygen is electronically reduced; this "respiration" is not necessarily in

Darkness, Water & Osteoporosis

Continued from page 130

volved in energy production — even "age pigment" can convert oxygen into water.)

Selye found that various stresses caused connective tissue disease, such as scleroderma and arthritis. He didn't explain the mechanism. I think it is significant that women are much more susceptible to many connective tissue diseases than are men. For example, a recent Arthritis Foundation advertisement says that women are twice as likely as men to suffer from arthritis. Fibrositis, myositis, etc., are common in women. (I think the studies of adhesions being prevented by progesterone provide a good model for understanding connective tissue disease, and allergies or "auto-immune" diseases; see Maurer and Bonaventura, *Fertility and Sterility 39* (4), p. 485, April 1983.) When cartilage is soaked in distilled water (or hypotonic body fluids) it swells with tremendous pressure. The microscopic mesh of this connective tissue is rich in negatively ionized groups, which attract neutralizing positive ions, such as potassium and sodium. As the neutralizing ions are leached away, the negative ions repel each other, causing swelling and attracting more water. As positively charged calcium ions are made available from the bones, the swelling is blocked. Continued for a long time, this seems likely to lead to conditions such as arteriosclerosis and scleroderma.

STRUCTURAL DISRUPTION of the mitochondria, as well as uncoupling phosphorylation from respiration, can be caused by cortisone. So it is interesting to see seasonal changes in the structure of the mitochondria, which are consistent with the ideas I have been talking about, that winter (and light deficiency in general) is what ages and kills us.

Winter stress, elevating cortisone, could also be the reason for the non-adaptive depression of thyroid function in the winter. Many people have noticed an association between stress and periodontal disease. Gum disease is common in hypothyroidism, and bone loss is an important part of the periodontal disease. This same endocrine pattern, and not "postmenopausal estrogen deficiency," is the most likely explanation for osteoporosis in aging women.

When I was working on my hamster thesis, about 1971, I noticed an article which reported that aging women's urine revealed an increased ration of estrogen to the 17-keto steroids. I think it was that article that started me thinking of aging generally in terms of a deficiency of anti-estrogens, including things such as DHEA.

DHEA has been studied in relation to osteoporosis. I. Hollo, et al., in *Acta. Med. Acad. Sci. Hung.* (Hungary) 35(1):53-59, 1978, reported that "Administration of norandrosterone decanoate or dehydroepiandrosterone to patients with menopausal osteoporosis resulted in normalization of the postload hypercalcemia. Calcium tolerance of menopausal patients without osteoporosis was not affected by dehydroepiandrosterone."

S. Brody, et al., in *Maturitas* (Netherlands) 4(2):113-122, 1982, found significant positive correlations "between basal levels of DHAS and the DHA response to ACTH respectively, and trabecular bone mineral content of the distal forearm."

There was a study of prolonged DHEA treatment on rat bones, which I haven't found: M. Boross, et al., *Aktuel. Gerontal* (Germany) 13(1):15-18, 1983.

I think rational therapy for osteoporosis would include thyroid, vitamin A, bright light, and either pregnenolone or a mixture of progesterone and DHEA, with a diet rich in protein and all the salts: calcium, magnesium, sodium, and potassium. A bias toward magnesium, rather than calcium, would protect against many conditions that occur in aging and stress.

Additional References

1. "Estrogen and Osteoporosis," *Nutrition for Women*, pp. 44-45.
2. Ahmad, P., et. al.: "Anti-inflammatory steroids, lysosomal stabilization and parachor," *Canad. J. Biochem* (Canada) 53(10):1047-1057, 1975.
3. Frolov, V.A.: "Seasonal structural and functional changes in the rabbit heart," *Bull. Exp. Biol., & Med.* 97(4):425-428, 1984.
4. Pasquali, R., et. al.: "Seasonal variations of total and free thyroid hormones in healthy men: a chronobiological study," *Acta Endocrinological* 107:42-48, 1984.

NOTE: A couple of other things to notice in regard to stress and season: in men, the sperm count, and in both sexes, the visibility of veins on the backs of hands when they are held at waist level.

Raymond Peat
1358 E. 19th
Eugene, OR 97403