Light, melatonin and aging

Russel J. Reiter

Department of Cellular and Structural Biology; University of Texas Health Science Center, San Antonio, TX, USA; E-mail: reiter@uthscsa.edu

The suppressive effect of light on the synthesis and secretion of melatonin from the pineal gland is well documented. The mechanisms of light suppression involve retinal photopigment activation, inhibition of the electrical activity of the biological clock (the suprachiasmatic nuclei or SCN) and reduced neural activity in the cephalic division of the peripheral sympathetic nervous system. This leads to the diminished release of norepinephrine from postganglionic neurons that end in the vicinity of the melatonin-producing cells in the pineal gland. As a consequence, the enzymes that mediate darkness-induced stimulation of melatonin production are shut down and the levels of melatonin in the blood are diminished. Imposition of light, when sufficiently intense and of the proper wavelength, during darkness even if of very short duration, suppresses the production of melatonin. Melatonin levels also are diminished in the elderly due to destruction of ?-adrenergic receptors on the pinealocyte membranes and a result of an attenuated neural message from the SCN as a consequence of destruction of the neurons in this nuclear group. One factor which significantly reduces the degenerative signs of aging in old animals, i.e., food restriction, also curtails the loss of endogenous melatonin production. A number of studies have suggested that the loss of melatonin may be consequential in the processes of aging or in the frequency/severity of age-related diseases. Thus, melatonin has been used, primarily in experimental animals but also in some human studies, to reduce the onset and progression of neuronal loss and the behavioral consequences of tissue destruction. Furthermore, the primary theory of aging implicates free radicals as a major cause of molecular damage in the aged. Antioxidants, such as melatonin, prevent free radical-mediated damage and preserve tissue integrity and function. Any factor that suppresses endogenous melatonin synthesis may be consequential in terms of the aging process. Thus, the loss of melatonin due to light exposure may contribute to free radical-induced mitochondrial damage, reduced ATP production, and organ deterioration. Indeed, supplementing mice during the latter half of their life with physiological levels of melatonin, maintains more optimal mitochondrial physiology and reduces oxidative damage, both of which could be significant in deferring processes of aging. Light exposure during the normal dark period inhibits melatonin production and the loss of this antioxidant may be consequential free radical-mediated, age-associated diseases.