



## Living Organisms Coupling to Electromagnetic Radiation Below Thermal Noise

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Ultralow frequency (ULF) and extremely low frequency (ELF) electromagnetic (EM) radiation is part of the natural environment. Prior to major earthquakes the local ULF and global ELF radiation field is often markedly perturbed. This has detrimental effects on living organisms. We are studying the mechanism of these effects on the biochemical, cellular and organismal levels.

The transfer of electrons along the Electron Transfer Chain (ETC) controls the universal reduction-oxidation reactions that are essential for fundamental biochemical processes in living cells. In order for these processes to work properly, the ETC has to maintain some form of synchronization, or coherence with all biochemical reactions in the living cells, including energy production, RNA transcription, and DNA replication. As a consequence of this synchronization, harmful chemical conflict between the reductive and the oxidative partial reactions can be minimized or avoided.

At the same time we note that the synchronization allows for a transfer of energy, coherent or interfering, via coupling to the natural ambient EM field. Extremely weak high frequency EM fields, well below the thermal noise level, tuned in frequency to the electron spins of certain steps in the ETC, have already been shown to cause aberrant cell growth and disorientation among plants and animals with respect to the magnetic and gravity vectors.

We investigate EM fields over a much wider frequency range, including ULF known to be generated deep in the Earth prior to major earthquakes locally, and ELF known to be fed by lightning discharges, traveling around the globe in the cavity formed between the Earth's surface and the ionosphere. This ULF/ELF radiation can control the timing of the biochemical redox cycle and thereby have a universal effect on physiology of organisms. The timing can even have a detrimental influence, via increased oxidative damage, on the DNA replication, which controls heredity.