*Response*: Bard points out the similarities between the light-emitting electrochemical cell (LEC) and the phenomenon known as electrochemiluminescence. We are, of course, aware of this important earlier work. Our understanding of the two approaches leads us to conclude that, although superficially similar, the mechanisms involved in electrochemiluminescence are, in fact, quite different from that involved in the LEC.

The electrochemiluminescent devices described and studied in detail by Bard and colleagues rely on transport of the oxidized or reduced light-emitting molecules (ions) themselves through the electrolyte between the electrodes, rather than transport of the electronic charge carriers between the electrodes. The oxidized and reduced species (ions) then react with each other (or the electrode) to form the original organic or metallo-organic species in an excited state, which may subsequently decay radiatively. Electrochemiluminescent displays have been described in which the electrochemiluminescent substance is dissolved in a solid electrolyte. Nevertheless, after generation of the oxidized and reduced species, the ions diffuse away from their respective electrodes and eventually meet somewhere between the two electrodes. Alternatively, electrochemiluminescent material can be fixed on one of the electrodes in an electrochemical cell and cyclically reduced and oxidized by an alternating potential. A direct current potential can be used only if the cell contains an additional species that serves to interact with the luminescent material in such a way as either to oxidize it at the same potential at which it is electrochemically reduced or to reduce it at the same potential at which it is electrochemically oxidized.

In the LEC, on the other hand, the oxidized and reduced macromolecules are immobile; they do not physically move from one electrode to the other. On the contrary, it is the electrons in the $\pi^*$-band and the holes in the $\pi$-band (that is, the electronic charge carriers) that move between the electrodes within the immobile semiconductor. When a voltage is applied between the contacts, the semiconductor is electrochemically reduced at the cathode to form an *n*-type region containing negatively charged carriers (electrons) and electrochemically oxidized at the anode to form a *p*-type region containing positively charged carriers (holes). Ions move only during the transient formation of the *p-n* junction; after reaching steady state under a fixed applied voltage, all ion transport stops. Moreover, ion transport is not directly involved in the light emission. Under the steady-state conditions with the voltage on and the *p-n* junction formed, electrons from the *n*-type region and holes from the *p*-type region combine in the compensated *p-n* junction to form neutral pairs that radiatively decay and give off light.

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## Women's Longevity

In his otherwise excellent News article (Women's health research blossoms, 11 Aug., p. 766), Charles Mann states that "For most of human history, men lived longer than women. That situation began to change a century ago, as modern medical practices came into use. By 1920, the average U.S. female life expectancy of 54.6 years had outstripped the male life expectancy of 53.6." The problem in assessing these statements is that there are no reliable mortality data for the world as a whole for

most of human history. Evidence to support the first statement is based on studies of skeletal remains (*1*). However, there are problems with interpreting paleodemographic data, including whether the skeletons reflect actual mortality by age and sex in the population being studied and whether mortality in the populations being studied reflects mortality more generally. To construct an accurate life table from skeletons would require all or a random sample of deaths to be found at the burial sites, either a stationary population so that the age distribution of deaths in the population is the same as that in the life table or a stable population with known rate of growth, and accurate estimation of age at death and determination of sex. Evidence against both the first and the second statements is provided by estimated mortality rates from more recent populations with very high mortality but with data whose reliability is far more certain. In Sweden, the country with the longest historical series of reliably recorded mortality data, expectation of life at birth ($e_0$) for females has exceeded that for males since the first period (1751–1790, when $e_0$ was 36.6 years for females and 33.7 years for males) that official estimates are available (*2*). Estimated life tables for other high-mortality populations also show a female advantage; in India [from the first period (1872–1881) for which estimates are available, when $e_0$ was 25.6 years for females and 23.7 years for males, through 1911–1921 (*3*)], among immigrants to Liberia [1820–1843, when $e_1$ was 24.6 years for females and 22.9 years for males who survived the calendar year of arrival (*4*)], and among the British peerage [from the first period (1550–1574) when estimates are available, when $e_0$ was 38.2 years for females and 37.8 years for males, through 1700–1724 (*5*)]. The statistics Mann gives for female and male life expectancy for the United States in 1920 are correct, but the differential favoring females also existed for each year from 1900 to 1919 (*6*). Although there are no official national estimates before 1900, when the national death registration area was established, estimated life expectancy for females has exceeded that for males in Massachusetts since the first year (1850) that estimates are available (*5*). In 1995, expectation of life at birth for females is estimated to exceed that for males in every country except Bangladesh, Bhutan, India, Nepal, and Pakistan (*7*). Discrimination against females in South Asia has long been recognized by demographers as the source of this anomaly.

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## MHC Class I Gene Expression

Harald Neumann *et al.* (Reports, 28 July, p. 549) elegantly demonstrate that major histocompatibility complex (MHC) class I genes are expressed by a portion of neurons cultured from the rat hippocampus. Cells expressing the MHC class I antigen did not exhibit spontaneous action potentials, while neurons that did not express the antigen had spontaneous action potentials. Treatment of electrically "silent" cells with gamma interferon increased expression of MHC.

We have previously demonstrated (*1*) that neurons of murine trisomy 16 (mts16) fetuses (16 to 18 days after conception) in vivo expressed large amounts of class I MHC H-2Kk antigen and increased synthesis of messenger RNA that binds a 33-base antisense complementary DNA probe to a region in exon 2 of the H-2Kk sequence. The reactive neurons were from the trigeminal ganglion, thalamus, and cerebellum. This finding is related to the report by Neumann *et al.* because mts16 animals have an increased gene dosage for interferon alpha and beta receptors (*2*); both interferons increase expression of class I MHC antigens, and an increased gene dosage for the receptor may cause cells to respond in a manner similar to that observed when high doses of interferons are administered.

An implication of the findings of Neumann *et al.* is that expression of class I MHC molecules occurs in functionally impaired neurons. The cerebellum is one of the more developmentally disturbed regions in mts16 brain (*1*). The neural dysgenesis seen in mts16 conceptuses may be a consequence of high numbers of interferon receptors on these cells and the resulting increase in MHC class I expression. Alternatively, it may result from other factors.

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