Skin Cancer Linked to Office Fluorescent Lights

The doubling in the past 30 years of the incidence of melanoma, a particularly vicious form of skin cancer, may be due to fluorescent lighting in the workplace, researchers at the London School of Hygiene and Tropical Medicine write in the Aug. 7 LANCET. In what they call "the first report of an association between melanoma and exposure to fluorescent light," Valerie Beral and her colleagues describe a doubling in melanoma risk in people who work under fluorescent lights.

The discovery "could be very important," says Frank Rauscher, senior vice president for research at the American Cancer Society and a past director of the National Cancer Institute. "We need to have it confirmed, certainly.

"The data look as though they have been well-handled and analyzed," he says. "There is nothing out of the ordinary, except the finding itself. We all sit under these lights, as I'm sitting under them right now."

The study was done in Australia, where the incidence of melanoma has risen dramatically in recent years. The researchers queried 274 female melanoma patients and 549 matched controls about certain lifestyle factors that might increase their risk of melanoma. Answers from the two groups were analyzed to see whether there were any statistically significant differences.

Fluorescent lighting in the office was associated with a two- to-two-and-a-half-fold increase in melanoma risk, and the risk increased with duration of exposure. The findings could not be explained by histories of sunlight exposure, skin color, hair color, or any other factor, Beral says. The investigators then reviewed data collected previously on 27 male melanoma patients and 35 controls and found an even stronger link between occupational exposure to fluorescent lights and melanoma risk—a 4.4-fold increase for exposure of 10 years or more.

The findings are certainly "intriguing," says Warwick Morison, a determatologist and photobiologist with the Frederick (Md.) Cancer Research Center. "The statistical analysis is good; it points toward an association."

Yet a vital, unanswered question is how fluorescent lighting could cause melanoma. "I have a lot of difficulty at the moment believing it is due to [ultraviolet] radiation coming out of the fluorescent light," Morison says. "There isn't enough radiation out there.... Might some chemicals be activated by fluorescent lighting? ... I suppose we're at a stage where it should be fluorescent lights plus something else. Now we've got to look for the 'something else.'"

Beral and her team, on the other hand, suspect that a qualitative difference between the ultraviolet emissions of sunlight and fluorescent light may be responsible. "Solar radiation produces a smooth spectrum of emissions with a sharp cut-off of wavelengths below 297 nanometers," they explain, "whereas fluorescent lights emit a jagged spectrum with peaks at 298, 302 and 313 nanometers...."

Another possibility that needs to be considered, they say, is that longer wavelength UV radiation (315-400 nanometers), which is generally present in fluorescent lights in large amounts, may be carcinogenic. These wavelengths have been found to cause cancer if used together with chemical photosensitizers. "It is not possible, however," they admit, "even to speculate about the likely quantity of ultraviolet emissions from fluorescent lamps to which the people in our survey were exposed, since it is so strongly determined by the type of lamp, the presence or absence of a plastic cover, and the distance from the lamp."

Yet another unanswered question about their research is why fluorescent lights in the home were not found to increase the risk of melanoma. It may be because they, unlike fluorescent lights in the office, are not left on for long periods of time and are often not the sole source of illumination, Beral and her team speculate. Clearly the question needs to be investigated further, they say. —*J. A. Treichel*

Gene cloned from human X-chromosome

Abnormalities of a single gene can have devastating effects. In Lesch-Nyhan syndrome, children suffer from compulsive self-mutilation, severe mental retardation and cerebral palsy. The syndrome results from lack of one enzyme, the product of one gene, important in synthesis of nucleic acids. Forms of another disease, gouty arthritis, are due to a partial deficiency of this enzyme, called hypoxanthine guanine phosphoribosyltransferase (HPRT).

The human gene for HPRT has now been isolated from the X-chromosome and reproduced in bacteria, report scientists at the University of California at San Diego. This gene is expected to be useful in determining what is wrong with the genes of Lesch-Nyhan and gouty arthritis patients. A more distant prospect is that copies of the normal gene might be transplanted into patients with Lesch-Nyhan syndrome to provide them with the enzyme they lack.

"The HPRT gene also offers the first possibility for probing how genes affect neurological function and behavior," says Theodore Friedmann, one author of the report in the August PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES. "It's the only gene characterized so far which, when it goes amiss, leads to retardation and behavioral changes."

The cloned gene will also be useful in studying the organization of the human X-chromosome. The HPRT gene is one of the first two identified X-chromosome genes to be isolated and cloned. (The other is the glucose-6-phosphate dehydrogenase gene, which is deficient in hemolytic anemia.) Because the HPRT gene is on the X-chromosome, Lesch-Nyhan syndrome is passed by female carriers to half their sons. The syndrome occurs approximately once in every 100,000 births. Friedmann and co-workers say, "The availability of a defined and authentic probe for a human X-chromosomal marker such as HPRT may make it possible to understand some important features of this region of the X-chromosome."

The work by Friedmann, Douglas J. Jolly, Abby C. Esty and H. Uli Bernard used an approach that may be useful in isolating other human genes. The scientists put bits of human DNA from placental cells into mouse cells deficient in the HPRT enzyme. They then selected the cells that had incorporated functioning human HPRT genes. To separate the human DNA from the mouse genetic material surrounding it, the researchers used a fragment of DNA that appears regularly in human genetic material. The group assumed that the human HPRT gene would be located near one of these characteristic "Alu repeat sequences." The DNA from the mouse cells was cut up and the pieces separated on a sheet of gel. Radioactively labeled Alu sequences bound to the human sequences, but not to the mouse material. The final product was a fragment of the human HPRT gene. This fragment was then used to select the full length HPRT gene from a library of large pieces of human DNA that had been prepared by Stanford University scientists. To demonstrate that they had the correct and functional gene, the large piece of human DNA was copied in bacteria and inserted into mouse cells lacking HPRT. The cells successfully produced the human form of the enzyme. "These studies demonstrate that it is possible to clone fragments of very large genes that are expressed at low levels ...," the scientists conclude. They are now working to determine the structure of the HPRT gene.

—*J. A. Miller*