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Fluorescence micrographs of a living motor nerve terminal (20 micrometers long) of a frog. The nerve was first stimulated vigorously to label all synaptic vesicles with a dye (top). Each spot is a cluster of vesicles. The nerve was then stimulated briefly to relabel a fraction of the vesicles with a different dye (middle). The uniform yellow color of these two images when superimposed (bottom) shows that the newly recycled vesicles were distributed randomly within the total vesicle pool. See page 200. [Digital film recording by G. W. Hannaway & Associates]
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Diet and Cancer in Humans and Rodents

The impact of diet on incidence of cancers in humans and rodents is well known to some scientists and physicians. It is slowly being comprehended by the public. The knowledge has been ignored by Congress and the Environmental Protection Agency.

We now know that cigarette smoking, alcohol, and ordinary foods together are associated with almost all of excess cancer. The contribution from foods is about as great as that from smoking. Two examples of causative agents in foods are excessive amounts of fats and salt. Western populations that derive 40 to 45 percent of their food calories from fats experience comparatively high mortality from cancers of the postmenopausal breast, distal colon, ovary, endometrium, pancreas, and prostate. Those Japanese who obtain only about 15 percent of their calories from fat have a dramatically lower incidence of these forms of cancer.

In rodents, substantial effects of diet on longevity and occurrence of cancer have long been known. Inbred strains of animals fed ad libitum (ad lib), that is, food always available, were found to be not nearly as long-lived nor free of cancer as animals having a calorie-restricted (CR) diet. The longevity of CR rodents is variable depending on strain but usually is of the order of 120 to 150 percent that of ad lib animals. The rats and mice fed a restricted diet are smaller, healthier, and sleeker than the obese, sluggish counterparts fed ad lib. Occurrence of tumors is variable with species, strain, and site. However, many studies have shown that incidence of tumors in ad lib animals usually exceeds that of CR rodents by factors of 3 and more. In effect, excessive fat or calories in the food intake are promoters of cancers. According to usual practice, in which ad lib–fed inbred rodents are provided food that includes the maximum tolerated amounts of test substances, ordinary foods should be categorized as carcinogens. Furthermore, when other potential carcinogens are tested using ad lib–fed animals, the results of experiments are clouded by the fact that two kinds of carcinogenic have been administered. A more scientifically sound procedure would be to use CR feeding.

Benefits from a restricted diet are not confined to longevity and relative freedom from spontaneous tumors. Rodents on a CR diet experience fewer tumors when exposed to substances that promote cancer in the ad lib animals. Examples include substantially reduced effects of methylazoxymethanol, diethylnitrosamine, benzo[a]pyrene, methylcholanthrene, and dimethylbenz[a]anthracene. It has been conventional practice to test potential carcinogens using highly inbred strains of rodents. The rationale was the supposed superior reproducibility of results compared with those obtained from wild-type animals. However, that assumption can be questioned. At least three examples of genetic drift of inbred strains can be cited. Rao has recently said that male Sprague-Dawley rats that once typically weighed 700 grams now weigh 1000 grams and that typical male Fischer 344 rats have had a 25% increase in weight. The higher body weights are associated with increased incidence of tumors and a substantial decrease in survival at 24 months. Earlier, reported on genetic drift in an inbred strain of mice. In the course of 10 years, mean body weight had increased. Many of the animals were obese. Lifetime expectation of developing one or another form of neoplasm had risen from 10 to 80 percent.

The use of inbred strains as test animals can further be questioned on the basis that they often develop spontaneous tumors in organs where cancers are not frequent in humans. For example, incidences of mouse liver tumors in 2-year-old B6C3F1 males has ranged from 17.8 to 46.9 percent. In contrast, death rate from liver cancer in the United States is about 0.005 percent.

Results of the animal studies raise questions about the validity of federal regulations that are based on ad lib–fed inbred strains of rodents. Are humans to be regarded as behaving biochemically like huge, obese, inbred cancer-prone rodents? Sooner or later Congress must recognize a new flood of scientific information that renders suspect the Delaney clause and procedures for determining carcinogenicity of substances.—PHILIP H. ABELSON

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ing patch clamping and molecular structures, coding of sensory information (split off from a chapter that formerly also discussed the somatic sensory system), visual perception, muscles, and the autonomic nervous system. In addition, new material (and in some cases new coauthors) have been added to many existing chapters; the distribution of topics has been rearranged and some chapters have been merged, especially in the sections on motor systems and development; some chapters (such as those on schizophrenia and affective disorders) have new authors and have been substantially reworked; and almost all chapters have been updated. The appendices have been shortened by dropping cerebral blood flow, physiological optics, and a problem set on mem- brain potencies, while retaining the old sections on electrical circuits, stroke, and cerebrospinal fluid.

Although the book was written for medical students, the editors list subsets of chapters that they believe would make the book suitable for graduate and undergraduate courses. For a graduate course, a minor weakness is that information in the text and figures is not linked explicitly to the reference lists, although one can often deduce sources. For an undergraduate neurobiology course, my choice of chapters would differ somewhat from the editors' suggestions. I would incorporate all six of the cell biology chapters (instead of just one) and the four chapters on vision (but not the somatosensory chapters, the reverse of their choice), and I would skip the chapters on myasthenia gravis and brain imaging (while otherwise following their choices for motor systems, development, and behavior).

But in spite of the editors' suggestions, I would be reluctant to use this book for an undergraduate course. Its depersonalized presentation of facts requires a prestructured, detailed curiosity that some medical students may bring with them but most undergraduates certainly lack. If they are in a course where the lectures carry most of the weight, undergraduates can use this book as a reference, but not as a book that will in itself inspire their learning. Aside from an occasional "as X and his colleagues showed," the people who created neuroscience are largely invisible, experimental details are relatively sparse, old controversies are presented as resolved, and a sense of thrilling and puzzling intellectual inquiry is absent. It is not a good way to learn about how new science is produced. For example, six pages on phototransduction describe in detail the steps linking rhodopsin to a cyclic GMP- gated sodium channel and the modulating role of calcium. The account is clearly and straightforwardly written, but it is presented as revealed truth. There is no hint that phototransduction was a major puzzle for many years, or that calcium was once suspected to be the intermediate messenger, or that rapid action by a cyclic nucleotide was a surprise. In fairness, the book’s homoge- nized approach is probably necessary for covering its vast territory, and comprehensiveness is definitely one of the book’s attractive features. From time to time, however, a glimmer of what might have been done comes through. The first two chapters, both by Kandel, provide examples of the best and the worst. The first chapter makes the general point that different parts of the brain are specialized for different functions by re- counting the historical clash of ideas about the localization of speech processing. It is vivid, concrete, and intellectually lively. The second chapter, in contrast, ploths through a synopsis of neural and glial cell types and the steps in neuronal signaling, all but inviting students to memorize definitions with min- ingal context. It would be a major advance if the next edition had more chapters like the first and fewer like the second.

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