ment of cultural content often has been little more than a laundry list of traits employed for easy identification of a group preparatory to discussion of its social behavior and organization. This tendency was identified earliest and best in 1969 in *Ethnic Groups and Boundaries*, a collection of essays edited by Fredrik Barth that focused on the boundaries between groups rather than on their internal aspects. Barth had been reacting to the notion that groups could be defined by their content alone. Unfortunately, he was interpreted by many as saying that one could ignore content altogether.

In reality, one cannot understand ethnic behavior without reference to both content and boundary, symbol and behavior. This volume attempts to wrestle with both dimensions. Keyes gives the mandate in his introductory essay by proposing a theoretical approach that considers both the cultural interpretation and the social manipulation of ethnic identity. The rationale for such an approach is summed up by the last essay, by Abner Cohen, in which he argues that ethnicity is always two-dimensional, both cultural and organizational. In between we have two papers that treat both dimensions explicitly. Judith Nagata incorporates both in her definition of ethnic identity. More important, she speaks cogently to the difficulty of determining the relationship between ethnicity and culture, of deciding when and why a cultural attribute is a primordial attribute. These concepts and distinctions are not simply hypothetical but have developed out of her on-going analysis of Malay identity. Bentley demonstrates the dialectical relationship between origin myths and high prestige. Furthermore, he shows us the complex relationship between such organizational aspects of ethnicity as political centralization and mercantile control and symbol manipulation, the cultural dimension.

In the remaining papers, the political and economic dimension is emphasized. Emphasis on this dimension is not surprising, for it is less difficult to study behavior than values and attitudes. Yet we do have ethnic groups for whom the most important identifications lie in the realm of ideology. Again, the value of a diachronic approach becomes clear. The alternation of periods in which such groups manifest themselves in organization and action with periods of little or no visibility gives the investigator material for the analysis of group development and response to environmental influences. Cohen, taking off from Weber, provides concepts that help in this kind of analysis. He contrasts two kinds of organization, communal and associative. The former is diffuse, all-pervasive, and nonutilitarian, whereas the latter is purposive, rationally organized, and efficient. At certain points, ethnic groups need the latter kind of organization to advance, but at other times relative invisibility is the best strategy.

The variety of approaches and ethnographic areas represented in this volume match well the complex, sometimes chimerical nature of the phenomenon of ethnicity. An additional virtue of the volume is that the papers derive their insights out of concrete cases. Many of the most perplexing questions about ethnic identity are perplexing only in the abstract. They tend to resolve themselves when confronted with empirical examples. *Ethnic Change* suggests some answers; more important, it poses new questions and provides, at the same time, models and methods with which to attack them.

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## The Neurobiology of Aging

**Brain Neurotransmitters and Receptors in Aging and Age-Related Disorders.** Papers from a symposium, Houston, Oct. 1980. S. J. ENNA, T. SAMORAJSKI, and BERNARD BEER, Eds. Raven, New York, 1981. xiv, 278 pp., illus. $32. Aging Series, vol. 17.

This volume resulting from a symposium is a good sampling of the subjects under study in a burgeoning branch of neurobiology. Particular emphasis is given to senile dementia (Alzheimer's disease). Until recently, age-correlated brain disorders were ascribed to neuronal loss, often through the use of B. D. Burns's 1958 estimate that humans lose 100,000 neurons a day. However, recent studies of humans and rodents show that many types of neurons are not decreased during aging. Major (more than 50 percent) neuronal loss exclusive of neurologic diseases may be exceptional and in most cases has unknown functional impact (discussed by Brizzee *et al.*, Coleman and Goldman, Diamond and Connor, Scheibel, and Rogers *et al.*, among others). Disease-related neuronal loss is exemplified in Parkinsonism, a disease of late mid-life, in which greater than 90 percent of the substantia nigra dopaminergic cells disappear by the time of death. The greater than 90 percent loss of cortical choline acetyltransferase activity in senile dementia also implies a major neuronal loss, but the localization and extent of cell loss is unclear. An incisive study by Coyle *et al.* shows cholinergic projections from the nucleus basalis to the cortex of the rodent, which may account for 70 percent of cholinergic activity in the frontoparietal cortex. Meanwhile, many are pursuing pharmacologic rectification of the putative cholinergic deficits in senile dementia, drawing from the precedent of treating the dopaminergic deficiencies of Parkinsonism with neurotransmitter precursors or receptor stimulants. Sadly, treatments with cholinergic precursors and agonists have not resulted in clear success in clinical trials, but some results are encouraging (discussed by Appel, R. C. Smith *et al.*, and Drachman). Pharmacologic models of cognitive dysfunctions are also described by Bartus and Dean and by Drachman. Transient memory impairments induced by the cholinergic blocker scopalamine in rats, primates, and humans resemble some aspects of aging and provide useful assays for potential therapies. The more precise biochemical and anatomic categorization of senile dementia will probably implicate other neurotransmitter changes to varying degrees: it would not be surprising for subtypes of senile dementia to be defined that have differing potentials for therapy. The responsiveness of the cortical dendrites of old rats to enriched environments suggests the plausibility of sociologic approaches to age- and disease-related neural deficits (Diamond and Connor).

The effects of age on neurotransmitter receptors include the experimentally robust 30 to 40 percent loss of striatal dopamine binding sites (Enna and Strong, Roth, Samorajski, and R. C. Smith *et al.*), a loss that may exceed the extent of neuronal loss in the striatum (Brizzee *et al.*). Because the dopamine agonist apomorphine induces smaller increases in the utilization of glucose by the nigro-striatal system in old than in young rats (C. B. Smith), dopamine receptor-limited functions may also change with age. However, the metabolism of cyclic nucleotides and neurotransmitter-related functions indicates a "remarkable stability during . . . aging," with major impairments detected only in cerebellum (Schmidt). Dramatic anatomic and electrophysiologic changes in the aging rat cerebellum are also described (Rogers *et al.*).

Hypothalamic age changes are dealt with, if briefly. The neuroendocrine hypothesis of aging that would link many

physiologic changes with hypothalamic alterations is illustrated in aging rodents by dampened pulsitile release of growth hormone (Meites) and by altered rhythms of hypothalamic serotonin levels in association with the loss of female cycles (Walker). Not included in the book are discussions of steroid-dependent age changes in the rodent hippocampus (via corticosterone) and hypothalamus (via estradiol).

In sum, the book illustrates for the general reader a dynamic subject that has recently left its phenomenologic past and entered a paradigmatic phase with rich experimental possibilities.

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