aggregation for most tumors. However, for breast cancer, I object to his multiplying of the average lifetime probability of female breast cancer (7%) by the relative-risk estimates (published by D. E. Anderson) for relatives in breast cancer families; for example, 7% × 5 = 35% probability for a woman whose mother and sister had premenopausal, bilateral tumors. I prefer a summary figure that recognizes the age-dependency of both breast cancer incidence and relative-risk estimates; for example, the annual probability for women between ages 35 and 39 with the same family history is 2% (52.5/100,000 per year × 39.5). (In fact, this estimate is too high because the age-specific incidence should omit women with the positive family history.) Such an annual probability needs recalculation every five years because the background incidence increases rapidly with age and the relative risk from family factor decreases.

Technical editing is poor: typographical mistakes abound, use of the possessive case for syndromes seems random, and the index is inaccurate and incomplete—"twins," "cancer family syndrome," "neural crest," "Knudson," "hit," and "mutation" are not in the index but appear in the text. Moreover, this book barely mentions Knudson's two-hit mutational theory of carcinogenesis and cancer cytogenetics, two unifying themes of the field.

In sum, sparkling with updated information on published reports and numerous personal observations, this volume is useful for those already acquainted with this field. Still better served are geneticists, oncologists, and students who will find it a brisk, authoritative introduction to the clinical and counseling aspects of familial and Mendelian determinants of human cancer.

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Genetic Disorders Among Jewish People. By R. GOODMAN. Baltimore: The Johns Hopkins University Press, 1979. Pp 493. $32.50.

Elisha was mocked by "little children" who "said unto him, go up, thou bald head . . ." (2 Kings 2:23). Deaf-mutism is described not only in the Bible (Psalms 38:14), but also in the Talmud. Gout may have affected King Asa (1 Kings 15:23 and 2 Chron. 16:12), and hemophilia A was recognized in the Talmud and discussed by the twelfth-century physician and Talmudist, Maimonides. Many other disorders of genetic interest were recorded in the Bible and Talmud.

Although knowledge of genetic diseases was gained over thousands of years of Jewish history, no book was devoted to these diseases in Jews until 1979. Then two excellent books on the subject made their appearances. Richard Goodman has written a remarkable monograph of almost 500 pages, which deals broadly and specifically with genetic diseases in Jews, and Dr. Goodman, together with Arno Motulsky, has edited a related volume [1] based on a symposium in New York sponsored by the National Foundation for Jewish Genetic Diseases.

The Jewish people can, like Gaul, be divided into three main parts: Oriental, Sephardic, and Ashkenazi. Historically, the oldest group is Oriental; the next, Sephardic; and the most youthful, Ashkenazi.

Oriental Jews: The Jews are historically a Middle Eastern people, Oriental Jews representing the original Jewish "gene pool." When the First Temple was destroyed in 586 B.C., some Jews remained in ancient Israel as the Palestinian branch of Oriental Jewry, while others moved east to Babylonia. Some stayed in Babylonia, now Iraq; others moved on, founding communities in Iran, India, Kurdistan, Afghanistan, Bukhara, and the Caucasus mountains.

Sephardic Jews: With the advent of the Greek and Roman empires, some Oriental Jews moved west. They came to the Iberian peninsula and were known, henceforth, as Sephardic

(Hebrew: Spanish) Jews. They grew in number and were persecuted, and in 1492, the year Columbus set sail for America, they were expelled by Ferdinand and Isabella from Spain. Sephardic communities developed around the Mediterranean (including in North Africa), in Western Europe, and in the Americas.

Ashkenazi Jews: A few Oriental Jews moved up into Europe under Roman rule, but the main migration to France and Germany was in the Middle Ages, and as many settled in Germany, they became known as Ashkenazi (Hebrew: German) Jews. Persecution prompted some to move eastward across Europe into the Baltic states, Poland, Russia, and other countries. The pogroms, beginning in 1881, led to migrations into Israel, England, and America. Of those who remained in continental Europe, more than half died in the Nazi holocaust.

Other communities: The Falashas, living in Ethiopia, are clearly distinct from the three main groups of Jews, and little is known about what genetic disorders they may have. The Karaites originated in Babylonia and Persia in the eighth century, and about 7,000 of them today live in Israel. They are known to have a high frequency of the gene for Werdig-Hoffmann disease, with one in 10 persons carrying the gene. The Samaritans, once about 300,000 strong, now number around 500. They are the world's smallest ethnic community, and have an increased frequency of chronic airway disease.

Numbers: The Falashas, Karaites, and Samaritans are few in number; most of the world's 14 million plus Jews belong to one of the three main groups: about 82% are Ashkenazi; 11%, Sephardic; and 7%, Oriental. In Israel, however, there is more balance: of the 3 million Jews living in Israel, 47% are Ashkenazi; 30%, Sephardic; and 23%, Oriental.

Knowledge of genetic diseases among Jewish people has grown rapidly, as the founding of modern Israel brought together sizeable numbers of Jews from the three major ethnic groups, and, hence, brought them under the study of perceptive medical geneticists.

Community-specific pattern: There are two large patterns of genetic disease in Jews. One pattern applies to Oriental and Sephardic Jews; the other, to Ashkenazi Jews.

Oriental and Sephardic Jews have, over the centuries, lived in distinct, fragmented communities, genetically isolated from other Jewish communities. Each Oriental or Sephardic community with a genetic disorder tends not to have its particular disorder shared with counterpart Jewish communities. For example, Moroccan Sephardic Jews have a remarkable frequency of ataxia-telangiectasia, its incidence at least 1 in 8,000, with a carrier frequency of 1 in 45. Libyan Sephardic Jews have a similarly spectacular frequency of cystinuria, likewise, an autosomal recessive disease. The incidence of cystinuria is about 1 in 2,500 with a carrier frequency of about 1 in 25. Phenylketonuria, once thought to be very rare in Jews, occurs in Oriental Jews from Yemen, where its frequency approaches that of it in non-Jews in Western Europe and the United States.

The main exceptions to this community-specific pattern of genetic diseases in Oriental and Sephardic Jews are glucose-6-phosphate dehydrogenase (G6PD) deficiency and familial Mediterranean fever (FMF). G6PD deficiency occurs in various communities of Oriental Jews. For example, it affects Oriental Jews in Iraq, Iran, and Kurdistan, and it, presumably, is due to selective advantage of heterozygotes vis-à-vis malaria. FMF is found in all Sephardic communities and in Oriental Jews from Iraq. FMF also occurs in high frequency among certain non-Jewish populations, such as among the Armenians in whom it is clinically different, suggesting that it may be genetically distinct from FMF in Jews.

Widespread pattern: A different pattern of genetic disease affects Ashkenazi Jews. This pattern includes the well-known "Jewish genetic diseases": Niemann-Pick, Tay-Sachs, spongy degeneration of the nervous system, Bloom syndrome, and familial dysautonomia. These diseases appear to have arisen geographically in the order given above along a north-south clime from the Baltic countries through east Poland/west Russia, down to north Hungary/Romania.

The explanation for this is still debatable: one can invoke one's favorite theory—chance, founder effect, selective factors, etc. No matter which theory proves most tenable, these diseases are widespread among Ashkenazi Jews, reflecting "mingling" and the absence of distinct ethnic subgroups in Ashkenazi Jews.

Ashkenazi Jews seem to have an unusual number of inborn errors of metabolism, which are inherited as autosomal recessive traits. Aside from those mentioned above, other such diseases include abetalipoproteinemia, essential pentosuria, and chronic adult noncerebral Gaucher disease. Of special interest, too, is a new form of mucolipidosis (type IV) in Ashkenazi children of eastern Polish origin; the gene for this may have arisen and been propagated in the same general corridor as the other autosomal recessive disorders: Niemann-Pick, Tay-Sachs, etc. Was this a "new mutation corridor"? The more we learn, the more puzzling it all becomes.

Practical precepts fall out of such population genetic studies. For example, in this past decade, it was found that Factor XI (PTA) is frequently deficient in the Ashkenazim: between 1 in 150 and 1 in 400 Ashkenazi Jews are homozygotes for PTA deficiency. Since the homozygote may not bleed spontaneously, but may bleed with trauma, it is essential to do a partial thromboplastin time in every Ashkenazi Jew having surgery. Since 95% of American Jews are Ashkenazi, this precept should apply to all Jews in America.

In the future, the genetics of complex disorders will surely be dissected with more precision. Among these common problems will be coronary heart disease, diabetes, and cancer. The study of the Jewish people may be helpful here, especially with the increasing frequency of cross-ethnic matings (Ashkenazi × Oriental, Oriental × Sephardic, etc.).

Everything learned about genetic disorders in Jews bears directly and indirectly on our overall insight into human genetic variation. In this sense, genetic knowledge of Jews is not parochial, but, rather, ecumenical in significance. The same applies to the genetic study of any group of people.

We are indebted to Richard Goodman for his monograph and to Goodman and Motulsky for their related volume.

How complete is our knowledge in 1980 of genetic diseases in Jews? The answer was given by the sixteenth-century French scientist and Catholic theologian, Blaise Pascal: "Knowledge is a sphere. The more it expands, the more it comes in contact with the unknown."

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Mutagen-Induced Chromosome Damage in Man. Edited by H. J. EVANS and D. C. LLOYD. New Haven, Conn., Yale University Press, 1978. Pp 355. $27.50.

This monograph is a collection of papers (mostly technical) presented at the *Second Symposium on Radiation Cytogenetics*, held at Edinburgh, Scotland, in 1977. The book contains 37 chapters, more than half dealing with various aspects of radiation damage or a combination of radiation and chemical mutagens.