



Geographic Origin of Jewish Migrants, Period of Immigration and Diversity of Acculturation: Is Israel a Cardiovascular Melting Pot?

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When examining chronic disease rates, frequent differences are found between diverse geographic areas, males and females, ethnic groups, and people living in different periods. This is particularly recognized in coronary heart disease [1]. Pathologic studies have shown major differences in the extent of atherosclerosis between different ethnic groups, whether separated geographically or co-residing in the same neighborhood [2]. Reasons for these differences have been sought. One of the better known inter-country and intercultural comparisons of differences in the prevalence and incidence of CHD – the Seven Countries Study organized by Ancel Keys and conducted over four decades ago [3] – implicated the variation in dietary habits as the major factor underlying such differences. While we are currently aware that a multiplicity of factors may be involved [1], these differences provide a fascinating topic for scientific investigation. Migrant studies are one way of examining the association of migration with changing rates of disease. One might think of comparing the incidence of CHD among migrants from several countries residing – sometimes also working – together, or comparing migrants from a certain country to those of another and to counterparts remaining in the homeland. The Ni-Hon-San study, comparing Japanese living in Japan, Hawaii and California, is a well-known example of the latter version [4].

Differences found in the above type of studies may reflect either the intervention of a new environment as a trigger causing an outburst of disease, the susceptibility to which was not expressed in the home country, or merely an effect of environmental and behavioral changes on the incidence of a disease that is primarily environmental in character. The susceptibility to the effects of migration may not be uniform. Some migrants arrive in a new environment whose lifestyle and attitudes are similar to those in their home country. Alternatively, migrants to a society that is either hostile or indifferent may experience major hardship in acculturation and in accommodating themselves to the new environment. Increased burden of disease is one expression of these changes [5].

Israel is a country built and developed by immigrants. Long

before the state was created some 50 years ago, differences between migrant groups were evident in the patterns of social and economic conditions, and by observation of the way they dressed, ate, danced and sang, spent their weekends, and preserved the religious habits of prior generations. Shortly after independence, Dreyfuss [6] spotted significant differences in the rates of hospitalization for coronary heart disease among Jews arriving from the various diasporas. Soon after, the changes in risk factors among Yemenite Jewish migrants arriving in Israel were noted for the first time [7]. Several years elapsed before Kallner and Groen [8] provided a more elaborate picture of the differences in mortality rate of the main chronic diseases among Jews from different geographic areas. The Israeli Ischemic Heart Disease study, initiated in 1963, included men who migrated between 1899 and 1962, but mostly in 1951. After a follow-up of 5 years, the incidence of myocardial infarction in 1963–68 differed significantly among the areas of origin [9]. Conversely, the length of time in the country showed little association with the same endpoint. When looked at later in 1986, in terms of 23 year CHD mortality, year of immigration related neither to CHD death nor to total mortality. One could thus conclude that whatever specific geographic-ethnic patterns related to CHD were “brought over” by the migrants, they tended to accompany them for a significant amount of time. This raised the first doubt with regard to the initially accepted paradigm of the host country (Israel) being a cardiovascular melting pot that would erase differences within a generation and produce common risks for the development of major chronic diseases.

The opportunity to investigate the issue in more depth arose as a result of the Israeli IHD study and also through linking information of individual countries of birth to census data in Israel, initiated by Peritz. Furthermore, Goldbourt [10], analyzing the 1970–74 nationwide mortality data in several countries of origin, clarified that many years after migration the rates of CHD mortality were still highly variable. This research focused primarily on migrants from Iran, Iraq, Morocco and Yemen. For example, the nationwide CHD mortality rates per 1,000 persons aged 25 and above were 13.5 and

CHD = coronary heart disease

IHD = ischemic heart disease

12.7 among male and female immigrants from Yemen, respectively. A surprising finding revealed that the more recent immigrants, namely those arriving in Israel in the "Magic Carpet" operation of 1949, exhibited rates of 18.5 and 18.1, respectively, resembling the rates among migrants from Iraq and Iran. The standardized mortality rates of the old-timers among Yemenite Jewry, calculated against the average of the Israeli Jewish population, were 0.33 and 0.36 for men and women – amazingly low. For the Magic Carpet Operation, Yemenite immigrants' SMRs were higher but still low, 0.52 and 0.48 [10].

If a cardiovascular melting pot was operative, why were the new immigrants at a higher risk of fatal CHD? Limited data on 383 Yemenite born male participants in the Israeli IHD study, begun in 1963, revealed that the early migrants had indeed approached "resident Israeli" levels of risk factors. For example, among 229 men emigrating from Yemen to Palestine before 1940, mean serum cholesterol was 201 mg/dl, mean systolic blood pressure 133 mmHg, and body mass index 2.37 kg/m², whereas among those migrating from 1940 and onwards the respective mean values were 182, 130 and 2.26. Yet, the more favorable risk profile among the latter immigrants was not associated with protection against CHD. Of note, the reported diet among the Yemenite immigrants of different migration periods was virtually identical, with 8–9% of total calories derived from saturated fat, 25–27% from total fat, 16% from protein and 57–59% from carbohydrates [10]. Food intake, then, appeared to be determined more along ethnic lines than by length of stay in the country. These results provided a hint that acculturation in Israel, in terms of risk factors, did not necessarily result in worsened prognosis for CHD even if risk factors for the latter appeared to follow a negative trend. In later years, a similar investigation of Ethiopian immigrants who arrived in "Operation Moses" in 1984 could only shed light on the initial profile and short-term changes in lipid and other risk factors [11]. The follow-up period is indeed too short for such a small group.

The riddle of apparently persistent Yemenite Jewish CHD rates has not been fully deciphered, as second-generation follow-up of risk factors and outcome have not been undertaken.

In the meantime, attention has shifted to the greatest wave of immigration to Israel, which occurred in the early 1990s and proceeds slowly to this day. I am referring to the immigration of Jews from the diverse republics of the collapsing USSR. Unlike previous "exotic" migrant groups, the emigrants from the previous USSR arrived in a society that had been greatly influenced, decades before, by early emigrants from the same lands. The Czarist Russian and subsequently USSR early emigrants to Palestine arose from an identical genetic background as well as a similar social, political and economic reality. Together with other immigrants from Eastern European Jewish communities, in particular Poland, they shaped the political form of regime and organization of the State of Israel to a significant extent. What could epidemiology teach us? The Israeli IHD Study permitted an analysis of risk factors and outcome of old immigrants from the previous USSR borders. It included 591 of

them, who subsequently showed somewhat elevated rates of CHD incidence between 1963 and 1968 [9]. Except for 42 men, they had all immigrated between 1920 and 1950. When long-term (23 years) CHD mortality was analyzed by country of birth, having migrated from the USSR was associated with a small non-significant excess CHD death risk of 8% as compared to other immigrants. Each year of stay increased the risk of fatal CHD within the group statistically by 2.7% (95% confidence interval, 0.5–4.8%, unpublished data). No such association was observed among emigrants from Poland, who come from identical stock and participated in the same study. Adjustment for conventional risk factors removed much of the alleged detrimental association between years in Israel and risk of CHD mortality, for which no biologically plausible reason can be found. However, this result certainly did not support the notion of an increased risk for the newer immigrants from the USSR at that period.

In the current issue of *IMAJ*, Brodov et al. [12] examine a much later wave of emigration from the USSR. While reporting on the mortality of 11,892 CHD patients who had emigrated from all corners of the globe to Israel, and comparing it to the rate among 1,850 Jewish patients born on the current territory of Israel, they further compare 228 new immigrants from the USSR who had lived in Israel for no longer than 6 years to 688 counterparts who had lived for 7–21 years and 359 veteran immigrants with at least a 21 year duration of living in Israel. In contrast to earlier work, they look at case fatality rates rather than first events. Thus, their study relates to the extent to which a cardiovascular melting pot might operate in Israel in terms of affecting the fate of new versus old immigrants who already had CHD. They focus on mortality after a follow-up period of 6.5 to 9 years (average 7.7 years). The two USSR migrant groups who had lived in Israel for over 21 years and for 7–20 years had similar mean ages. The relative newcomers were about 3–4 years younger. Rates of mortality were similar between the two latter groups (18.8 and 17.0%), whereas the veterans exhibited a much lower rate (12.0%). There were, however, important differences between these groups in terms of factors potentially affecting mortality. In particular was a heavy burden of New York Heart Association class 2 and above for the newest immigrant group (43.5% as compared to 28.1 and 31.1%), and the newcomers also showed excess of angina. This would suggest poorer care of these people in the USSR than would be the case for those who came earlier. Important information could be added had the analysis taken into account the year of disease onset, where it was treated and what treatment changes have occurred, but the data were not available.

Brodov's team proceeded from the initial comparison of crude mortality to adjust for the differences in disease severity, as well as in age, gender, and history of myocardial infarction, stroke, diabetes, peripheral vascular disease, chronic obstructive pulmonary disease, angina and cigarette smoking, applying the proportional hazard life table regression model by DR Cox. This model assumes proportional hazards, namely that the old migrant/new migrant ratio of CHD mortality risk remains constant over time. The reported result – an approximate mortality excess of 70% – cannot be adequately judged, since if the length of time in the country does

SMR = standardized mortality rate

affect the risk of mortality in these patients, then the assumption of a constant excess risk, while these immigrants had aged by almost 8 years spent in the country, might hamper the model and confound the estimation.

Brodov et al. speculate on the issue of adjustment to unfamiliar conditions in the new country as a possible cause for the poorer prognosis of the more recent immigrants. They consider the changes in climate and social environment as well as the need to study a new language as potential stressors. One of the possible explanations suggested by the authors – psychological stress experienced over an extended time in the USSR – is not compatible with previous experience in Israel of migrants from other areas (Arab countries of North Africa and the Middle-East), who shortly after migrating to Israel exhibited lower rates of CHD than did veteran migrants; nor is it compatible with the earlier USSR emigrants who participated in the Israeli IHD study. There are, however, two important differences. The early migration from the USSR had brought men and women into a society that was fighting for economic improvement and showing little or no aspects of influence. In addition, the current study by Brodov et al. refers to patients with CHD, and the effect of the various stressors may not necessarily act the same in promoting a first manifestation of CHD as it does in influencing the treatment and fate of those who are already ill.

With the limited data, mostly assessed at a single point in time – i.e., screening for the Bezafibrate Infarction Prevention trial between 1990 and 1992 – it is difficult to pinpoint a factor responsible for “importing the high CHD death rate” from the USSR; or why, if it did occur, would this increased risk disappear only after 20 years. Is there a lesson to be learned from these comparisons? Are there practical implications for policies of prevention or public health? Brodov and co-workers recommend further investigation into the role of possible risk factors, including psychological stress. If past experience in Israel is any guide, this is unlikely to materialize. The majority of the former Soviet Union immigrants are involved in a process of amalgamation into Israeli society, including individuals already making their way in the realms of academic studies, music, theater, teaching and coaching and...medicine, cardiology obviously included. Some of those arriving with putative qualifications for higher-ranking professional jobs have indeed been restricted to a disappointing working experience, but the younger generation is already on its way to, or at the forefront of, academia, elite sports and other avenues of excellence.

Eight years ago, Rennert [13] predicted an expected immediate increase of 6% in mortality among males and 4.4% in females in Israel, assuming the mortality patterns reported from the European Republics of the previous USSR. Reports from the Israel Bureau of Statistics have refuted this prediction and the life expectancy of Israeli males and females has continued to increase. Rennert's recommendation to “target areas requiring intervention in order to both protect the host country and facilitate assimilation” has not, to the best of our knowledge, been adopted. Whether the increased CHD fatality risk of recently arrived CHD patients is real or

confounded, it is most likely a passing phenomenon that will not leave a major mark on the public health agenda. Is there justification, from current research, to designate particular policies for primary or secondary prevention among immigrants, as the concluding sentence by Brodov et al. suggests? This question will probably remain moot. With all the concerns of a country with a continuously increasing life expectancy on the one hand and the necessity to combat both the increasing health costs and terror activities on the other, I find it difficult to envision such an origin-specific policy being undertaken by statewide or local health authorities, as the rapid process of amalgamation proceeds

References

1. Goldbourt U. Differences in frequency of atherosclerosis and coronary heart disease between populations and ethnic groups. In Goldbourt U, de Faire U, Berg K, eds. *Genetic Factors in Coronary Heart Disease*. Lancaster, UK: Kluwer Academic Publishers, 1994:3–20.
2. Tejada C, Strong J, Montenegro MR, Restrepo C, Solberg L. Distribution of coronary and aortic atherosclerosis by geographic location, race, and sex. The geographic pathology of atherosclerosis. *Lab Invest* 1968; 18:509–26.
3. Keys A, ed. Coronary heart disease in seven countries. American Heart Association Monograph 29. *Circulation* 1970;41/42(Suppl 1):186–95.
4. Kagan A, Harris BR, Winkelstein W Jr, et al. Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California: demographic, physical, dietary and biochemical characteristics. *J Chronic Dis* 1974;27:345–64.
5. Wessen F. The role of migrant studies in epidemiological research. In: Davis AM, Sacks M, eds. *Symposium on Cancer and Other Chronic Diseases in Migrants to Israel*. *Isr J Med Sci* 1971;7:1584–9.
6. Dreyfuss F. Incidence of myocardial infarction in various communities in Israel. *Am Heart J* 1953;45:749–55.
7. Toor M, Agmon J, Allalouf D. Changes in serum lipids, total cholesterol and lipid phosphorus in Jewish Yemenite immigrants after 20 years. *Bull Res Counc Isr* 1954;4:202.
8. Kalner G, Groen JJ. Mortality and hospitalization in relation to coronary and cerebral vascular disease in Israel. *J Atheroscler Res* 1966;6:419–29.
9. Medalie JH, Kahn HA, Neufeld HN, Riss E, Goldbourt U. Five-year myocardial infarction incidence. II. Association of single variables to age and birthplace. *J Chronic Dis* 1973;26:325–49.
10. Goldbourt U. Coronary risk factors and cardiovascular morbidity and mortality among Yemenite-born Israeli males, in comparison with other ethnic groups. A study in genetic versus environmental susceptibility. PhD Dissertation. Tel Aviv University, 1984.
11. Rubinstein A, Landau E, Goldbourt U, Reisin LH. Lipids and lipoproteins in new immigrant Ethiopian Jews in Israel. *Am J Epidemiol* 1988;128:153–64.
12. Brodov Y, Mandelzweig L, Boyko V, Behar S. Is immigration associated with an increase in risk factors and mortality among coronary artery disease patients? A cohort study of 13,742 patients. *IMAJ* 2002;4:326–30.
13. Rennert G. Implications of Russian immigration on mortality patterns in Israel. *Int J Epidemiol* 1994;23:751–6.

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