Jews and Inflammatory Bowel Diseases

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Ulcerative colitis is three to five times more common in Jews than in non-Jews living in Western countries [1-7]. Jews living in South Africa have an incidence of 17/100,000 compared with 5/100,000 in white populations [8]. In Israel, Ashkenazi Jews have a higher incidence than Sephardi Jews, but a lower incidence than Ashkenazi Jews in the United States or Northern Europe [9]. The prevalence rate rose from 121.0/100,000 in 1987 to 167.2/100,000 in 1997. These rates were higher in women than in men. Prevalence was highest in Israeli-born members in 1987 but in European/American-born members in 1997. The average annual incidence rate for the 10 year period was 5.04/100,000/yr [9].

Jews have higher incidence of Crohn’s disease than gentile populations [1, 4].

Within Israel, the prevalence rate has risen from 25.53/100,000 in 1987 to 65.11/100,000 in 1997, and then to 112.99 in 2007 [10]. The prevalence rate was higher in women than in men, and in Israeli- or European/American-born than in Asian/African-born individuals. The rise in prevalence was steady from 1987 to 1997 and then to 2007 in all subgroups, except for Asian/African-born kibbutz members. In the last group, a decrease in prevalence was evidenced between 1987 and 1997, and then a sharp increase between 1997 and 2007 [10].

The higher incidences and prevalence of inflammatory bowel diseases in Jews compared with other local populations have presented an unclear situation, which have led some people to consider Jewish ethnicity as a risk factor [11].

Earlier, I identified a lack of the provision of vegetables and fruits as a common phenomenon in English history since the Tudor times. This raises a hypothesis that a lack of antioxidants in the provided diets and the resultant overproduction of Reactive Oxygen Species (ROS) and the consequent development of genetic polymorphisms in previous generations and consequently their inheritance to current generations may have contributed to a higher incidence of degenerative diseases in Western countries compared with the East [12].

The explanation for this hypothesis is that free radicals are very reactive, combining with and altering almost any molecule with which they collide. If enough free radicals are formed, some are bound to collide with nucleic acid molecules and alter them. The result is a mutation [13]. The single electron reduction of molecular oxygen to superoxide initiates the formation of a family of ROS that create a constant and insidious threat to aerobic organisms. Homeostatic mechanisms have developed to minimize the steady-state levels of ROS and to repair oxidative damage. Of all the cellular targets that reactive oxygen can damage, DNA may be the most significant because of the potential mutagenic and genotoxic consequences.

The reactivity of ROS with DNA is well documented [14] and damage can occur in both the nitrogenous base and deoxyribose moieties [15]. Superoxide radicals and hydrogen peroxide do not react with DNA at physiological concentrations. However, the hydroxyl radical, produced from superoxide and hydrogen peroxide through transition-metal catalyzed Fenton chemistry and by ionizing radiation is indiscriminately reactive, breeding numerous radical adducts and their datives. Furthermore, the negative charge of DNA smoothes the progress of the local generation of hydroxyl radical through the binding of transition metals.

The overall consequence is that ROS can generate oxidatively modified bases as well single and double strand breaks in DNA. If un-repaired, such oxidative damage can lead to mutations that can range from single nucleotide substitutions to gross chromosomal rearrangements, which in metazoans can have somatic as well as germ-line consequences. In the oxygen-rich aerobic environment, the genomic integrity is maintained by minimizing the access of ROS to DNA and by biochemical mechanisms that recognize and repair oxidatively damaged DNA [14, 16].

In principle, increased mutation rates can arise through increased oxidative stress, by failure of ROS defence.
metabolism, or by failure of DNA repair. The interaction of modern dietary constituents’ intake and ancient genetic mutations may be contributing to the variation in the individual and regional predisposition to degenerative diseases. It is likely that ROS create a similar condition of genetic instability to the effects of irradiation which cross across the generations and have lead to the expression of the disease when appropriate cultures have been available [17]. The concept of inheritance of genetic aberrations has been verified in different studies [18, 19].

Zinc deficiency and overproduction of ROS in subjects with inflammatory bowel diseases are well known [20, 21]. Yet it is uncertain whether this lack of zinc is a cause or consequence of the activity of the disease. However, deficiency of zinc may facilitate the proposed interaction between modern dietary intakes and the inherited genetic polymorphisms. In addition, lack of the produced superoxide dismutases would result in genetic instability and consequently to more damage [22].

In a study aimed at analyzing the maternally inherited mitochondrial DNA in each of nine geographically separated Jewish groups, eight non-Jewish host populations, and an Israeli Arab/Palestinian population, and then comparing the differences found in Jews and non-Jews with data collected from the same populations in an earlier study using Y-chromosome samples [23], it was found that most Jewish communities were founded by relatively few women, that the founding process was independent of the geographical distinction to this, the paternally inherited Y chromosome showed diversity similar to that of neighbouring populations and showed no evidence of founder effects. It was also reported that the immigrants Jews had adopted their lives to those of the locals [24].

This life style of the Middle Eastern Jews who moved to Europe and to the States may give a clue to the possible reason of the high incidences and prevalence of inflammatory bowel diseases compared with whites and other Jewish subgroups.

We propose that environmental factors and the specific life style of the Ashkenazi may be a contributory factor to the occurrence of inflammatory bowel diseases, in contrast to their neighbours in Israel and West Bank.

Conflicts of interest

None to declare.

References