European **Rev**iew for **Med**ical and **Pharmacol**ogical **Sci**ences 2009; 13: 66-67

Letter to the Editor

A new hypothesis on inflammatory bowel diseases

Dear Sirs,

Crohn's disease (CD) and Ulcerative Colitis (UC) are recognised chronic inflammatory disorders of the intestine (IBD) caused by a combination of genetic and environmental factors. Three polymorphisms within the NOD2/CARD15 gene are highly associated with CD, increasing the risk from 2 up to 44-fold.

In a recent published article, Annese et al[1] revealed that they identified one risk allele in ninety eight healthy relatives of patients with IBD compared with two risk alleles in further eight relatives. Can we consider this information as an absolute prediction for the occurrence of inflammatory bowel disease in those identified healthy relatives? Can we screen for the development of the disease by examining the presence of those risk alleles in healthy relatives of patients with IBD?

These are just two examples of many other raised speculations as consequences of the reported data by Annese and his colleagues. Yet, the answer is obviously explained in de Vries and his co-workers' report. In this published article, de Vries et al demonstrated that they found variations in gene frequencies observed for C3, Gm, HLA-B, and GLO in the descendents of the Dutch immigrants to Surinam during the 19th Century and who survived epidemics of typhoid and yellow fever compared with the general Dutch populations[2].

This positively validates earlier published Sutton' s opinion. In 1903, Sutton explained that dominance and recessiveness are features of "chromatin entities" rather than morphological characters; they are thus functional properties of alleles, i.e. DNA variants at a particular gene locus, rather than allelomorphs[3].

In another way, dominance and recessiveness are properties of genetic information resulting in a certain function rather than the function itself.

With taking this note into account, this means that genetic maps mirror the influence of individual' s environmental factors and it is likely that the resulted alterations to be inherited throughout generations.

References

1) Annese V, Latiano A, Palmieri O, Lombardi G, Andriulli A; Italian Group of Inflammatory Bowel Disease. NOD2/CARD15 in healthy relatives of IBD patients. Eur Rev Med Pharmacol Sci 2006; 10: 33-36.
2) de Vries RR, Meera Khan P, Bernini LF, van Loghem E, van Rood JJ. Genetic control of survivals in Epidemics. J Immunogenet 1979; 6: 271-287.
3) Sutton WS. The chromosomes in heredity. Biol Bull 1903; 4: 231-251.

AM El-Tawil
Department of Surgery University Hospital
of Birmingham UK B15 2TH
E-mail: atawil20052003@yahoo.co.uk