CANCER REPORT 2012:

a PROPOSAL on the ATTACHMENT of WATER to GUANINE

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a Proposal on the Attachment of Water to a Guanine.

This year I have attempted to interest Government in my research by working through my local MP, John Hayes. As part of that process I have submitted research material from Taiwan, a study by Yang et al, "Chlorination of Drinking Water and Cancer Mortality in Taiwan," 1997, which shows the effects of chlorinated water supplies on cancers by site. The increased incidence of cancers by site which make up the cancer epidemic in New Zealand also follow the pattern found by Yang et al, that is, there is an increased incidence of cancers of the rectum, lung, badder and kidney with liver involvement in men. The New Zealand experience differs only in an additional involvement with the prostate.

The Yang et al study also casts doubt on the role of trihalomethanes but recognises that cancer sites affected are related to water consumption.

The epidemiology so far shows a clear correlation between water treatment, conductive pipes and cancer types by site. What it not clear is how the changes occur at the scale of DNA.

As I have neither the resources nor any working collaboration with other researchers, this proposal is based on epidemiological data alone which suggest the involvement of hydronium energised by passing through electrically live conductive water pipes. I am putting this proposal forward in the hope that the reasoning behind it is sufficient despite lack of referencing to computer modelling or other analyses.

Noam Agmon of Jerusalem University helped earlier with explaining how the bond strengths in the hydrated hydronium structure are stronger inward and thus the hydronium is not as strongly bonded to the water matrix as it would otherwise be. This helps explain why the hydronium, while larger than a water molecule, is more mobile.

Recent research has suggested that the hydrated hydronium is often on the outside of the structure.

What is not clear so far is that the reaction of adding part of a water molecule to the damaged guanine seems to require more energy than is available. Barnett, Bongiorno, Cleveland, Joy, Landman and Schuster have proposed that the bonding of part of a water molecule to a guanine in DNA is initiated by the presence of a sodium counter-ion near the electron

"hole." However, the epidemiology of the cancer epidemic suggests that it is hydronium which is central to the mutation process rather than a combination of a water molecule and a sodium. Therefore:

I propose:

In 1953 Watson and Crick proposed a double helix structure for DNA, having doubts about the then proposed triple helix. That proposal has now become the accepted model.

Since then the sequence of events which lead from damage of DNA to mutation has not been fully understood. There remains a change to the guanine molecule at C8 which appears to require more energy than is available.

In an effort to find a source of this energy, researchers have proposed that the presence of a sodium counter-ion in the vicinity of an electron "hole" in the guanine, caused by oxidative damage, provides the energy necessary to initiate the attachment of a water molecule to that hole. That proposal suggests that an oxygen with one of it's hydrogens attaches at C8 with the other hydrogen transferring to an adjacent water molecule. The partner water molecule thus becomes an hydronium to bond with an adjacent phosphate to complete the change to the guanine. However, the probability of these events occurring spontaneously and concurrently is too high and does not correlate in any way with the observed increase in cancers over the past 60 years. Also, animal studies have suggested that phosphate buffer pairs mitigate the over-production of reducing agents in the presence of oxidising free radicals and this seems contrary to the notion of a phosphate facilitating mutation.

I propose that a more probable mechanism involves an hydronium rather than a sodium cation. I further propose that the hydronium has an altered valency as a result of passing through electrically live water piping.

The mutation begins with an oxidising free radical scavenging an electron from DNA. The electron "hole" may migrate along the DNA until it reaches a guanine, where no further reaction can take place as the guanine has the lowest attachment bonding to it's electrons, or it may occur at the guanine itself.

In this proposed mechanism, the hydronium involved in forming an OH on the damaged guanine at C8 is not short lived. The hydronium starts as a long lived cation which is energised by the negative half wave of alternating current and can be regarded as behaving like an anion. It is in a larger form of hydronium molecule structure rather than a simple H_3O. The hydrated proton resides on the surface of this molecule rather than being within a cage. In this surface location on the

molecule the hydronium can bond with guanine at the electron hole. The process is completed when the oxidising free radical which began the process, but which now has it's electron, is bonded to that part of the now de-energised hydronium structure remaining after an OH component has been bonded to the guanine at C8. Thus the proposed process completes in the presence of two molecules, being the oxidising free radical and the energised hydronium, and requires no catalysts for the reaction to complete.

This proposal must be regarded as unproved, but offers a possible epidemiologically based explanation for the change to guanine at C8.

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