Independent evolution of the bitter taste sensitivity in humans and chimpanzees a...

Independent Evolution of Bitter-taste sensitivity in Humans and Chimpanzees Background: One of the first described and widely accepted balanced polymorphism genes are those encoding for bitter-taste sensitivity. During its initial description it was observed by Fisher et al In 1939 that both humans and chimpanzees share this mutation, and share also its origin before the human-chimpanzee divergence. They accurately described that this trait, phenotypically expressed by bitter taste to phenylthiocarbamide (PTC), is controlled by 2 alleles present at the same loci in both species.

The authors of this article however disputed the fact that Fisher et al At similar loci, are not shared with humans. It is worth noting that the balanced polymorphism here is maintained by the usefulness of this trait in averting poisonous food for animals and food selection and diet choices for humans. Introduction: Two alleles at the TAS2R38 locus control the PTC sensitivity in humans. The same locus in responsible for controlling the taster and non-taster phenotype in chimpanzees. These genes encode for G-protein-coupled receptors in the taste buds.

Evidence points to variation in this gene even at a humanoid levels and early humans during their evolution leading to variation in PTC taste sensitivity among humans and primates as well. When this was described by Fisher et al, they postulated an equally prevalent dominant and recessive allele of the taster gene in both humans and chimpanzees. They also pointed to the fact that this can only happen if the mutation is shared by both before they diverged. Methods, Discussion and analysis: The human locus, or hTAS2R38 gene, is a 1kb located on chromosome 7.

The genotypic variation in PTC sensitivity is related to variations at 3 positions: 49, 262 and 296 leading to either a hTAS2R38(PAV) meaning proline, alanine and valine; or hTAS2R38(AVI) meaning alanine, valine, and isoleucine. In vitro and in vivo studies showed that the first variation hTAS2R3(PAV) is related to tasters whereas the other is related yo non-tasters. To evaluate the original Fisher et al hypothesis, they sequenced the same locus, chTAS2R38, in chimpanzees. They selected 86 chimpanzees (37 wild and 49 captive born).

Seven variable nucleotide positions were detected, none of which is shared by hTAS2R38, the human genotype. However, they found a T to G (ATG to AGG) substitution of the initiation codon leading to attenuation of chTAS2R38 translation. Thus this may be responsible for non-taster in chimpanzees. Depending on the above analysis, in addition to statistical evidence that " not only has non-taster alleles been independently derived in humans and chimpanzees, but they seem to have arisen under different selective regimes" (pp. 931), they were able to prove the non-validity of Fisher's hypothesis.

They further investigated the translation and transcription effect of the above mentioned chTAS2R38 mutation (ATG to AGG) in vitro and in vivo. These studies showed that the polypeptide produced by the chTAS2R38(AGG) is 96 aa residues short and this mutation will lead to a nonfunctional PTC receptor and consequently a non-taster phenotype. To further confirm the functional PTC receptor polypeptide product of chTAS2R38(ATG), they cloned the gene and transfected it into cells in vitro.

When these cells are subjected to micromolar quantities of PTC there was a considerable increase in cytosolic Ca++.

This was not observed when the chTAS2R38(AGG) was used. The later condition was completely reveresed when an initiation codon was introduced upstream thus eliminating the effect of AGG. To test the above in vitro results, i. e. chTAS2R38(ATG) is the taster genotype chimpanzees and chTAS2R38(AGG) the non-taster genotype, they run in vivo testing. They presented 42 captive born chimpanzees with apples impregnated with PTC (control being water). The chimpanzee response was evaluated by a behavioral specialist blinded to the genotype. The phenotype was correlated to the genotype for each animal.

According to the genotype analysis, 2 groups were formed: predicted tasters and non-tasters. The correlation between the genotype and PTC taste sensitivity were statistically significant. This proved the authors hypothesis and further confirmed the rejection of the original Fisher et al common origin hypothesis for the PTC taste sensitivity between human and chimpanzees maintained by balance polymorphism. Critique: This experimental study is built around a specific clear purpose which the authors were able to achieve. Their aim was to disprove the original 65 years old widely accepted bitter taste balance polymorphism hypothesis by

Fisher et al in which it is suggested that humans and chimpanzees share this variance in taste sensitivity due to a shared common origin of the involved alleles. This aim was skillfully achieved by applying various scientific and statistical methods. In view of the recent advances in human and animal

genomics whereby the whole genome has been sequenced, the significance of such work may be diminished, however, the ability to correlate genotypic and phenotypic characteristics, both in vivo and in vitro present a very important step in understanding functional genomics interspecies relationships.

Moreover, the utilization of scientific advancements to correct previous misconceptions is very important as well. As mentioned above, the recent sequencing of the human and primate genomes can be used in correlation with such studies of functional genomics to add a lot to our understanding of genetics. Work cited Wooding, Stephen, Bernd Bufe, Christina Grassi, Michael T. Howard, Anne C. Stone, Maribel Vazquez, Diane M. Dunn, Wolfgang Meyerhof, Robert B. Weiss, and Michael J. Bamshad. “ Independent Evolution of Bitter-taste Sensitivity in Humans and Chimpanzees. ” Nature 440. 7086 (2006): 930-34.