

Original Research Article

Study of TAS2R38 Genes for Bitter Taste Depending on Heredity of Taster in Three Generations of Mathur Population

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Received: 04/12/2015

Revised: 23/12/2015

Accepted: 30/12/2015

ABSTRACT

The present study was done in humans, based on responses to some bitter compounds. Some show a bimodal distribution that distinguishes two phenotypes, tasters and non-tasters. Phenylthiourea (PTU), is an organosulfur thiourea containing a phenyl ring. The main objective of this study was to determine the gene frequencies of dominant and recessive alleles for PTC taste sensitivity among my 20 families (relatives). The present study provides brief information on the distribution of PTC tasters and non-tasters in different populations of Mathurs in many parts of India and Abroad. Members selected were in the age range of 05-75 years, to observe taste sensitivity for Phenylthiourea. 60 individuals of this population were divided into PTC tasters (T) and non-tasters (NT) and obtained the following results from a set of 26 Mathur families in my relation. According to the study, the exceptional four families with NT-NT Parents having Taster children, might possibly be because of the tt (recessive gene) of TAS2R 38 gene at 7q. There were equally male and female tasters and non-tasters in this population, tasters (50%) males (50%) females. Individuals were divided into PTC tasters (T) and non-tasters (NT). In the first generation, 8 couples were T & NT, which had either tasters and non-tasters offsprings and two couples were NT x NT who had one taster and one T child. In the second generation, 14 couples were with phenotype T & NT, out of which all these couples had T and NT, both genes in their children except two families, which were NT x NT and had taster (T) child. No Taster-Taster couples were seen in this Mathur population. Among the 30 males and 30 females under study, 18 females were Non-tasters while 12 females came out to be tasters. In males 10 were NTs and 20 were tasters; more of females were non-tasters and more of males were tasters. This study has been proved to be challenging as different results have been obtained from the studies done on Muslim Population in Hyderabad. So, study shows that genes may vary in different populations, genders as well as on environmental changes during life time locus. It can be due to atavism as it has been observed that the recessive gene did not express for last 4 generations and then suddenly expressed in one generation. One more possible reason might be mutation must have occurred in the children of NT x NT parents. It was noticed that these families migrated from their original habitat. And hence this may also be the possible reason of changes in genes, as genes are also affected by environmental changes, both internal environment as well as external environment. Cast and sex also play role in genetic dominance and recessiveness.

Keywords: Phenylthiourea, Taste Buds, Humans, Heredity, Genes, Mathurs.

INTRODUCTION

Bitter perception generally occurs through bitter taste receptors located on the surface of taste cells of the tongue. [1] These receptors are encoded by T2R genes that show 25-89% amino acid sequence

identity between the 60 different members of this gene family. These differences presumably allow a wide variety of different chemical shapes, sizes, and functionalities to be bound by these receptors and perceived as bitter. The

importance of the ability to taste bitter chemical compound PTC was realized by, [2] it has the unusual property that it either tastes very bitter or is virtually tasteless, depending on the genetic makeup of the taster. The inheritance of the ability to taste PTC was dependent on a single autosomal dominant gene. PTC is a bitter tasting, harmless chemical compound, [3,4] which is a member of a class of compounds known as “thioureas.” These compounds are having the chemical group N-C=S, which is responsible for their characteristic bitter taste. [5,6]

Much of the variation in tasting of PTC is associated with polymorphism at the TAS2R38 taste receptor gene. [7] In humans, there are three SNPs (single nucleotide polymorphisms) along the gene that may render its proteins unresponsive. [8] There is conflicting evidence as to whether the inheritance of this trait is dominant or incompletely dominant. [9] Any person with a single functional copy of this gene can make the protein and is sensitive to PTC. Some studies have shown that homozygous tasters experience a more intense bitterness than people that are heterozygous; other studies have indicated that another gene may determine taste sensitivity. [10]

Kim et al. [11] have identified a small region on Chromosome 7q, and harbours a gene that encodes a member of the TAS2R bitter taste receptor family. A major locus on chromosome 7q35-q36 and a secondary locus on Chromosome 16p have been localized by genome scan for PTC taster gene. [12] Tasters are those who taste the substance (PTC) while non-tasters cannot taste at all. Tasters have the genotype TT and the non-tasters have tt.

The frequency of PTC taster and non-taster alleles varies in different human populations. [13] The widespread occurrence of non-taster alleles at intermediate frequencies, much more common than recessive alleles conferring genetic disease, across many isolated

populations, suggests that this polymorphism may have been maintained through balancing selection. [14] Differential ability to taste the bitter compound phenylthiocarbamide (PTC) was discovered more than 80 years ago. [15] Since then, PTC tasting ability has been mapped to chromosome 7q and, several years later, was shown to be directly related to TAS2R38 genotype. There are three common polymorphisms in the TAS2R38 gene- A49P, V262A, and I296V—which combine to form two common haplotypes and several other very rare haplotypes. The two common haplotypes are AVI (often called “nontaster”) and PAV (often called “taster”). Varying combinations of these haplotypes will yield homozygotes- PAV/PAV and AVI/AVI- and heterozygotes- PAV/AVI. These genotypes can account for up to 85% of the variation in PTC tasting ability: people possessing two copies of the PAV polymorphism report PTC to be more bitter than TAS2R38 heterozygotes, and people possessing two copies of the AVI/AVI polymorphism often report PTC as being essentially tasteless. These polymorphisms are hypothesized to affect taste by altering G-protein-binding domains.

Because bitter substances are usually toxic, the presence of a “nontaster” geno- and phenotype seems evolutionarily undesirable. Several studies have suggested, however, that the AVI polymorphism may code for an entirely new receptor which processes a different and as-yet undiscovered bitter compound. [16,3] Furthermore, the presence of the nontaster allele may reflect the desirability of maintaining a mostly heterozygous population; this group of people may possess flexibility in their bitter taste perception, enabling them to avoid a greater number of toxins than either homozygotic group. Other studies, however, suggest that the AVI nontaster

genotype has no functional ligand. This genotypical alteration of taste phenotype is currently unique to TAS2R38. Though genotype has been proposed as a mechanism for determining individual taste preferences, TAS2R38 is so far the first and only taste receptor to display this property.

Chimpanzees and orangutans also vary in their ability to taste PTC, with the proportions of tasters and non-tasters similar to that in humans. [17,19] The ability to taste PTC is an ancestral trait of hominids that has been independently lost in humans and chimpanzees, through distinct mutations at TAS2R38. [15,18] PTC sensitivity is often used as an example of a simple Mendelian trait with dominant inheritance. However, tasters vary greatly in their sensitivity to PTC. [16,17] And while the PTC gene has about 85% of the total influence over whether someone is a taster or a non-taster, there are many other things that affect PTC tasting ability. Because there were no taster offspring of two non-taster parents, this fits the model of PTC tasting being determined by two alleles of a single gene, with tasting being dominant over non-tasting.

The present study was carried out to analyze the prevalence of PTC taste sensitivity and to determine the gene frequencies among some Mathur populations of different places in India and America. This study has some physiological relevance to highlight the adaptability of endogamous groups to behavioral traits in different places.

MATERIAL AND METHODS

- 1) For the present study, PTC Paper stripes (Pre- Prepared) of Precision Laboratories (Branded), were used.
- 2) Consents were filled and signed by my all relatives who tasted the PTC stripes and were involved in my present research.
- 3) I divided 60 individuals into PTC tasters (T) and non-tasters (NT) and

obtained the following results from a set of 26 mathur families (4 generations) in my relation.

- 4) The present study was carried out to analyze the prevalence of PTC taste sensitivity and to determine the gene frequencies among my relative Mathur population of India and Abroad, (endogenous groups).
- 5) Healthy individuals of both sexes (Male-30Female-30) belonging to Mathur populations (20 families, 60 individuals) of my relatives in India and Abroad were selected with the age range of 05-75 years, to observe taste sensitivity for Phenylthiourea.
- 6) Individuals involved in my research were between 5 years to 75 years agers.

RESULTS AND DISCUSSIONS

The phenotypic frequency of tasters was exactly the same as compared to non-tasters, and there were equally male and female tasters and non tasters in my family, tasters (50%) males (50%). I divided individuals into PTC tasters (T) and non-tasters (NT) and obtained the following results from a set of 26 families, 60 individuals.

In the first generation, 8 couples were T &NT ,which had either tasters and non tasters offsprings and two couple were NT xNT who had one taster and one T child (Table:1&3)

In the second generation,14 couples were with phenotype T & NT, out which all theses couple had T and NT, both genes in their children except two families ,which were NTxNT and had taster (T) child .No Taster x Taster couples were seen in this population.(Table : 1&2)

Table: 1- Showing Combinations of taster and non –taster parents and their off springs.

Parents.	T offspring	NT offspring
T x T	NIL	NIL
T x NT	27	18
NT x NT	4	2

Table: 2-Showing number of tasters and non-tasters in relation to sex.

Total Male Tasters	Total Female Tasters	Total Male Non-Tasters	Total Female Non Tasters
20	12	10	18

Table: 3-Showing number of families in three generations with different combinations of tasters and non-tasters

S.no.	No. of Families (TxNT)	No. of Families (NTxNT)	No. of Families (TxT)
Generation :1	04	2	NIL
Generation :2	12	2	NIL
Generation :3	NIL	NIL	2
Generation :4	01	NIL	NIL

All 46 individuals among 60 of my relatives followed the hereditary trend, i.e., all the off springs who were tasters, had either of the parents as tasters (but not both parents). Only two families had Non taster parents (both father and mother), but son were taster. Because in 76.66% cases no taster offspring were observed, of two non-taster parents., this fits the model of PTC tasting being determined by two alleles of a single gene, with tasting being dominant over non-tasting.

According to me, the exceptional four families with NT-NT Parents having Taster children might possibly be because of the tt (recessive gene) of TAS2R 38 gene at 7 q locus. It can be atavism as it has been observed the gene getting recessive for 3 generations and then expressing in next generation. One more possible reason might be mutation must have occurred in the children of NT xNT parents. By this result we can also conclude that sex and migration (Environmental changes) also have role in genetic changes to occur. As per our observations, the families showing NTxNT parents with Taster off spring migrated to different parts of India and Abroad, from their original habitat. So, this can be a responsible factor which might have caused mutations in the TAS2R 38 genes, as already studied that external and internal factors are responsible for genetic mutations.

CONCLUSION

The frequency of PTC tasters is equal to non-tasters and the females have equal non-taster phenotypes as compared to males, which is a different output from

my study from the study done on Muslim population attempted in Uttar Pradesh, India previously. These are described here for comparison. The present work shows some differences from earlier studies on PTC taste blindness among Mathur populations of India and America.

The experience of bitterness occurs after certain chemicals contact taste receptors located in cells on the surface of the tongue. Some investigators hypothesize that this sense provides information so that people do not ingest bitter-tasting toxic chemicals. [3] Studies of sensitivity to the bitter tasting compound, PTC have shown this to be an inherited trait and non-taster status has been linked to a variety of medical and health disorders. [4] A high incidence of non-tasters has been reported among patients with nodular goiter, [5,6] congenital athyreotic cretinism, [7, 8] and dental caries. [9] Bitter perception generally occurs through bitter taste receptors located on the surface of taste cells of the tongue. [10] These receptors are encoded by T2R genes that show 25-89% amino acid sequence identity between the 25 different members of this gene family. These differences presumably allow a wide variety of different chemical shapes, sizes, and functionalities to be bound by these receptors and perceived as bitter.

PTC tasting is largely determined by a single gene, TAS2R8, with two common alleles, and the allele for tasting is mostly dominant over the allele for non-tasting. However, both classical family and twin studies, and modern molecular genotyping, show that there are other genes or environmental factors that

influence PTC tasting. As a result, there is a continuous range of PTC tasting, not absolute separation into tasters and non-tasters. PTC tasting would be a fascinating subject for an advanced genetics class, but it does not fit the one-gene, two-allele myth well enough to be used to demonstrate simple Mendelian genetics. The frequency of PTC tasters is equal to non-tasters and the females have lower non-taster phenotypes as compared to males. This type of study will provide background information about genetic structure of population and serves as useful interaction of genetics. According to me, the exceptional four families with NT-NT Parents having Taster children might possibly be because of the *tt* (recessive gene) of TAS2R38 gene at 7q locus. It can be due to atavism as it has been observed that the recessive gene did not express for last 4-5 generations and then suddenly expressed in one generation. One more possible reason might be mutation must have occurred in the children of NT xNT parents. It was noticed that these families migrated from their original habitat. And hence this may also be the possible reason of changes in genes, as genes are also affected by environmental changes, both internal environment as well as external environment. Healthy individuals of both sexes (Male-30 Female-30) belonging to Mathur populations (26 families, 60 individuals) of my relatives in India and Abroad were selected with the age range of 05-75 years, to observe taste sensitivity for Phenylthiourea. 60 individuals of this population were divided into PTC tasters (T) and non-tasters (NT) and obtained the following results from a set of 26 Mathur families in my relation. According to me, the exceptional four families with NT-NT Parents having Taster children, might possibly be because of the *tt* (recessive gene) of TAS2R38 gene at 7q. There were equally male and female tasters and non tasters in my family, tasters (50%)

males (50%). This observation was statistically significant. Individuals were divided into PTC tasters (T) and non-tasters (NT) and obtained the following results from a set of 26 families, 60 individuals. In the first generation, 8 couples were T & NT, which had either tasters and non tasters offspring and two couples were NT x NT who had one taster and one T child. In the second generation, 14 couples were with phenotype T & NT, out of which all these couples had T and NT, both genes in their children except two families, which were NT x NT and had taster (T) child. No Taster-Taster couples were seen in this Mathur population. Among the 30 males and 30 females under study, 18 females were Non-tasters while 12 females came out to be tasters. In males 10 were NTs and 20 were tasters; more of females were non tasters and more of males were tasters. This study has been proved to be challenging as different results have been obtained from the studies done on Muslim Population in Hyderabad. So, study shows that genes may vary in different populations, genders as well as on environmental changes during life time. Locus it can be due to atavism as it has been observed that the recessive gene did not express for last 4 generations and then suddenly expressed in one generation. One more possible reason might be mutation must have occurred in the children of NT x NT parents. It was noticed that these families migrated from their original habitat. And hence this may also be the possible reason of changes in genes, as genes are also affected by environmental changes, both internal environment as well as external environment. [6] Atavism, mutations due to changes in external and internal environment, cast and sex equally play role in genetic makeup and hence TAS2R38 taster gene showed such variations in results from studies done on muslim population in Hyderabad and Mathur population in India and Abroad.

ACKNOWLEDGEMENT

I dedicate my work to my Nana, Nani, Arvind Mama and Prason Mama and my brothers Kartik and Mohit Without their blessings my work was not possible. I acknowledge my heartiest feelings for the support of my Mother Dr. Manisha Mathur and my Dad Mr. Sanjay Mathur without whose guidance I stand nowhere and who acted like a backbone in guiding me and supported me during my study. I thank from bottom of my heart to all the family members who were involved in this research and believed in me and became model for my research. Last but not the least, I would like to extend my sincere thanks to my only sister Rimjhim Mathur who extended great help to me at the time of research in collecting the data. Without whom it would have been a lonely lab.

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How to cite this article: Mathur G. Study of TAS2R38 genes for bitter taste depending on heredity of taster in three generations of Mathur population. *Int J Health Sci Res.* 2016; 6(1):410-416.

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