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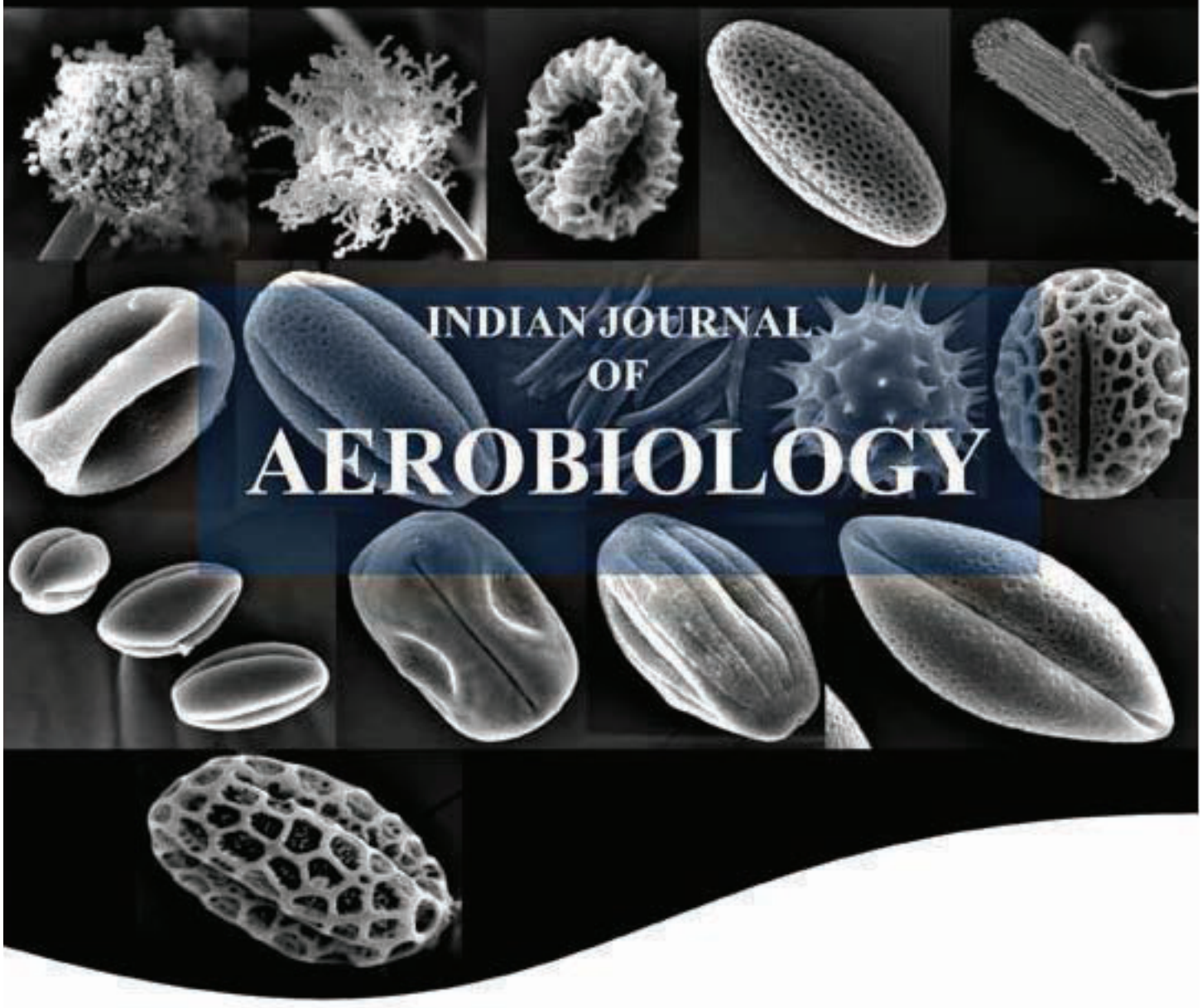

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Editorial

I am happy to publish the 31st volume of Indian Journal of Aerobiology. This volume is comprised of five important papers in various aspect of Aerobiology. In this volume you will get two review articles as well as three original research articles. The first review article entitled, "The apocalypse of p-value" by Dr. Arindom chakraborty is a multidisciplinary approach, especially is found to be very important aspect in Biological research. Due to the misuses and misconceptions of p-values, the reviewer has substantiated p-value with other approaches such as confidence, or Bayesian methods; alternative measures of evidence like likelihood ratios or Bayes factors. A comprehensive review on food allergy was done by Dr. Nandini Ghosh and Dr. Swati Gupta Bhattacharya. The results highlighted the world wide prevalence of food allergy with special emphasis on food allergy in India. They also emphasized on the application of immunotherapy in control of food allergy. The original research article entitled, "Circadian atmospheric pollen incidence of Hyptis suaveolens (L.) Poit by Satyajit Oraon and Subrata Mondal is found to be an interesting aspect on pollination biology. The authors have enlightened on role of insect visitor on pollination mechanism in Lamiaceae. A preliminary study on bioaerosols and their relation to allergic diseases in Ara town of Bihar was conducted by Usha Kumari and Raj Kishor Sharma. The last research article by T. Gaine and others highlighted on the effect of air pollutants like $PM_{2.5}$, PM_{10}, NO_2, SO_2, NH_3 on respiratory health problems of roadside exposed people in Kolkata metropolis. I received immense help from many persons, otherwise it would have not been possible for me to complete this work. First of all, I would like to express my appreciation and thanks to Prof. Kashinath Bhattacharya of Visva-Bharati University for his kind help in every aspect in composing this volume. Thanks are also due to my students specially, Pampa Chakraborty (Associate Editor), Partha Karak, Gaurab Sircar and Soumyo Subhra Gupta. We hope that you will find this volume valuable and will consider submitting your own work in the subsequent volumes. The next volume i.e., Vol. 32(2019) will be published in November, 2019. We welcome your comments so that we may improve the journal in near future.

Swati Gupta Bhattacharya

Chief Editor

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THE APOCALYPSE OF P-VALUE

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P-values are widely used in science to test null hypotheses. But P values are slippery, and sometimes, significant P values vanish when experiments and statistical analyses are repeated. This paper provides a non-technical introduction to the p-value statistic. In light of misuses of and misconceptions concerning p-values, in this paper, other approaches like confidence, or Bayesian methods; alternative measures of evidence such as likelihood ratios or Bayes factors will be discussed.

Key Words: P-value, null hypothesis, Bayesian methods, Bayes factors

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INTRODUCTION

To quantify the statistical significance of observed results, p-values are widely used in both natural and social sciences. With the emergence of computational social science, which relies mostly on analyzing large scale datasets, the popularity of p-values increased even further. In a study by Chavalarias *et al*.[2], it can be seen that within a span of 25 years (1990-2015), the usage of p-values has increased in manifolds. According to the study, reporting p-values in abstracts has increased from 7.3% in 1990 to 15.6% in 2014. MEDLINE and PMC databases have been used for this text mining. In 2014, p-values were reported in 33% of abstracts from 151 core clinical journals (n = 29725 abstracts), 35.7% of meta-analyses (n = 5620 abstracts), 38.9% of clinical trials (n = 4624), 54.8% of randomized controlled trials (n = 13544)[2]. According to Gerber, Green and Nickerson[8], there is a publication bias that favors papers that successfully reject the null hypothesis. Such publication bias may have resulted in the spread of usage of p-vathe lue. Unfortunately p-v,alues are routinely misunderstood and misused by many practitioners. The p-value is probably the most ubiquitous and the same time misunderstood, misinterpreted, and occasionally miscalculated index[5] in all of the biomedical research. The standard significance level of 0.05 produces an overall false discovery rate (FDR) that is far higher, more like 30%[12].

A lot of public debate has already been taken place after an editorial decision of *Basic and Applied Social Psychology* (BASP) to ban the use of p-values[11]. This decision evoked responses from the International Society for Bayesian Analysis (ISBA). Among others, the American Statistical Association (ASA)[13] also stated the use of p-values in decision making. Demidenko[3] in subsequent fallout has mentioned that p-value has "an ambiguous interpretation". In a publication in The American Statistician, Andrew Gelman put much of the blame on statistical education[6,7]. The difficulty in the interpretation of p-value was very much there since the inception of the idea by R.A. Fisher. An informal system was proposed by him which he never could describe straightforwardly what it meant from an inferential standpoint[9].

The term "significant", deliberately coined by Fisher[4], was attached to small p-values. In the first modern

statistical handbook *Statistical Methods for Research Workers* (1962), he wrote:

Personally, the writer prefers to set a low standard of significance at the 5 per cent point….A scientific fact should be regarded as experimentally established only if a properly designed experiment rarely fails to give this level of significance

In other words, the operational meaning of a p-value less than 0.05 was mere that one should repeat the experiment.

For better or worse, Null Hypothesis Significance Testing (NHST)[1,5] with its associated "P" values has become the standard for most published medical literature. However, "P" values are difficult to understand and interpret, even for established researchers. This has led to a lot of unfavourable attention to this issue in the recent past, especially in the context of research misconduct. It has become the perennial butt of scientific cartoonists such as Randall Munroe at http://xkcd.com [Figure 1]. It is also equally important to go beyond the criticism and to understand what information is contained in a p-value.

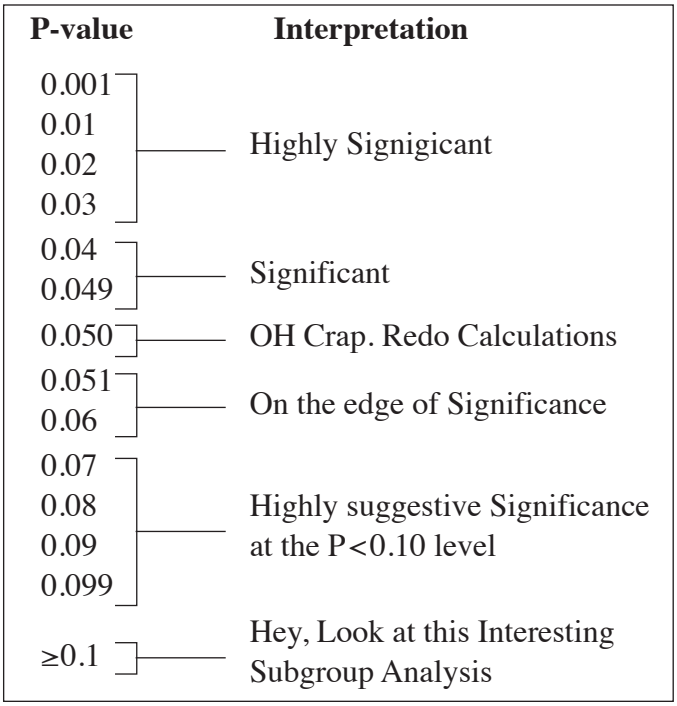

Fig. 1: "If all else fails, use 'significant at P > 0.05 level' and hope no one notices." (http://xkcd.com/1478/, Randall Munroe, Creative Commons Attribution-Non Commercial 2.5 License)

WHAT IS A P-VALUE?

The idea of generalizing results from a sample to the population is the basic idea of statistical inference. For the validity of the generalization, one may use an objective measure from some significant tests. With the introduction of the machinery of "hypothesis testing", the p-value story got incomparably more complicated. In a standard approach of hypothesis testing, one has a null hypothesis (H_0) and an alternative hypothesis (H_1). The alternative hypothesis is just the opposite and mutually exclusive patterns regarding some phenomena. In essence, the main purpose of hythe pothesis test is to help the researcher to take a decision about two competing views of reality. Apart from null and alternative hypothesis, testing involves "accepting and rejecting" hypotheses, type I, II error rates, "power" and other related ideas.

The formal view of p-value as a probability conditional on the null is mathematically correct but typically irrelevant to research goals (Gelman, 2013)[6]. In algebraic notation

$$p\text{-}value = Prob\,(X \geq x|H_0)$$

Where "X" is a random variable corresponding to some way of summarizing data (such as mean or proportion), "x" is the observed value of that summary in the current data (Figure 1) (Goodman, 2008)[9]. p-value statistic is a conditional probability, the probability of obtaining the observed or more extreme result given that the null hypothesis is true.

Theoretically speaking, the p-value is a continuous measure of evidence, but in practice, it is trichotomized approximately into highly significant, marginally significant and not statistically significant at conventional levels, with cut offs at $p \leq 0.01$, $p \leq 0.05$ and $p > 0.10$ (Gelman, 2016)[7].

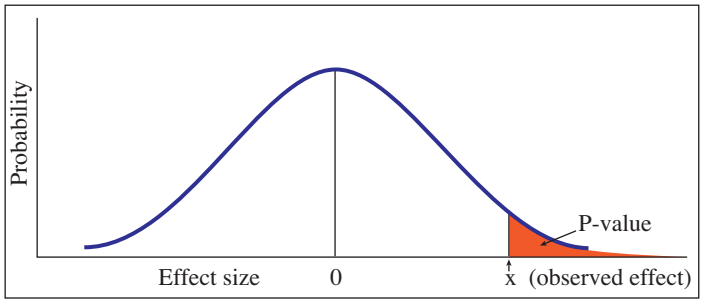

Fig. 2: Graphical depiction of the definition of a (one-sided) p-value. The curve represents the probability of every observed outcome under the null hypothesis. The p-value is the probability of the observed outcome (x) plus all "more extreme" outcomes, represented by the shaded "tail area" (Goodman, 2008)9

One big problem with p-values is that they can not be compared easily. For example, confusion may arise when the sharp cut-off misleadingly separates the

$p = 0.051$ cases from $p = 0.049$. Even an apparently huge difference between clearly significant and clearly non-significant is not itself statistically significant.

The p-value is itself a statistics and can be a noisy measure of evidence. This nature is common with any mathematically equivalent procedure, such as summarizing results by whether the 95% confidence interval includes zero (Gelman, 2013)[5]. Consider the famous Anscombe's[1] data where four different relationships between X and Y can be summarized by the same statistics (F, standard error, b, beta etc.). Figure 2, replicates his data, presenting four different relationships between variables with same significant p-value of 0.002 and R square value is 0.67. However graphical analysis shows that the nature of relationship between the variables is markedly different. As long as the researcher only examines p-value, he/she would never get this information.

Six principles formulated in the statement of the American Statistical Association (ASA)

A lot of literature has come out reiterating misconceptions regarding p-value (Goodman9, Greenland *et. al.*[10] and references therein). Much distortion arises from basic misunderstanding of what p-values and their relatives (such as confidence intervals) do not tell us. In the statement released in 2016, ASA reported that the p-value could be a useful statistical measure; it is commonly misused and misinterpreted. This has led to some scientific journals discouraging the use of p-values, and some scientists and statisticians recommending their abandonment, with some arguments essentially unchanged since p-values were first introduced (Wasserstein and Lazar[13]). This statement articulates in non-technical terms a few select principles that could improve the conduct or interpretation of quantitative science, according to widespread consensus in the statistical community. To be fair, even the ASA acknowledges that the practice of statistics is not a straightforward endeavour, which clearly defined rules of agreement.

Consider a specific clinical research scenario to demonstrate each of the statements issued by ASA[14]. Suppose a new antiemetic Drug X has been tested against placebo in a sample of patients undergoing day care gynaecological surgery. The number of patients vomiting on the first postoperative day was lower in the treatment group (45/100) compared to the placebo group (60/100) with a P-value of 0.03.

Ad 1. P-values can indicate how incompatible the data are with a specified statistical model.

"A P-value is one of the ways of summarizing the incompatibility between the observed data and a proposed model for the data. The most common model we use is the so-called "null hypothesis," which in practice essentially proposes that Drug X does not affect whatsoever. The smaller the p-value is, the larger the incompatibility of the data with the null hypothesis. A p-value of 0.03 says that 45% of patients in the Drug X group will vomit by chance in 3% of samples drawn from a population, in which Drug X does not affect. It does not say anything about the population in which Drug X affects."

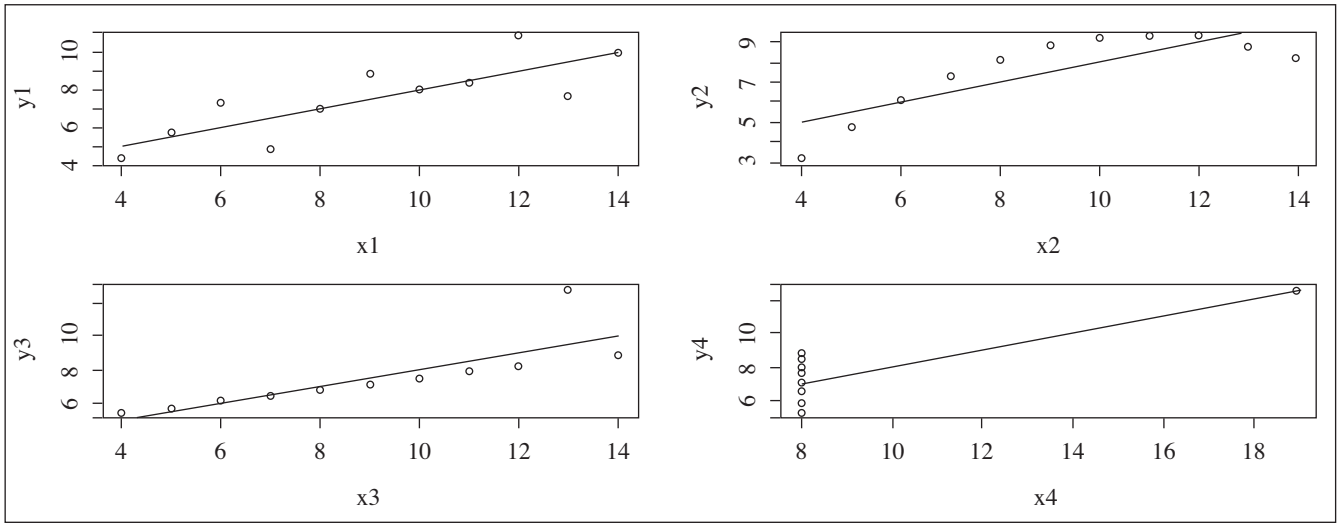

Fig. 3: Replication of Anscombe's1 (1973) data

Ad 2. P-values do not measure the probability that the studied hypothesis is true or the probability that the data were produced by random chance alone.

"A p-value of 0.03 does not mean that the probability of Drug X not having any effect is 3%. Neither does it mean that 45% of patients in the Drug X group vomited by chance. As the last sentences in Principle 1 explain, a p-value is a statement about the data in the context of a null hypothesis. It does not say anything about the null hypothesis or the alternate hypothesis. It is not an error probability."

Ad 3. Scientific conclusions and business or policy decisions should not be based only on whether a p-value passes a specific threshold.

"The results of this study should not lead you to conclude that Drug X reduces postoperative nausea. A conclusion is not binary yes/no because of one study. Researchers should consider the context of the study while deriving scientific inferences. These should include the design of the study (e.g., improper randomization or poor concealment of allocation, leading to selection, or other types of biases), the quality of measurements (e.g., if vomiting is not documented in real time, but is based on recollection at an interview conducted a week later, leading to recollection bias), the external evidence for the phenomenon under study (e.g., studies showing that Drug X does not penetrate the blood-brain barrier, or that it does, or that it causes profound sedation), and the validity of statistical assumptions that underlie the data analysis. A low p-value should never be the sole basis for a scientific claim."

Ad 4. Proper inference requires full reporting and transparency

"All the analyses done should be reported fully. Conducting multiple statistical tests on the same data and reporting only those with low p-values makes the reported analysis uninterpretable. For example, in the given study, the number of patients vomiting may have been analyzed for the period that the patient was in the post anesthesia care unit, or the first 6, 24, 48 or 72 h. The number of episodes of vomiting per patient in all these given periods could have been tested instead of the number of patients. That gives us ten possible tests. If all ten are conducted, and only the ones with a $P < 0.05$ are reported, as commonly happens, it amounts to research misconduct going by various names such as cherry-picking, data dredging, p-hacking, significance chasing, or more politely "selective inference." This is one of the main causes of the spurious excess of statistical significance in published literature. Valid scientific conclusions based on p-values and related statistics cannot be drawn without at least knowing how many and which analyses were conducted, and how those analyses (including p-values) were selected for reporting."

Ad 5. A p-value, or statistical significance, does not measure the size of an effect or the importance of a result.

"The threshold of statistical significance that is commonly used is a p-value of 0.05. This is conventional and arbitrary. It does not convey any meaningful evidence of the size of the effect. A p-value of 0.01 does not mean the effect size is larger than with a p-value of 0.03. The p-value would have been 0.000002 if we had sampled 1000 patients instead of 200 in the Drug X study and obtained the same results (i.e., the same effect size).

Similarly, if an effect is measured to a high enough precision, the p-value will change. For example, if in the above study, the incidence of postoperative vomiting was 46%, the p-value would have been 0.047, which is considered statistically significant. If we then measure the incidence more precisely and get a value of 46.4%, the p-value would become 0.054, considered non significant. A similar change can take place in the opposite direction as well.

Statistical significance does not automatically equate to scientific, human, or economic significance. It might be that even if Drug X does reduce the incidence of vomiting by 15%, this might not be clinically relevant. It might not matter to the patient, if, for example, it produces severe dysphoria which is more uncomfortable for the patient. It might not make economic sense to use the drug, if, for example, it costs 10,000 rupees to treat one patient."

Ad 6. By itself, a p-value does not provide a good measure of evidence regarding a model or hypothesis.

A context-less P-value without any other evidence provides very limited information. A large P-value is not evidence of your alternate hypothesis since an arbitrarily

large number of other hypotheses are consistent with the observed data. Data analysis should not conclude with the calculation of the p-value. Correct and careful interpretation of statistical tests requires examining the sizes of effect estimates and confidence limits as well as precise p-values. Other approaches may provide more direct evidence of the size of an effect or the correctness of a hypothesis albeit with further statistical assumptions. These approaches include methods of emphasizing estimation over testing, Bayesian methods, likelihood ratios, and others.

It is perhaps useful to understand how to measure true evidential meaning and look at the p-value from that perspective using Bayes' theorem which can be written in words as:

Odds of null hypothesis after obtaining the data = Odds of null hypothesis before obtaining the data × Bayes' factor

where $odds = \frac{Probaility}{1\text{-}Probability}$

It measures how strongly the observed data are predicted by two competing hypotheses, and is a measure of evidence that has most of the properties that we mistakenly ascribe to the p-value. Bayes' factor is compared to p-value as an evidential factor in Table 1.

Table 1: Evidential properties of Bayes' factor versus p-value

Evidential Property	P-value	Bayes' factor
Information about effect size?	No	Yes
Uses only observed data?	No	Yes
Explicit alternative hypothesis?	No	Yes
Positive evidence?	No	Yes
Sensitivity to stopping rules?	Yes	No
Easily combined across experiments?	No	Yes
Part of formal system of inference?	No	Yes

CONCLUSIONS

In 1880, the biologist T. H. Huxley stated "… it is the customary fate of new truths to begin as heresies and end as superstitions". After almost 60 years of originally being adopted, p-value thresholds have evolved into a superstition. Meanwhile, in the hopes of minimizing harms to current practice, we can offer several guidelines for users and readers of statistics, and re-emphasize some key warnings from our list of misinterpretations:

1. Don't look for a magic alternative to null hypothesis testing (NHT), some other objective mechanical ritual to replace it. It doesn't exist.
2. NEVER draw a conclusion merely based on a p-value
3. Even we seek to generalize from data; we must seek to understand and improve them. A breakthrough to the approach to data, emphasizing "detective work" rather than "sanctification".
4. Report descriptive statistics (mean, sd) and values of statistics (t-value, Z-value, F-value) apart from p-values.
5. Report p-values with a = rather than with a < or a>. Replicability of a null hypothesis rejection is a continuous, increasing function of the complement of its p-value. That is, it is more informative to describe a result as (say) p = .003 rather than as either "p < .05" or "statistically significant," and, similarly, it is more useful to describe a result as (say) p = .07 rather than as either "p > .05" or "not significant". Because the most interpretable characteristic of p values is well captured by counting the number of successive zeros to the right of the decimal point, a useful rule of thumb may be to report p values rounded to the kth decimal place, where k is the position in which the first nonzero digit occurs: For example, p = .3, .07, .002, .0008, ..., 6×10^{-7}.
6. Treat p = .05 as an Interesting, but unconvincing, support for an isolated NHT Result. Therefore, when p =.05 for an isolated finding, an appropriate response on the part of the researcher should be to seek further support by conducting a replication. There will be situations in which an isolated null hypothesis rejection at p = .05 warrants publication without replication. And, when the cost of collecting replication data is very expensive, the information value of reporting precious available evidence may outweigh the more ordinary value of establishing that the observed phenomenon is (in Fisher's sense) demonstrable.

7. Report Results for all important hypothesis tests. Selective reporting is a well-recognized flaw of NHT. There exist several methods for an upward adjustment of p-values. For example, consider a researcher who has used NHT in a series of five tests of the same hypothesis, only one of which yielded a null hypothesis rejection. If the report of this research neglects to mention the four non-rejection results, the reader can have no idea that a p-value reported at (say) $p = .03$ might be more properly interpreted as $p = .15$, a value that is Bonferroni adjusted for alpha inflation and assumes the five tests to be independent of one another.
8. Report Enough Data to Permit Secondary Analysis. Even while undertaking to document some virtues of NHT that justify its use with care and caution. The result of NHT, when reported in minimal form (i.e., as $p \leq \alpha$ or $p > \alpha$), has little use beyond the immediate one of informing a dichotomous reject-non-reject judgment.
9. Always include "statistically" as a prefix to the word "significant".
10. Routinely report effect size in the form of confidence limits which contains all information to be found in significance tests and much more. We sometimes don't report them intentionally (embarrassingly large!!!!!). Larger sample sizes reduce the size of the confidence intervals as they increase the statistical power of NHT.
11. Explain effect size under the context of "clinical/practical" significance

Perhaps architect Daniel Libeskind summed it up best when he said, "Life is not just a series of calculations and a total of statistics, it's about the experience, it's about participation, it is something more complex and more interesting than what is obvious." The solution is not to reform p-values or to replace them with some other statistical summary or threshold, but rather to move toward a greater acceptance of uncertainty and embracing of variation. To achieve this, we recommend saying NO to binary conclusions in our collaboration and consulting projects: resist giving clean answers when that is not warranted by the data. Instead, do the work to present statistical conclusions with uncertainty rather than as dichotomous. In closing, we note that no statistical method is immune to misinterpretation and misuse, but prudent users of statistics will avoid approaches especially prone to serious abuse.

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MODERN PERSPECTIVES OF FOOD ALLERGY: A COMPREHENSIVE REVIEW

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Food allergy is an increasing problem worldwide. In this review routes of sensitization, pathogenesis, prognosis and treatment of food allergy were discussed in details. Food allergy may be IgE mediated or non-IgE mediated or mixed type. In case of IgE mediated food allergy, sensitization may occur either by gastro-intestinal route after ingestion of offending foods or it may be caused by the respiratory route through sensitization of pollen having shared epitopes. In the human body, there are several mechanisms for protection against harmful substances, the overreaction of which may sometime lead to allergic symptoms. Currently, the use of antihistamines and steroids are a mainstay in the treatment of food allergy. Hypo-allergenic vaccines may be useful for the treatment of food allergy in future.

Key Words: Food allergy, IgE, hypersensitivity

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INTRODUCTION

Food allergy is the consequence of immune systems response to otherwise harmless food proteins[1]. Up to 8% of children and 5% of adults suffer from food allergy worldwide[2]. Ninety per cent of food allergies is caused by peanuts, milk, egg, wheat, nuts, soybeans, fish, crustaceans, and shellfish[3]. Tremendous work has been done on the prevalence of food allergy in the developed world. A US population-based study (released in 2013) reveals that food allergies among children increased approximately 50% between 1997 and 2011[4]. In the USA, more than 300,000 children (below 18years) per year visit hospital due to food allergy[5]. Milk allergy is the most common allergic disorder affecting 2.5% of US children[6]. Approximately 12% of children of below 15 years of age suffer from food allergy in UK[7]. Population-based studies on the occurrence of food allergies have been carried out in different countries including Australia[8], Canada[9], Denmark[10], Israel[11], UK[7] and USA[3,12]. However, few reports have been found in Asia, middle-east Africa and South America[2]. Food habit, genetic factor and culture of inhabitants have direct effects on provoking allergic reactions to some food items[13]. The bird's nest soup, a Chinese delicacy, is a major cause of food allergy in Singapore, which signifies how dietary habit contributes to food allergy[14].

Food-induced allergic reactions include a variety of health disorders ranging from adverse skin reaction, respiratory tract, gastrointestinal tract related symptoms to anaphylaxis in extreme cases[15]. Food allergy may occur in IgE mediated or non-IgE mediated/cell-mediated pathways[16]. IgE mediated food allergy is inheritable as nod1 (nucleotide oligomerizing domain receptor) polymorphism is linked with it[16]. Food allergy differs from food intolerance which can be defined as adverse physiologic reactions to food and may occur due to inherent properties of the food (i.e. toxic contaminant, pharmacologically active component) or to characteristics of the host (i.e. metabolic disorders, idiosyncratic responses, psychological disorder)[17].

Nowadays extensive studies have been started in different parts of the world, including India, for a better understanding of the mechanism of food-induced adverse reactions and its symptoms among children and adults. Scientists pay greater attention to the management of food allergy. Currently, management of food

allergies consists of educating the patient to avoid ingesting the responsible allergen and initiating therapy if ingestion occurs. However, new approaches are being developed, including sublingual, oral immunotherapy and others with a hope for the future. In this review, causes of food allergy, its prevalence, pathogenesis and clinical disorders associated with a food allergy are discussed along with its treatment, management and recent research regarding therapy are also elaborated.

TYPES

Food allergy is of 2 types, viz class1 and 2, depending on the route of exposure of allergen (Fig. 1). Class 1 food allergy is caused by food proteins that are ingested through the gastrointestinal tract and stable after digestion; whereas class 2 food allergy occurs when sensitization takes place through respiratory roots by pollen proteins sharing a common epitope with the food items[18]. Caseins in milk, vicillins in peanut, and ovomucoid in egg cause class 1 food allergy. Epitopes of these allergenic proteins are generally glycoproteins and resistant to denaturation by heat or acid[19]. Bet v 1 allergen of birch pollen often cause the pollen-food syndrome in the UK as it is highly cross-reactive with many fruits such as apple, cherry, kiwi, peach (*www.allergyuk.org*). Pollen-fruit cross-reactivity due to polygalacturonase (Cari p 1) of papaya is prevalent in India[20]. So ingestion of these types of fruits can cause class 2 food allergy in case of patients sensitized to respective pollen allergens.

Based on the immunological mechanism involved, food allergies may be further classified into 3 categories: (a) IgE-mediated, (b) non IgE mediated/ cell-mediated and (c) mixed IgE mediated- cell mediated[17]. The commonest type is IgE mediated pathway. During initial sensitization, the allergenic food proteins that are stable after digestion stimulate the production of IgE specific to that protein. It then binds to tissue basophil and mast cell. During subsequent consumptions of that food, this particular protein binds to specific IgE antibodies and trigger release of mediators, such as histamine, prostaglandins, leukotrienes etc., and causing allergic reactions[19]. IgE mediated food allergy is characterized by the quick onset of symptoms after consumption of food. Main symptoms of IgE mediated food allergy include oral allergy syndrome, urticaria, angioedema and anaphylaxis. Cell-mediated food allergy occurs when T cells directly respond to allergenic proteins leading to release of inflammatory mediators. It mostly involves the gastrointestinal tract and skin related disorders, such as dietary-protein enterocolitis, dietary-protein enteropathy, dietary-protein procolitis, Celiac disease, dermatitis herpetiformis etc.[19]. Sometimes both IgE and immune cells are involved in the reaction causing mixed IgE-cell mediated response. Symptoms of such type include atopic dermatitis, eosinophilic gastroenteritis and eosinophilic esophagitis[19].

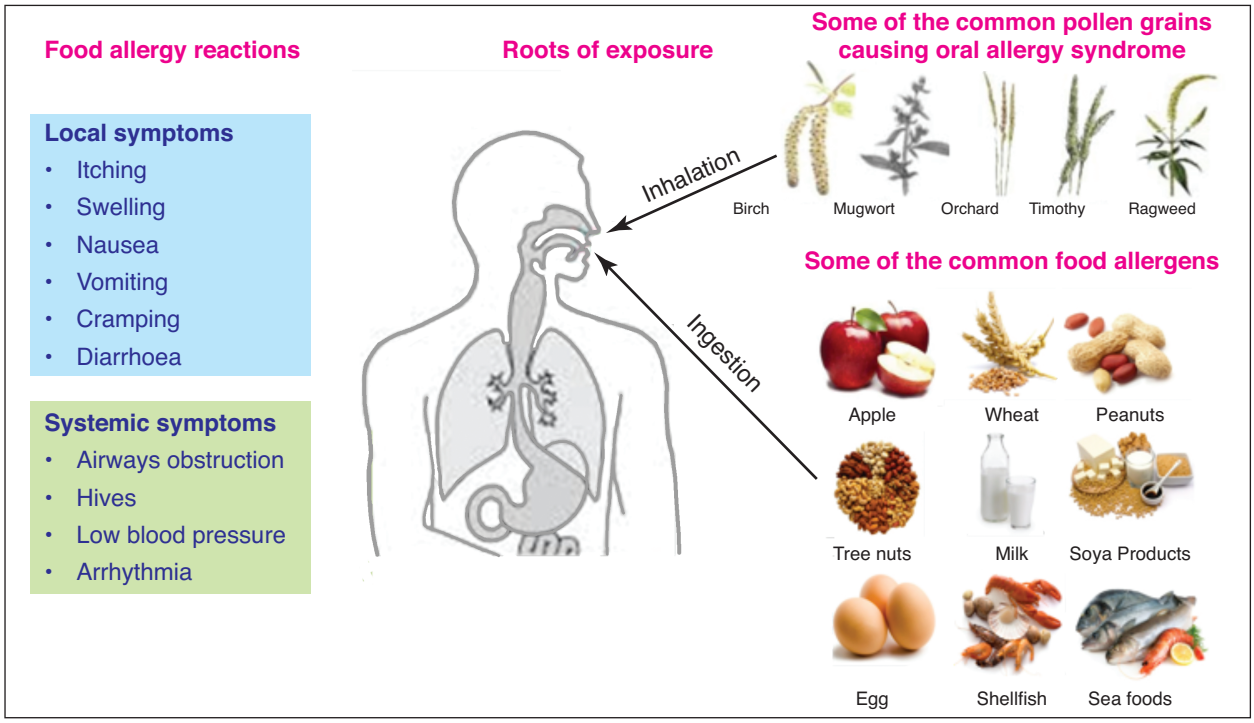

Fig. 1: Food allergens enter into the body either by ingestion or inhalation causing the appearance of local and systemic symptoms

PREVALENCE

In the world:

Prevalence of food allergy has doubled over the last decade in the United Kingdom[21,22] and the United States of America[23,24]. The most commonly reported food allergens in the European and North American populations are cow's milk, egg, peanuts, tree nuts, wheat, crustacean shellfish, fish and soy[25]. Peanut and tree nut allergy are unique in the western world and are the major cause of anaphylaxis; whereas nut allergy is extremely lower in Asian countries. Only 0.67% in Korean infants[26], 0.47 to 0.64% in Singaporean children, 0.43% in Filipino children[26] and 0.5 to 1.1% children in Taiwan[27,28,29] suffer from nut allergy. There are almost no cases of nut allergy found in China[30] and Thailand[31]. These rates are at least half of those reported in the United Kingdom (1.2 - 3.3%)[21], Canada (0.26 - 1.0%)[32], USA (0.6 - 2.7%)[5,24] and Australia (3%)[33]. Studies suggest that food-induced anaphylaxis is increasing in the industrialised world specifically in the United States, United Kingdom and Australia[33]. Few questionnaire-based surveys in Europe and USA suggest the prevalence of food allergy in 3-35% children of below16 years[34,35]. Gupta *et. al.*[36] used an electronic US household survey (n = 38,480) in 2009-2010 and estimated that 8% of children have a food allergy, 2.4% have multiple food allergies, and approximately 3% experience severe reactions. A study conducted in an urban paediatric emergency department of New York[37] suggests that cow's milk is the most common trigger of anaphylaxis in children of below 6 years of age. Shellfish allergy occurs mainly in children of 7-18 years of age. According to Rona *et. al.*[38], 2.2% of children of below 4 years suffer from cow's milk allergy in the Netherlands. In the USA, 1.8% of children of 1-5 years suffer from the same[36]. A study conducted by Peter *et. al.*[39] in Melbourne, Australia on 5276 12-month old infants showing egg and peanut as a major allergen. In Brazil, 35% of cases of anaphylaxis occur due food allergy[40].

In Asia:

Asia is the most populous region in the world having citizens with varying culture, food habit, life style and socio-economic condition. However, little is known about the epidemiology of food allergy among Asian countries. Shellfish is the most common food allergen in Asia[29]. Egg and cow's milk allergy is most prevalent among children and infants of this region[29].

In comparison to western countries, peanut allergy is extremely lower in Asia[29]. A questionnaire-based survey in Taiwan depicts the prevalence of food allergy is 7.02% among children of 4-18 years[28]. A study on 3677 Chinese pre-school students of 2-7 years of age suggests that shellfish (15.8%), egg (9.1%), peanut (8.1%), beef (6.4%), cow's milk (5.7%) and tree nut (5%) are the six leading cause of food allergy in this area[41]. In China, the prevalence of food allergy rises from 3.5% in 1999 to 7.7% in 2009[42]. Wheat allergy is the most common cause of anaphylaxis in Japan and Korea, while it is uncommon in other Asian countries[29]. Shellfish is the leading cause of food-induced anaphylaxis in adults living in South-East Asia. In the Philippines and Singapore 5.12% and 5.23% of people suffer from Shellfish allergy[43]; whereas only 0.7% of the US population suffers from the same[43]]. Prawn and Shrimp allergy is also common among South Eastern Asian population. Tropomyosin is a major allergen from prawn, shrimp and shellfishes and it is a pan allergen[44]. Tropomyosin is found in house dust mite also. So it is thought that house dust mite tropomyosin may be a primary sensitizer for shellfish or prawn allergy as house dust mites are very abundant in our environment[28]. Studies have been carried out from time to time in China, Thailand, Philippines, Taiwan, Indonesia, Malaysia[45], Singapore, Korea[46], Saudi Arabia[47] and India[1] which portrays Shellfish, egg, peanut, beef, cow's milk, tree nuts, soy, wheat, fish, rice, sesame, pork, seafood and banana as major food allergen in this area. There are some food allergies which are unique to specific regions of Asia, such as Bird's nest soup is a common cause of anaphylaxis in Singapore[14]; in India legumes, particularly chickpea, and brinjal are major allergy-causing food items, due to high consumption of these vegetables by the Indian population[48].

In India:

Indian population has a varied and distinctive dietary habit. Different types of vegetables constitute the major side dish for most of the Indians. Hence vegetables, such as, brinjal, lady's finger, tomato, cauliflower etc. and pulses, such as, chickpea, soybean, lentil etc. are main cause of food allergy in this area, which is uncommon

in other parts of Asia1. Studies have been carried out in different parts of India, viz., Delhi[48], Bangalore, Mysore[49], Bikaner[50], Kolkata[1] etc. depicting the prevalence of food allergy among the Indian population. Kumari *et. al.* report cases of black gram sensitization among Delhi patients[48]. They identified 8 IgE reactive proteins of 78, 56, 47, 43, 40, 30, 28 and 16 kDa from raw black gram extract[48]. Hegde *et. al.* report a case of mango allergy from Mysore[51]. Cases of mango allergy have also been reported from Kolkata. According to recent Kolkata based study, the top five allergenic food items in this area are banana, brinjal, egg, lady's finger and wheat[1]. Egg and wheat are well-investigated food items causing allergy. Kolkata based study portrays that allergy in banana, egg, milk, soybean, cucumber, chickpea and grapes are prevalent among the children (age below 15 yrs), whereas, allergy in cereals such as wheat and rice is highest among elderly population (age above 40 yrs.). At the same time, an almost equal number of patient distribution is found in case of papaya, potato and coconut dry allergy in different age groups[1]. Sarkar *et. al.* (2018) reported a novel polygalacturonase allergen Cari p 1 from papaya pollen and fruit showing the prevalence of sensitization among Indian population[20].

PATHOGENESIS

Gut barrier:

Gut barrier or gastrointestinal mucosal barrier is a complex structure. It consists of several physical barriers (Epithelial cells, glycocalyx, mucins, tight junctions, salivary amylases, gastric acid, pepsins, lysozyme etc.), as well as, immunologic mechanisms (antigen-specific s-IgA in the gut lumen, serum antigen-specific IgA and IgG etc.), which prevent penetration of foreign antigens[15]. Moreover, mucosal immune system encounters a relatively greater amount of antigens daily concerning the systemic immune system and generally suppress immune reactivity to harmless foreign proteins (e.g. , food proteins), generating tolerance[6]. However, in the case of infants, the immature immune system reduces the efficiency of mucosal barriers. Therefore, the introduction of numerous food antigens stimulates excessive production of IgE resulting in an adverse immune response, especially in genetically pre-disposed infants[15]. Fergusson *et. al.* conducted a prospective study over 1200 unselected infants and showed a direct linear relationship between the number of solid foods introduced into the diet by 4 months of age and the subsequent development of atopic dermatitis, with a 3-fold increase in recurrent eczema at 10 years of age in infants who had received more than 3 solid foods before 4 months of age[52]. Although it is more common in infants, cellular and IgE-mediated hypersensitivity to food antigens can develop at any age[15]. It is reported that neutralizing stomach pH can increase the risk of food allergy[53]. However, only a small amount of food antigens (2%) can invade through the gut barrier and transport all over the body in immunologically intact form[54].

Oral tolerance induction

Oral tolerance induction is the process using which the immune system (local and systemic) becomes educated to avoid sensitization to ingested food antigens that means it is a state of immune unresponsiveness to foreign proteins[55]. Several works have been carried out time to time to know the underlying mechanism of oral tolerance induction. Different mechanisms associated with oral tolerance include active regulation by T_{reg}, clonal deletion and clonal anergy (anergy means lack of reaction of the body's immune system to foreign substances) of T cells. Studies suggested that T regulatory cells (T_{reg}) play a central role in oral tolerance, which involves stepwise and anatomically defined processes of induction, maintenance, and migration of T_{reg}[55]. According to Siewert et al. Foxp3 + Treg are present in highest frequency and confer oral tolerance when a high dose of antigen is administered[56]. CD103 + DCs (Dendritic cells) and the mLNs (mesenteric lymph nodes) of the intestinal immune system, seem to have special abilities to drive the generation of Treg. Pabst *et. al.* (2012) proposed a multi-step process of oral tolerance to soluble antigens. According to the proposed model, antigen-loaded CD103 + dendritic cells (DCs) migrates from the lamina propria (LP) to the mesenteric lymph nodes (mLNs), initiating oral tolerance. Lamina propria (LP) constitute the main effector sites of the intestinal immune system, harbouring large populations of activated T cells and antibody-secreting plasma cells; whereas mesenteric lymph nodes (mLNs) compose inductive site of the intestinal immune system. In the mLN, antigen-specific T cells express two gut-homing receptors $\alpha_4\beta_7$ integrin and CCR9 as induced by

retinoic acid (RA). This favours TGF-β-dependent differentiation of Foxp3 + induced regulatory T cells. T_{reg} comes back to the intestinal lamina propria and undergoes secondary expansion under the influence of interleukin-10 (IL-10). In the last step, some of the Treg may exit from the mucosa via lymph and bloodstream, disseminating throughout the immune system, and promoting the systemic consequences of oral tolerance[55].

Gene-environment interaction

Food allergy is a complex polygenic disorder influenced by gene-environment interaction[57]. In the previous section, the role of antigen-presenting cells, T cells, humoral immune responses, homing receptors, signalling pathways, dietary factors and other aspects of the immune response to dietary antigens has been reviewed. However, explaining gene-environment interactions is crucial for the understanding of immuno-pathogenesis of food allergy. Family history is a strong risk factor for the development of food allergy. Dey *et. al.* (2014) reported that 63.18% of food allergic patients of Kolkata showed a family history of food allergy[1]. According to Sicherer *et. al.* heritability of peanut allergy was 81.6% [58]. Liu *et. al.*[59] stated IL4 polymorphism as one of the major risk factors for the development of sensitization towards different food allergens. Several candidate genes associated with food allergy had been reported by various workers time to time, these are major histocompatibility complex (HLA) gene family[60], CD 14 gene[61], FOXP3[62], STAT6[63], SPINK5[64], IL10[65] and IL 13 gene[66]. Currently, several works on the effect of gene-environment interaction on allergic disease have been going on. Hong *et. al.*[67] reviewed the effect of C-159T polymorphism on the CD14 gene, and it seemed to depend on environmental sources of microbial simulation. However, there are several things unexplored in this area, and it is a promising field of research.

CLINICAL DISORDER

Gastro-intestinal food hypersensitivity reaction

Gastro-intestinal (GI) tract is the first to confront several food allergens. IgE mediated food hypersensitivity reactions include immediate GI hypersensitivity and oral allergy syndrome[15]. Symptoms caused by immediate GI hypersensitivity typically develop within minutes to 2 hours of consuming the responsible food allergen and consist of nausea, abdominal pain, colic, vomiting, and diarrhea[15]. Oral allergy syndrome (OAS) affects up to 40% of adults with pollen allergy, especially to birch, ragweed, and mugwort pollens[15]. OAS is a form of contact allergy that affects oropharynx. Mixed IgE and non-IgE mediated food hypersensitivity reactions include eosinophilic esophagitis, eosinophilic gastritis and eosinophilic gastroenteritis. As reviewed by Sampson *et. al.* (1999), allergic eosinophilic esophagitis is most frequent during infancy through adolescence and presents with chronic reflux (gastroesophageal reflux), intermittent emesis, food refusal, abdominal pain, dysphagia, irritability, sleep disturbance, and failure to respond to conventional reflux medication. Allergic eosinophilic gastritis also is more common between infancy and adolescence, and first symptoms appear as postprandial vomiting, abdominal pain, anorexia, early satiety, hematemesis, failure to thrive, and gastric outlet obstruction[68]. Allergic eosinophilic gastroenteritis may occur at any age and may appear with symptoms similar to esophagitis, gastritis, or both. Non-IgE mediated food hypersensitivity reactions include dietary-protein enterocolitis, dietary-protein proctitis and dietary-protein enteropathy. Signs of non-IgE mediated food allergies mostly appear within first few months of life. In dietary-protein enterocolitis infants encounter several symptoms like irritability, protracted vomiting, diarrhoea etc.[69], whereas dietary protein proctitis appears as blood-streaked stools. About 60% of dietary protein proctitis were seen in breast-fed babies[70-72], indicating heritability of the disease. Protracted diarrhoea, vomiting, failure to thrive, abdominal distention, early satiety, and malabsorption are the symptoms of dietary-protein enteropathy. Celiac disease is a type of dietary protein enteropathy characterized by a more extensive loss of absorptive villi and hyperplasia of the crypts, leading to malabsorption, chronic diarrhoea, steatorrhea, abdominal distention, flatulence, and weight loss or failure to thrive[15].

Cutaneous food hypersensitivity reaction

Cutaneous food hypersensitivity reactions include acute and chronic urticaria and angioedema occurred by IgE mediated response; atopic dermatitis took place by mixed IgE- non IgE mediated pathway and dermatitis herpetiformis occurred by non IgE mediated pathway[15]. Pruritus, hives and swelling are the symptoms of urti-

caria and angioedema. Acute urticaria and angioedema are the commonest sign of food allergy, whereas chronic urticaria and angioedema are infrequent. Marked pruritus and eczematous rash are the characteristic symptoms of atopic dermatitis, which mostly develops at early ages of life. Dermatitis herpetiformis is a chronic blistering disorder, characterized by a papulovesicular rash over exterior surfaces[6].

Respiratory food hypersensitivity reaction

Allergic rhinoconjunctivitis, asthma and pulmonary hemosiderosis, occurred by IgE mediated, mixed IgE-non-IgE mediated and non-IgE mediated pathways respectively encompass respiratory food hypersensitivity. Periocular pruritus, tearing, sneezing, rhinorrhea, conjunctival erythema, nasal congestion are symptoms of allergic rhinoconjunctivitis. Asthma is an uncommon manifestation of food allergy. Food-induced pulmonary hemosiderosis or Heiner's Syndrome is a very rare syndrome characterized by recurrent episodes of pneumonia associated with pulmonary infiltrates, hemosiderosis, gastrointestinal blood loss, iron deficiency anaemia, and failure to thrive in infants and young children[73]. It is most often associated with hypersensitivity to cow's milk[74].

Anaphylaxis

Food allergy is the most common cause of anaphylaxis. According to FARE (Food Allergy Research and Education) reports, the foods most likely to cause a severe reaction are peanuts, tree nuts, fish and shellfish (*http://www.foodallergy.org*). People who have both asthma and a food allergy are at greater risk for anaphylaxis. Symptoms of anaphylaxis appear within a few minutes of ingesting allergic food item and may be fatal if not treated immediately. Symptoms of anaphylaxis include difficulty in breathing, reduced blood pressure, vomiting, diarrhoea, cramping, loss of consciousness etc. In the case of 25% of patients, the second wave of symptoms appears after one to several hours of first symptom appearance (*http://www.foodallergy.org*).

DIAGNOSIS

The first step in food allergy diagnosis is going through thorough medical history. Next, the allergologist may conduct tests to help to identify a food allergy. Diagnostic tests for food allergy include Skin Prick Test (SPT), blood test (ELISA, RAST), oral food challenge and trial elimination diet (*http://www.foodallergy.org*). SPT and specific IgE ELISA or RAST tests are useful to detect IgE mediated food allergies. Many investigators use atopy patch test in addition to SPT to detect non IgE mediated food allergy, primarily in patients with atopic dermatitis and allergic eosinophilic esophagitis. Endoscopy and biopsy are the most definitive approach for diagnosing gastro-intestinal food hypersensitivity[15]. A double-blind placebo-controlled oral food challenge is a highly accurate method for diagnosing food allergy. To help to diagnose a food allergy, sometimes specific food items are eliminated from the patient's diet. This method, combined with skin or blood tests, can help diagnose both IgE-mediated food allergies and related disorders, such as allergies that affect the gut.

THERAPY

Early management of food allergy includes avoidance of allergy-provoking food items. Administering epinephrine injection is useful to treat life-threatening anaphylaxis (*http://www.foodallergy.org*). Clinical tolerance develops in most food allergens over time, except peanut, nut and seafood. Antihistamines relieve symptoms of oral allergy syndrome and IgE mediated skin problems, but do not block systemic reactions[15]. Systemic corticosteroids are useful in treating chronic IgE, and non IgE mediated disorders. Some researchers reported the success of applying fluticasone on patients of allergic eosinophilic esophagitis[73]. Some novel immuno-therapy methods are developed for treating IgE mediated food allergies. Recent advances in technology have enabled researchers to map allergenic epitopes of a particular protein. Mutation of these epitopes helps to prepare hypoallergenic proteins which are useful in immunotherapy. Shin *et. al.* (1998) altered immunodominant epitopes of 3 peanut allergens Ara h1 to Ara h3 by single amino acid substitution, which leads to reduced IgE binding to individual epitopes[75]. Other immuno-modulatory approach under investigation includes the use of immuno-stimulatory sequence (CpG motifs) which can reverse IgE mediated sensitization in patients[76].

FUTURE RESEARCH

Nowadays several advances have been made in the field of immunotherapy (such as developing allergen-specific immunotherapy). Patients who are unresponsive to anti-

histamines and corticosteroids, immunotherapy is the only mean for them to combat allergy. Preparation of hypo-allergenic vaccine has gained much interest in recent days which may be the most appropriate treatment approach for food allergy. Multi-epitope vaccines were introduced for several viral diseases. However, no such effort has been made to combat with food allergy in poly-sensitized patients. These may open up new avenues in allergy research.

Besides these, over the past decade biomarker discovery has gained much attention due to its promising role in drug development and leading part towards personalised medicine approach. Biomarkers are naturally occurring molecules or genes, which are the indicator of a particular state (normal biological processes, pathogenic processes, or pharmacologic responses) of a cell at a particular time. Tests for the presence of several biomarkers in the body fluid have been employed in cancer research which gives valuable information about susceptibility and disease state of an individual. However, no such diagnostic methods are available for allergy prognosis. We also lack quantitative biomarkers reflecting the clinical efficacy of allergen-specific immunotherapy. Efforts have been made in different parts of the world including India for searching allergy biomarkers.

CONCLUSION

The prevalence of food allergy is rising worldwide for unclear reasons. Change in life style, modernization and environmental changes may be responsible for this. Complex gene-environment interactions seem to have resulted in a loss of oral/gastrointestinal tolerance leading to several food allergy symptoms starting from vomiting, diarrhoea, abdominal pain, sneezing, rhinorrhea to fatal anaphylaxis. Besides administering antihistamines and corticosteroids, immunotherapy is the only treatment available for food allergy. In recent years, although several advances have been made in the research field, more research is needed to improve food allergy diagnosis, treatment, and prevention.

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CIRCADIAN ATMOSPHERIC POLLEN INCIDENCE OF HYPTIS SUAVEOLENS (L) POIT

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Pollen grains of Lamiaceae were reported as allergenic from different parts of the world. Present paper deals with the hourly pollen release of *Hyptis suaveolens* (L.) Poit. a common allergenically potent weed is belonging to the family Lamiaceae. It blooms from September to December and flowers generally open at 06:30-08:30 hrs. Each flower produces 2430 ± 25 pollen grains and nectar, which are the chief floral rewards to the flower visitors. After flower opening different insects like *Apis cerana indica, A. Dorsata, Amegilla* sp., *Ceratina* sp. *Megachile* sp., of Hymenoptera; *Episyrphus* sp. of Diptera; *Borbo cinnara, Catopsilia pyranthe, Pachliopta aristolochiae and Papilio demoleus* of Lepidoptera were found to visit flowers for their forage. During forage, flower visitors help in pollen dispersal as well as pollination. The maximum 17.2% of pollen grains were trapped at 09:30 hrs. on sunny weather (temperature: 34.5ºC and R. H. 80%) using 'Rotorod' sampler from the ambient air. During cloudy weather (temperature: 27ºC and R.H. 93%) the frequency of insect visitation as well as atmospheric pollen incidence decreased as a result of 12.8% atmospheric pollen incidence at 10:30 hrs. was recorded. The atmospheric pollen incidence was low at the morning, and frequency gradually increased with the flower opening, anther dehiscence and flower-visitors activity, while the atmospheric pollen frequency gradually decreased at the evening when the pollen grains were either consumed or precipitated.

Key Words: Flower-visitor activity, Pollen incidence, 'Rotorod' sampler, *Hyptis suaveolens* (L.) Poit.

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INTRODUCTION

The study of floral phenology, pollen dispersal and atmospheric pollen frequency provide a basic idea in the various fields of clinical, aeropalynological and pollination research. Pollen grains which are the male genetic material, play a vital role in plant sexual reproduction and also a key factor to provoke different allergic manifestation in human as well as animals. So, the knowledge of airborne bio-particles, particularly the allergic pollen grains are very much essential, and the awareness about the circadian rhythmicity of pollen release for susceptible individuals as well as clinicians are required. The anemophilous plants contribute the major pollen grains in the air. However, the pollen grains of different entomophilous plants also contribute a good amount of pollen grains in the atmosphere[1-7]. Floral phenology, anthesis, pollen release and flower-visitors activity are key factors to understand the atmospheric pollen release. The study of the aeropalynological process was based on the five key steps like source, release, dispersal, deposition and impact which are influenced by the environmental factors[8]. The pollen grains of *Hyptis suaveolens* (L.) Poit. having allergenic potential were recorded from atmosphere[9-11] and the pollination mechanism of the plant has been studied[12-14] but, the details mechanism of its dispersal and atmospheric pollen incidence is very important for further monitoring. So, the present study reveals the hourly atmospheric pollen incidence concerning the flower-visitors interaction and meteorological parameters.

MATERIALS AND METHODS

The study was conducted following the standard methods[15-18] to observe different phenological events during September to December with plants growing in and around our University campus at Visva-Bharati, Santiniketan (87°42´ E and 23°42´ N). Pollen grains per anther and per flower were quantified[1]. The behaviour of the flower visitors 16 was observed and the atmospheric pollen incidence at different time intervals was recorded by 'Rotorod sampler'[19]. The frequency of trapped pollen was calculated[18, 20].

RESULTS AND DISCUSSION:

Hyptis suaveolens, an annual herb produces bisexual, zygomorphic, odoriferous flowers from September to

December and flowers were opened at 06.30–08.30hrs. After flower opening anthers dehisced explosively by longitudinal slits. A single flower produces 2430 ± 25, hexacolpate and powdery pollen grains (Table-1). The flower produces 3 μl nectar which along with the pollen grains are the chief floral rewards for the flower visitors. As soon as flowers start to open, different members of Hymenoptera (*Apis cerana indica, A. dorsata, Amegilla* sp., *Ceratina* sp., *Megachile* sp.); Diptera (*Episyrphus* sp.) and Lepidoptera (*Borbo cinnara, Catopsilia pyranthe, Pachliopta aristolochiae, Papilio demoleus*) were found to visit the flowers for their forage (Table-2; Fig. 2). Among the flower-visitors, bees were the most dominant. During the visit, their bodies get dusted with a considerable amount of pollen grains which help in pollen dispersal and pollination. At the time of unfolding of corolla lobes, the stamens were remained coiled, though it remained under tension and subjected to the external force. The pollen release occurs explosively. This process was triggered when insects visit to collect nectar from the flowers. There are some reports on entomophilous plants where insects facilitate in dislodging of pollen grains in ambient air[2, 5, 7, 21, 22]. Though flowers were visited by different insects, a considerable amount of pollen grains were recorded from ambient air. Powdery pollen grains from the exposed anthers come into the air easily. The amount of pollen grains in the atmosphere depends on relative humidity and temperature of the ambient air. On a sunny day (34.5ºC and 80% R. H.) circadian rhythmicity of pollen grains started with low (5%) at the early morning (05:30 hrs.) when the flowers were unopened, and gradually the atmospheric pollen frequency increased after flower opening and reached at maximum 17.2% at 09:30 hrs (Table-3) when temperature was little bit higher along with higher frequency of insect visitation (Table-3; Fig. 1). Then circadian rhythmicity was gradually decreased from afternoon to evening when the pollen grains were either consumed or shed off but, a considerable amount of pollen grains were always found in the atmosphere during the peak flowering period. So, the atmospheric pollen frequency was influenced by the insect activities along with the nature of anther dehiscence which takes place explosively[13]. During the visit, insects disturbed the floral parts that result in liberation of pollen grains to float into the ambient air. Besides, some amount of pollen grains were also dislodged from their body parts during their movements. Actually atmospheric temperature is very much important to desiccate the anther tissues which are very essential for pollen liberation[23]

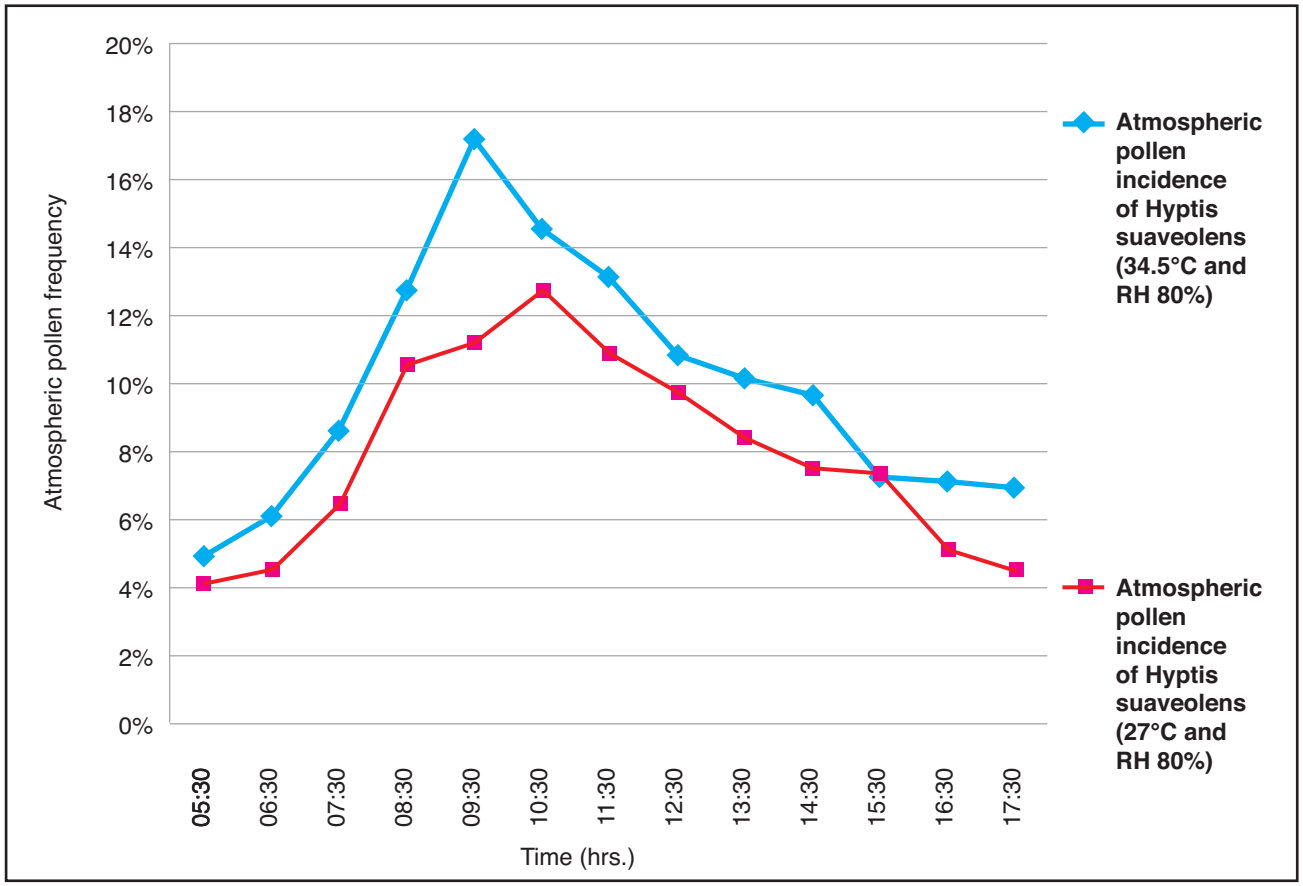

Fig. 1: Atmospheric pollen incidence of Hyptis suaveolens

and with the increase of atmospheric temperature the pollen grains which were attached to the anthers and floral parts, gradually dried up and increased the buoyancy of the pollen grains, which were suitable for dispersal in the ambient air. It was observed that the *Amegila* sp., *Apis dorsata* and *Apis cerana indica* were most dominant visitors. Circadian rhythmicity of atmospheric pollen deposition was recorded earlier in Poaceae[17] and *Alangium salviifolium*[24]. The effect of atmospheric temperature, relative humidity and flower-visitor interactions on circadian atmospheric pollen incidence of *Alagium salviifolium* was recorded[24]. On a cloudy day (27° C and 93% R. H.) maximum 12.8% atmospheric pollen frequency was recorded at 10.30 hrs because of relative humidity, and temperature decreases the buoyancy of pollen grains (Table-4; Fig. 1). The daily course and intensity of pollen release also depend on the meteorological variables[17]. Present observation suggests that though the taxon is entomophilous[13, 14] but a considerable amount of pollen grains were always present in the atmosphere during peak flowering period which may cause allergic disorder to susceptible persons.

Table 1: Floral characters of Hyptis suaveolens

Floral Characters	Observation
Flowering period	September-December
Flower type	Bisexual, Zygomorphic
Odour	Present
Nectar	Present
Flower opening time	06.30 hrs to 08.30 hrs
Mode of anther dehiscence	Longitudinal & Explosive
Pollen production/Flower	2430 ± 25
Pollen aperture	6-colpate

Table 2: Flower-visitors of Hyptis suaveolens

Floral visitors	Peak Foraging period	Mean abundance (%)	Forage type
Amegilla sp.	08:30-10:30	35	Pollen, nectar
Apis cerana indica	08:30-10:30	10	Pollen, nectar
Apis dorsata	08:30-10:30	15	Pollen, nectar
Borbo cinnara	09:30-11:30	5	nectar
Catopsilia pyranthe	09:30-11:30	7	nectar
Cearatina sp.	09:00-11:30	4	Pollen, nectar
Episyrphus sp.	09:30-11:30	2	Pollen, nectar
Megachile sp.	09:30-11:30	8	Pollen, nectar
Pachliopta aristolochiae	09:30-11:30	3	nectar
Papilio demoleus	09:30-11:30	11	nectar

Table 3: Atmospheric pollen incidence of Hyptis suaveolens (Temperature: 34.5°C and R.H. 80%)

Time (hrs.)	05.30	06.30	07.30	08.30	09.30	10.30	11.30	12.30	13.30	14.30	15.30	16.30	17.30
Atmospheric pollen frequency (%)	5.0	6.2	8.7	12.8	17.2	14.6	13.2	10.9	10.2	9.8	7.3	7.2	7.0

Table 4: Atmospheric pollen incidence of Hyptis suaveolens (Temperature: 27°C and R.H. 93%)

Time (hrs.)	05.30	06.30	07.30	08.30	09.30	10.30	11.30	12.30	13.30	14.30	15.30	16.30	17.30
Atmospheric pollen frequency (%)	4.2	4.6	6.6	10.6	11.3	12.8	10.9	9.8	8.5	7.6	7.4	5.2	4.6

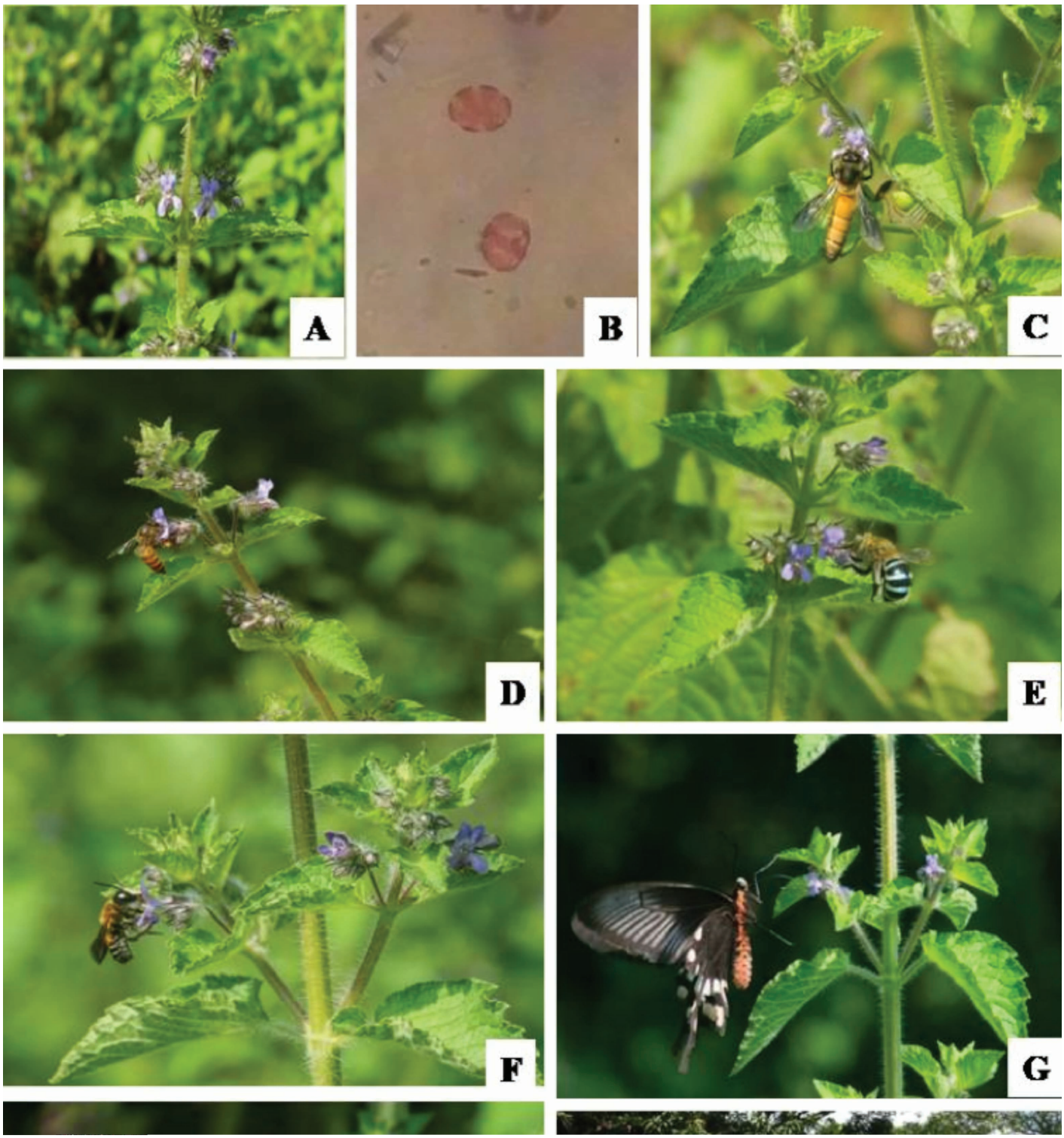

Fig. 2: A. Flowering plant; B. Pollen grains; C. ; D. ; E. sp.; F. sp. ; G. ; H. ; I. Rotorod sampler.

CONCLUSION

The ambient air quality in most of the time exceeds the permissible limit for PM_{10} as well as $PM_{2.5}$. The highest levels of SO_2, NO_2 and NH_3 were mostly accounted in the mid or end of February 2019. In comparison with primary data, the reference data that was also collected from the official website of WBPCB shows that the PM_{10} values exceeding the permissible limit and in some of the cases it is approximately double the standard value. Gariahat crossing and Ballygunge Circular Road are the busiest roads of Kolkata and the vehicular emission is the main contributor of this pollution. Roadside vendors are easy targets of vehicular as well as other sources of air pollution because of their profession. They spent most of the times beside the roads which increase the probability of risk. Spirometry based data suggests that the estimated lung age is quite higher than their biological age which predicts an alarming situation. According to the severity index, several individuals come under the very severe category which increases their health hazards. .From the questionnaire survey, it is found that 80% population is unaware of the air pollution. It can be concluded that along with mass awareness, individual-level protection is very much important or else the percentage of affected individuals will increase. In Kolkata, along with the other measurements, the number of vehicles should be checked by the authority. In future, a large scale detailed survey along with spirometry test is needed on the roadside population of Kolkata, so that this data can be utilized by medical experts and researchers to know and improve their health and lung condition through proper treatment. Also, measures should be taken to improve the scenario of air pollution and also stringent the environmental laws related to pollution.

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SELECTED ABSTRACTS OF INDIAN SCIENTISTS

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IN PARMA, ITALY

AEROBIOLOGY, EPIDEMIOLOGY AND DISEASE FORECASTING OF FALSE SMUT DISEASE OF RICE IN WEST BENGAL, INDIA

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Back ground: In India, false smut disease of rice has been observed in severe form since 2001 in major rice growing states. This disease is difficult to tackle, because pathogens infect the plant during flowering stage and the symptoms only visible after emergence of panicle. Thus disease forecasting is necessary to predict the chance of disease in a certain set of conditions.

Methods: We analysed three years (2013-2015) aerobiological data including pathogenic spore (*Ustilaginoidea virens*) concentration over the rice canopy in West Bengal, India. The crop phases in both rabi and kharif seasons during which rapid development of disease takes place were also recorded. Multiple regression analysis of PDI values was done using age of the plant, pathogen concentration and meteorological factors.

Results: In rabi season, the false smut disease severity depends on age of the plant as well as pathogenic spore concentration, while in kharif season the severity depends upon spore concentration, RH and minT. From all the analysed data it can be predicted that spore is the common factor for false smut disease incidence and severity in rice. If *Ustilaginoidea virens* spore concentration is increased by one unit, there will be 0.981 unit increased in the expected value of disease severity. With odds ratio value, it can be assumed that with 1 unit change in spore concentration, the odds of "Severe" versus "High" or "Moderate" or "Low" or "Absent" combined will be 1.1031 times greater, when all other variables held constant .

Conclusion: The regression model made from the present study would be beneficial to the farmers for early prediction of false smut disease of rice in West Bengsl, India. Model proposed in the present study needs to be validated.

Key words: Aerobiology, false smut disease, rice. epidemiology, forecasting, West Bengal, India.

MODERN POLLEN SPECTRA FROM TROPICAL WEST BENGAL, INDIA – ALTITUDINAL GRADIENTS

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Background: Tropical forests of India have received very little attention in modern pollen studies. The objective of the present study is to describe the modern pollen rains in dry deciduous forests of West Bengal, India.

Methods: We collected and analyzed the pollen content of 46 surface soil samples from dry deciduous foerst of West Bengal (between 22°00” to 24°50” N, and 87°00” to 88°00” E). We also made vegetation relevés to relate the pollen rain with the vegetation composition at the sampling sites. Dendrograms were constructed following Multivariate analysis using STATISTICA 6.0. We used Canonical Correspondence Analysis (CCA) by CANOCO 4.5 to get a comprehensive picture regarding the distribution of the taxa with regard to altitudinal gradient.

Results: A total of 62 pollen types were retrieved from soil samples showing dominance of arboreal pollen over non-arboreal types. Among the arboreals, *Shorea robusta* is the most dominating element, followed by *Pavetta, Buchanania, Madhuca,* etc. The arboreal taxa showed comparatively lower frequencies as regards to their actual representation in the forest vegetation, though they represent a more or less identical forest floristics. Dendograms were constructed for both site data and pollen taxa. Cluster analysis of pollen taxa shows five distinct clusters which reflect correlation and co-existence of different plant taxa. It has clearly been shown that altitudinal gradient is well expressed by the modern pollen rain. The dispersion of the sites indicates a clear distinction between high and low elevation taxa.

Conclusion: The study shows distinct pollen assemblages which can be used as markers of tropical dry deciduous forest vegetation. Altitudinal gradients are well expressed by the modern pollen rain and are found to be valuable criteria to distinguish high and low elevation forests. This justifies further use of pollen data in biome modeling.

Key words: Modern pollen spectra, tropical forests, deciduous, cluster analysis, CCA.

MOLECULAR AND ANTIGENIC ANALYSIS OF AN AEROALLERGEN RHI O 1 FOR MOLECULAR DIAGNOSIS AND IMMUNOTHERAPY OF FUNGAL ALLERGY

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Introduction: Rhizopus oryzae is a predominant indoor mold in India that causes IgE-mediated sensitization among the atopic individuals.

Methods: IgE-serology and mass spectrometry based proteomic analyses was performed to detect the major allergen of R. oryzae. The native allergen was purified by chromatography and the followed by Edman sequencing. The full length cDNA was PCR amplified, and cloned in bacterial vector and recombinant allergen was affinity purified. Immunobiochemical properties were studied by CD spectroscopy, enzyme assay, ELISA-inhibition and degranulation assay. The IgE-epitopes were mapped by peptide. Critical residues of IgE-epitope were identified by alanine substitution. The reduction in allergenic activity of the point mutants was studied by IgE-binding assay, basophil activation test and interleukin assay. The vaccination potential of the hypoallergen was studied by IgE-inhibition and histamine release suppression activity of anti-mutant IgG.

Results: The major allergen, designated as Rhi o 1 by IUIS, was identified as a non-cytosolic aspartic protease. Recombinant Rhi o 1 displayed >30% IgE-reactivity in fungal allergy patients and induced >50% histamine release from challenged granulocytes. rRhi o 1 exhibited cross-reactivity with cockroach allergen Bla g 2 in the mAb4C3 recognizable epitope. In addition, two unique IgE-epitopes (^{44}T-N^{60} and ^{311}G-K^{327}) were mapped as a spatially clustered antigenic determinant on Rhi o 1 surface. The T49/Y52/K314/W316 mutant showed 100-fold lower IgE

binding and reduced allergenic activity. The TYKW mutant retained T-cell epitopes, as evident from its lympho-proliferative capacity but down-regulated IL-5 secretion. The mutant induced enhanced focusing of blocking IgG antibodies that competitively inhibited binding of IgE antibodies to Rhi o 1 up to 70% and suppressed allergen-mediated histamine release by 10-fold.

Conclusion: Rhi o 1 is a marker allergen for molecular diagnosis and the genetically engineered hypoallergen is a candidate molecule for immunotherapeutic applications.

Key words: *Rhizopus oryzae*, Rhi o 1, aeroallergens, molecular characterization, immunotherapy.

IMPACT OF AERO-ALLERGENS ON HUMAN HEALTH AND MINING NOVEL ALLERGENS FROM MORINGA OLEIFERA POLLEN GRAINS

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Objective: Pollen grains are major cause of inhalative allergy. Around 20-30% of world population suffers from pollen allergy, with India being not an exception. The aim of the present study is to measure aero-pollen load of Kolkata megacity of West Bengal, India, their impact on human health and immuno-proteomic studies on a selected pollen grain.

Methodology: A continuous air sampling was conducted at Rajarhat sampling station of Kolkata during the period of August 2012 to August 2014, using a seven-day Burkard automatic volumetric sampler to record concentration of air-borne pollen grains. Prevalence of pollen allergy was investigated among the patients admitted to nearest hospitals with pulmonary diseases using a questionnaire. As allergic outburst occur mainly at spring season, the most potent aero-allergen during this season was selected for immuno-proteomic studies. Its allergenic potential was investigated by SPT, ELISA, histamine release assay and immunoblotting (1D and 2D). Periodate acid Schiff staining was performed to identify the glycosidic nature of IgE reactive epitopes. Hierarchical cluster analysis was performed to identify the major allergen. The major allergen was partially purified by anion exchange chromatography.

Results: During spring season (January - March) *Moringa olifera* pollen grains were found to be most important aero-allergen in the study area. Around 23% patients of 18-50 years of age were found to suffer from allergy against *Moringa olifera* pollen grains. Blood was collected from 20 patients with +3 grade of positivity in SPT. In specific IgE ELISA 13 patients showed positive results with P/N ratio ≥2.5. In histamine release assay there was 15% - 30% increase in histamine release upon allergen challenge. 1D immunoblot using allergenic patient sera against *Moringa* pollen protein revealed the presence of 9 IgE reactive bands within m.w. range of 18-140 kDa, being 90kDa as the most prevalent which was a glycoprotein. In 2D immunoblot several IgE reactive spots of 90 kDa molecular weight, but varying pI value was found, which may be the isoforms of a single protein. This 90 kDa major allergen was further purified partially using anion exchange chromatography.

Conclusion: The present study reports the impact of allergy causing pollen grains on human health. Novelty of this report is the identification of a panel of allergens from *Moringa* pollen at immuno-proteomic level, which substantiated the clinical evidence of *Moringa* pollen allergy. Further studies of these allergens will improve the component resolved diagnosis of pollen allergy.

Key words: *Moringa olifera*, pollen, allergens, immune-proteomics, purified allergen.

0.11 - HLA DR-B1 TYPING STUDIES FROM SELECTED POPULATION OF DAVANGERE SUFFERING FROM ASPERGILLOSIS

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Objective: Respiratory disease is a broader concept which includes affecting airways or any of its structures and organs. According to the World Health Organization, chronic respiratory diseases affect hundreds of millions worldwide. However, lower respiratory infectins are one of the leading causes of death throughout the world. The recent data collected from India in 2017 records shos that 334,23,107 cases of respiratory diseases have been reported across the country, of which 19,863 patients died due to serious infections.

Methodology: The application of DNA-based typing methods for the analysis of class-II antigens has resulted in the continuous detection of new variants. A total of 120 patients were identified for predisposition test of which 60 patients who have allergy for more than 5 years were selected for HLA DR-B1 predisposition test. Peripheral blood samples were collected from selected patients at SSIMS & RC. Genotyping was performed by Luminex PCR-SSOP methodology at Lalpath labs according to the manufacturer's recommended procedure.

Results: 120 patients' nasal samples were recorded with fungi of which 38 patients with Aspergillus niger, 37 patients with Aspergillus fumigatus and 25 patients with Aspergillus flavus sp. have been recorded. Further 20 patients' nasal samples were recorded with Bacterial cultures, and these bacteria's were identified by using standard biochemical tests. Correlation studies of bacterial role in respiratory allergy needs detailed investigation.

Conclusion: Out of 60 patients subjected for predisposition test 33 patients showed predisposition test positive and 27 negative. 33 patients occupational air sampling studies were carried out in order to record type of bio-load present in the air. The sampled results shows maximum fungal spores with Aspergillus species recorded. In these patients HLA-DRB1 region was not expressing antigen binding proteins for immunological reaction and thus making them to suffer from Aspergillosis.

Key words: HLA-DRB1, Aspergillosis, Predisposition.

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