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A mathematical model for the prediction of the prevalence of allergies in Zimbabwe

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ABSTRACT

Background: The prevalence of allergies has been observed to be increasing in the past years in Zimbabwe. It is thus important to consider the long term prevalence of allergies. Our interest is in investigating the trends of allergies in the next 2 decades.

Method: We formulate a deterministic model with 6 compartments to predict the prevalence of allergies in Zimbabwe. The human population is divided into 4 distinct epidemiological, classes based on their exposure to 2 allergen groups (food and inhalants), represented by 2 compartments. The model is used to predict the prevalence of allergen sensitization. The number of human allergen groups in each compartment are tracked through a system of differential equations. Model parameters were obtained by fitting observed data to the model. Graphical solutions of the model were developed using Matlab and Excel.

Results: The rate of sensitisation to food allergen sources is found to be lower than the rate of sensitisation to inhalant allergens. The rate at which individuals develop tolerance to food allergen sources is found to be almost twice the rate of developing tolerance to inhalant allergies. The equilibrium solutions (the long-term states of the populations) of the model are found to be non-zero implying that there will never be an allergy-free population. Our results also show that the prevalence of food allergy is likely to increase in the next 2 decades while inhalant allergy prevalence is expected to decrease.

Conclusion: Our long-term solutions show endemicity in allergies in Zimbabwe. So, allergy will be endemic in the Zimbabwean population; hence there is a need for allergy care and management facilities to be increased. These results are critical in policy development and planning around allergies in the near future.

Keywords: Food allergy, Inhalant allergy, Mathematical model, Zimbabwe

INTRODUCTION

Type 1 allergic conditions are characterized by the expression of allergen specific IgE antibodies to triggering allergen sources that may be through inhalation or ingestion. The most frequent inhaled allergen sources locally are house dust mites,

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grass or tree pollen, and molds. The range of food allergen sources includes egg white, milk proteins, wheat, nuts, fruits, and fish. The food allergen sources to which an individual is exposed is influenced by dietary options that change as the population migrates from rural settings where traditional food items dominate to urban settings where the choices are greater and the variety ever increasing.[1] The flora and fauna of urban settings is dominated by ornamental trees, weeds, and lawn grass and is different from that obtaining in often parched rural settings.

Studies of allergic conditions have shown that these have erratic progression, are difficult to diagnose, and have potentially distressing consequences on the quality of life of affected individuals as well as an increasing health care burden of most countries worldwide.[2] Some descriptive studies that were conducted in Sub-Saharan countries including South Africa,[3] Nigeria,[4] Zimbabwe,[1] and Botswana,[5] all suggested that there is an increase in the prevalence of allergies.

In Zimbabwe, health priorities are, however, biased towards tropical infectious diseases. The relevance of allergic diseases in general and their impact on the quality of life is not prioritized in a country seized with major infectious diseases including HIV and AIDS, tuberculosis, and malaria. As a result, essential tiers of allergy care including human resource planning, training, and deployment for optimum mitigation, diagnosis, and disease management are overshadowed and lowly prioritized to the detriment of millions of affected individuals of all ages. Allergy data that are routinely captured by Ministries of Health are guided by World Health Organization (WHO) templates and tend to be limited to asthma. The prevalence, prevalence rates, periodicity, and seasonal fluctuations in numbers of patients with inhalant allergic diseases and the respective case fatality rates from these non-communicable allergic diseases are not systemically collected at the national level, and such tallying is not recommended by WHO. Resultantly relevant, informative data are not captured to the detriment of allergy patient care. We have observed substantial increases in the prevalence of allergy morbidity primarily in the private sector setting. Such data are not captured by the Ministry of Health; therefore, the extent to which non-communicable allergic diseases contribute to morbidity and mortality is not appreciated and opportunities to intervene are lost. The inclusion of the diagnosis and management of allergic diseases in a list of Ministry of Health priorities can only be informed by an understanding of the magnitude of the problem and its trajectory. We used data collected from the only allergy clinic in the country to attempt to project the trajectory of food and inhalant allergies over the next 20 years.

We have observed that numbers of sensitized individuals in the country are increasing. When a susceptible, usually atopic individual is exposed to allergen sources, sensitization can occur, and upon re-exposure, allergic symptoms can manifest. There is a large variety of allergen sources and a wide population of susceptible individuals, and the interplay between them is dynamic and variable. Additionally, as the environment continuously changes, so are the allergens available to interact with individuals which poses an increasing burden of allergy in a population. It is important to understand the rates at which susceptible individuals get sensitized to allergen sources as this is important information for public health planning and decision making. The few descriptive studies carried out in Zimbabwe lack the element of mathematical dynamics between allergens and the human population. Mathematical modelling studies that have been pivotal to the understanding of infectious diseases may also be useful in understanding the current and projected burden of allergies, whose results should inform relevant public health policy formulation.

This study was carried out to characterize the trends of food and inhalant allergy prevalence, to project the prevalence and to determine estimates of allergy epidemiology. The causal effect of allergy susceptibility and exposure were incorporated into a mathematical model to understand the extent to which allergic diseases prevalence has been changing over time and to forecast future changes.

The model

A standard *S* (Susceptible), *E* (Exposed), *I* (infected), *R* (Recovered) infectious disease model (SEIR)[6] was modified and used to illustrate the progression

of allergic diseases. It is important to stress here that modelling allergies is not equivalent to modelling infectious diseases but rather diseases with environmental transmission pathways (Lanzas, 2020).[7] A nonlinear mathematical model to study the spread of asthma, with a similar framework, due to inhaled pollutants from industry as well as tobacco smoke from smokers in a variable size population is presented in Naresha and Tripathi.[8] In this model, the entire population is considered to be susceptible to allergens, and after contact with a specific allergen, one is sensitised to the specific allergen and in this case only 2 allergen groups, food and inhalants, are considered. Once sensitised, an individual may become tolerant.

The SEIR model was adopted since we are focusing mainly on the population level spread of allergies through the interaction of the human population with allergens. This is a new method being used to understand the dynamics between the human population and allergens and to predict the prevalence of allergies in the Zimbabwean population.

In our model, the susceptible class (S) refers to the healthy individuals that have not been exposed to an allergen. The exposed individuals are those who ingest food, become sick, and get sensitised to a particular food allergen and join the class (X_f). The food allergens, A_f, grow at a rate $\wedge_f$ in the environment and are consumed at a rate $\nu_f A_f$. Inhalant allergens A_i, grow at a rate $\wedge_i$ and they decrease at a rate $\nu_i A_i$. When susceptible individuals get exposed and interact with inhalant allergens, they move into the class X_i which is the class of individuals that are sensitised to a particular inhalant allergen. The tolerant T_t are individuals who were once sensitised to either food or inhalant allergens and can later be exposed to either food or inhalant without getting affected. We assume that once individuals are tolerant to an allergen, then they belong to a single class irrespective of what they are tolerant to. The description of the model is depicted in Fig. 1.

The model was built under the following assumptions.

1. The rate of exposure to the allergens is assumed to be different for food and inhalant allergens.
2. The population in each class is assumed to be homogeneous for the allergen they are allergic to.
3. The interactions of the susceptible individuals and the allergens are assumed to be homogenous.
4. We assume a mass action form of interaction as humans and the allergens are assumed to have an equal chance of coming into contact.
5. We assume that tolerance is the final state any individual who develops allergies will get to.

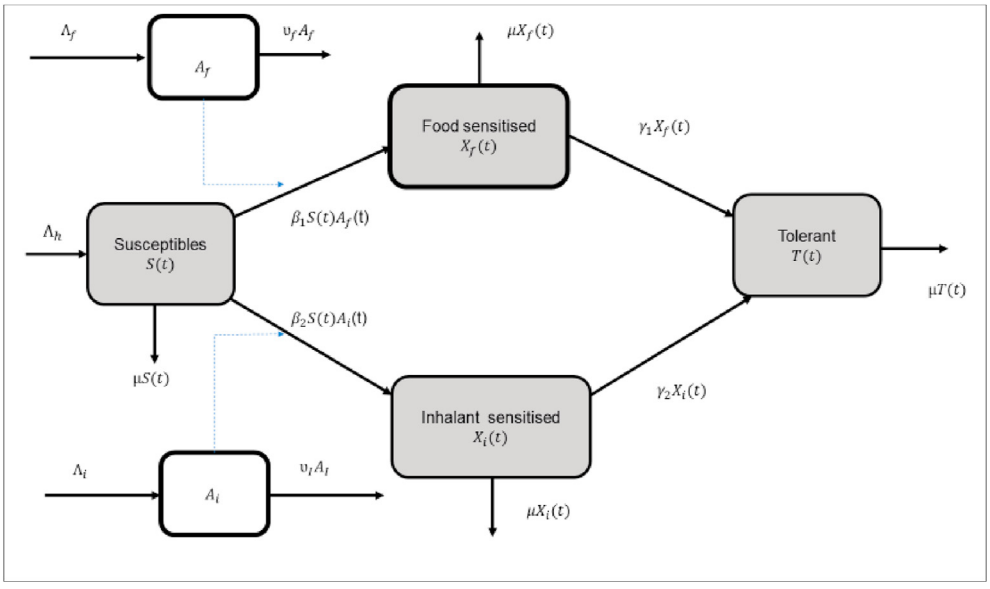

Fig. 1 Model framework

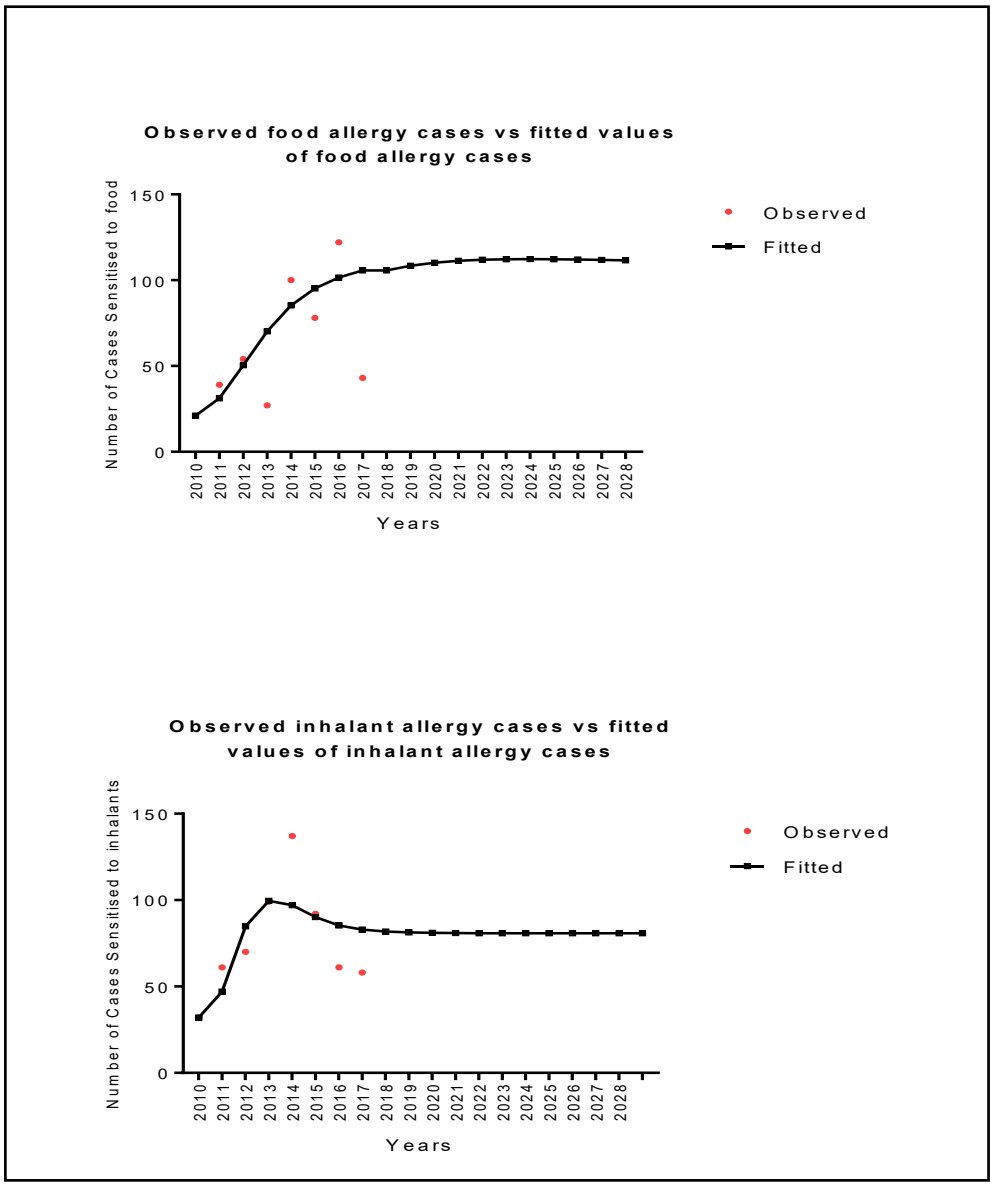

Fig. 2 Trends of food and inhalant allergies recorded in Zimbabwe from 2010 to 2017

6. The rate at which individuals develop tolerance for allergens is assumed to be different for food and inhalant allergens.
7. Allergy is not fatal; hence there are not disease-related parameters for the sensitised.

Model equations

When individuals get exposed to allergens, some of them interact with the allergens and develop an immunological response resulting in the production of IgE antibodies. They show allergy symptoms and are referred to the physician where they get tested for allergy. They may express antibodies specific to some allergen(s) or they may not be sensitised to an allergen.

The rate of change of the susceptible population is increased through births at a rate, Λ_h. This population is reduced by the sensitisation to food and inhalant allergens at rates β_1 and β_2, respectively and through natural mortality (assumed for all the human compartments) that occurs at a rate μ.

Hence the rate of change of the susceptible population is represented by the ordinary differential equation below.

$$\frac{dS}{dt} = \wedge_h - \beta_1 SA_f - \beta_2 SA_i - \mu S, \tag{3.1}$$

The rate at which individuals leave the susceptible population is equal to the rate at which they enter the sensitised classes. The proportion of individuals in the sensitised population increase since those in the susceptible class become exposed and some get sensitised to the allergens over time.

Let γ_1 and γ_2 be the rates at which sensitised individuals become tolerant to the food and inhalant allergens, respectively. Then, the rate of change of the population sensitised to only food allergens is given by

$$\frac{dX_f}{dt} = \beta_1 SA_f - (\mu + \gamma_1)X_f. \tag{3.2}$$

The rate of change of population sensitised to only inhalant allergens is given by

$$\frac{dX_i}{dt} = \beta_2 SX_i - (\mu + \gamma_2)X_i, \tag{3.3}$$

and the rate of change of the tolerant population is given by

$$\frac{dX_T}{dt} = \gamma_1 X_f + \gamma_2 X_i - \mu X_T. \tag{3.4}$$

The amounts of the allergens also vary with time depending on the availability and interaction of the available allergens with humans. The growth rates of food and inhalant allergens in the environment are respectively given by $\wedge_f$ and $\wedge_i$. On the other hand, the depletion rates of food and inhalant allergens are given by υ_f and υ_i respectively.

The rate of change of the amount of food allergens is given by

$$\frac{dA_f}{dt} = \wedge_f - \upsilon_f A_f, \tag{3.5}$$

while the rate of change of the amount inhalant allergens is given by

$$\frac{dA_i}{dt} = \wedge_i - \upsilon_i A_i. \tag{3.6}$$

We thus have the following model:

Human population

$$\frac{dS}{dt} = \wedge_h - \beta_1 SA_f + \beta_2 SA_i - \mu S, \tag{3.7}$$

$$\frac{dX_f}{dt} = \beta_1 SA_f - (\mu + \gamma_1)X_f. \tag{3.8}$$

$$\frac{dX_i}{dt} = \beta_2 SX_i - (\mu + \gamma_2)X_i, \tag{3.9}$$

$$\frac{dX_T}{dt} = \gamma_1 X_f + \gamma_2 X_i - \mu X_T \tag{3.10}$$

Allergens

$$\frac{dA_f}{dt} = \wedge_f - \upsilon_f A_f, \tag{3.11}$$

$$\frac{dA_i}{dt} = \wedge_i - \upsilon_i A_i. \tag{3.12}$$

The sensitisation rates, tolerance rates, birth rate/death, growth rate of allergens, and depletion rate of allergens are all non-negative. We summarise the descriptions of the state variable and parameters in the following tables.

Before we carry out any analysis of the model, it is important to look at the model properties to guarantee the existence and uniqueness of solutions, which in turn guarantees that our model is biologically feasible.

Model properties

Given that the summation of the equations for the human population N, is

$$\frac{dN}{dt} = \wedge_h - \mu N, \tag{3.13}$$

then, when we separate variables we have

$$\frac{dN}{\wedge_h - \mu N} = dt. \tag{3.14}$$

Integrating and solving for N gives

$$N(t) = \frac{\wedge_h}{\mu} + N_0 e^{-\mu t} \quad where \quad N_0 = N(0). \tag{3.15}$$

We observe that $lim_{t\to\infty} N(t) = \frac{\Lambda_h}{\mu}$. The human population will thus not exceed $\frac{\Lambda_h}{\mu}$. We thus mathematically say the population is bounded by the

expression $\frac{\Lambda_h}{\mu}$.

Solving the equations of the allergens we have, $A_f \to \frac{\wedge_h}{\upsilon_f}$ as $t \to \infty$ and $A_i \to \frac{\wedge_h}{\upsilon_i}$.

Therefore the state variables remain biologically meaningful in the set $\Omega = \left\{(S, X_f, X_i, T, A_f, A_i) \in \mathbb{R}_+^6 \middle| 0 < N \leq \frac{\wedge_f}{\mu}, \quad 0 < A_f \leq \frac{\wedge_f}{\upsilon_f}, 0 < A_i \leq \frac{n_i}{\upsilon_i}\right\}$ for all positive initial conditions in $\mathbb{R}^6$. The set Ω is said to be positively invariant and all solutions of system (3.1)–(3.6) with initial conditions $(S_0, X_{f0}, X_{i0}, A_{f0}, A_{i0}) \in \mathbb{R}^6$ remain in Ω for all $t > 0$.

The system is thus mathematically and epidemiologically well-posed, as the solutions are positive and bounded in Ω. We shall thus base our analysis on the solutions generated in Ω.

Model equilibria

By using the right-hand side of system (3.7)–(3.12) the model equilibria are obtained from

$$0 = \wedge_h - \beta_1 S A_f - \beta_2 S A_i - \mu S \tag{3.16}$$

$$0 = \beta_1 S A_f - (\mu + \gamma_1) X_f \tag{3.17}$$

$$0 = \beta_2 S A_i - (\mu + \gamma_2) X_i \tag{3.18}$$

$$0 = \gamma_1 X_f + \gamma_2 X_i - \mu X_T \tag{3.19}$$

$$0 = \wedge_f - \upsilon_f A_f \tag{3.20}$$

$$0 = \wedge_i - \upsilon_i A_i. \tag{3.21}$$

Using Mathematica, we establish that the model has one non-trivial equilibrium point given by

$$E = \left(S^*, X_f^*, X_i^*, T, A_f^*, A_i^*\right) \tag{3.22}$$

where:

$$S^* = \frac{\wedge_h \upsilon_f \upsilon_i}{\mu \upsilon_f \upsilon_i + \beta_1 \upsilon_i \wedge_f + \beta_2 \upsilon_f \wedge_i}. \tag{3.23}$$

$$X_f^* = \frac{\beta_1 \wedge_f \wedge_h \upsilon_i}{(\mu + \gamma_1)(\mu \upsilon_i \upsilon_f + \beta_1 \upsilon_i \wedge_f + \beta_2 \upsilon_f \wedge_i)} \tag{3.24}$$

$$X_i^* = \frac{\beta_2 \wedge_i \wedge_h \upsilon_f}{(\mu + \gamma_2)(\mu \upsilon_i \upsilon_f + \beta_1 \upsilon_i \wedge_f + \beta_2 \upsilon_f \wedge_i)} \tag{3.25}$$

$$X_T^* = \frac{\wedge_h (\beta_1 \gamma_1 \wedge_f \upsilon_i (\mu + \gamma_2) + \beta_2 \gamma_2 \wedge_i \upsilon_f (\mu + \gamma_1)}{\mu(\mu + \gamma_1)(\mu + \gamma_2)(\mu \upsilon_i \upsilon_f + \beta_1 \upsilon_i \wedge_f + \beta_2 \upsilon_f \wedge_i)} \tag{3.26}$$

$$A_f = \frac{\wedge_f}{\upsilon_f} \tag{3.27}$$

$$A_i = \frac{\wedge_i}{\upsilon_i} \tag{3.28}$$

The existence of non-trivial steady states suggests that there will never be an allergy-free population (see Table 1) (see Table 2).

Variable	Description
$S(t)$	The number of susceptible individuals in the population at time, *t*.
$X_f(t)$	The number of individuals sensitised to food allergens in the population at time, *t*.
$X_i(t)$	The number of individuals sensitised to inhalant allergens at time, *t*.
A_f	The amount of food allergens in the country at a time, *t*.
A_i	The amount of inhalant allergens in the country at a time, *t*.
$T(t)$	The proportion of individuals who develop tolerance to the allergens at time*t*.

Table 1. Variables and their descriptions

Parameter	Description
β_1	The rate sensitisation of the susceptible population when they interact with the food allergens.
β_2	The rate of sensitisation of the susceptible population when they interact with inhalant allergens.
μ	Natural birth/death rate.
γ_1	The rate of developing tolerance.
γ_2	The rate of developing tolerance.
$\wedge_h$	The rate of growth of the susceptible population.
$\wedge_f$	The growth rate of food allergens in the environment.
$\wedge_i$	The growth rate of inhalant allergens in the environment.
υ_f	The rate of depletion of food allergens.
υ_i	The rate of depletion of inhalant allergens.

Table 2. Parameter symbols and their description

NUMERICAL SIMULATIONS

Numerical simulations on the allergies model were done by fitting the model to the data. Matlab was used, and the values of the parameters obtained are in Table 3. The effects and changes that occur in the model after changing the values of some compartments in the model were investigated. Time was measured in years.

Records form the Asthma Allergy and Immune Dysfunction clinic show that allergies are endemic in Zimbabwe.[1,9] For example, from 2010 to 2014, there was a general increase in the number of both food and inhalant allergy cases, though a drop in the food allergy cases was seen in 2013.

Using the formulated model and fitting the observed data to the model and the results are depicted in Fig. 2. The sensitisation rate of inhalant allergens was observed to be higher than the sensitisation rate to food allergens, 0.066 and 0.2363, respectively. Food allergens, depletion and growth rates were observed to be higher than those of inhalant allergens. The population develops a tolerance to food allergens at a faster rate than inhalant allergens.

A susceptible population growth rate of 50, food and inhalant allergen availability increasing at a rate of 0.053 and 0.071, respectively, the food allergen sensitised compartment increased gradually each year and the inhalant allergen sensitised increase between 2010 and 2011 and remained constant from 2011 to around 2015 then began to decrease as shown in Fig. 3. The susceptible population decreased sharply because of the interaction of the susceptible population with allergens and moving to the food and inhalant allergies compartments.

Fig. 4 shows that doubling the growth rate of the susceptible population and the growth rates of the allergens resulted in an increase in the numbers in both the food allergy and inhalant allergy compartments with the rate of increase of the food allergy compartment being faster than that of inhalant allergens. The susceptible population increased in the first 4 years and began to decrease as more and more individuals continuously move to the food and inhalant sensitised compartments due to increased rates of interaction with the allergens. The number of individuals in the inhalant allergies compartment will decrease in the future while that in the food allergies compartment is seen to increase.

DISCUSSION AND CONCLUSION

In this paper, a mathematical model of the prevalence and acquisition of food and inhalant allergies was studied. A standard SEIR model was adopted and modified to predict the prevalence of food and inhalant allergies as well as determining the parameter estimates for acquiring allergy after exposure to allergens.

By analysing the model, we found that the prevalence of food allergy will increase in the next 2 decades and the prevalence of inhalant allergies will decrease. The existence of non-trivial steady states suggested that allergy will be endemic in the Zimbabwean population. Incidentally, when individuals are exposed to food allergens and interact with them, there is a 0.066 probability that the individuals get sensitised to the food allergens. Whereas, if individuals get exposed to inhalant

Parameter	Symbol	Value
The sensitisation rate of the susceptible population when they interact with food allergens	β_1	0.066
The sensitisation rate of the susceptible population when they interact with inhalant allergens	β_2	0.2363
The depletion rate of food allergens	υ_f	0.347
The depletion rate of inhalant allergens	υ_i	0.2124
Natural birth/death rate.	μ	0.0342
Rate of developing tolerance.	γ_1	0.0116
Rate of developing tolerance.	γ_2	0.005
Rate of growth of the susceptible population.	$\wedge_h$	1.00
The growth rate of food allergens in the country.	$\wedge_f$	0.8945
The growth rate of inhalant allergens in the country.	$\wedge_i$	0.7608

Table 3. Parameter estimates

allergens and interact with them, there is 0.2363 probability of getting sensitised to the inhalant allergens.

The model also showed that individuals develop tolerance to food allergens at a faster rate than the rate at which they develop tolerance to inhalant allergens with rates and γ_2 (0.01164 and 0.005), respectively. This means most people outgrow food allergies and most people live with inhalant allergies.

It was also seen that increasing the growth rate of the susceptible individuals, food allergens availability and inhalant allergen availability, increases the rate at which individuals interact with the allergens thereby increasing the number of individuals

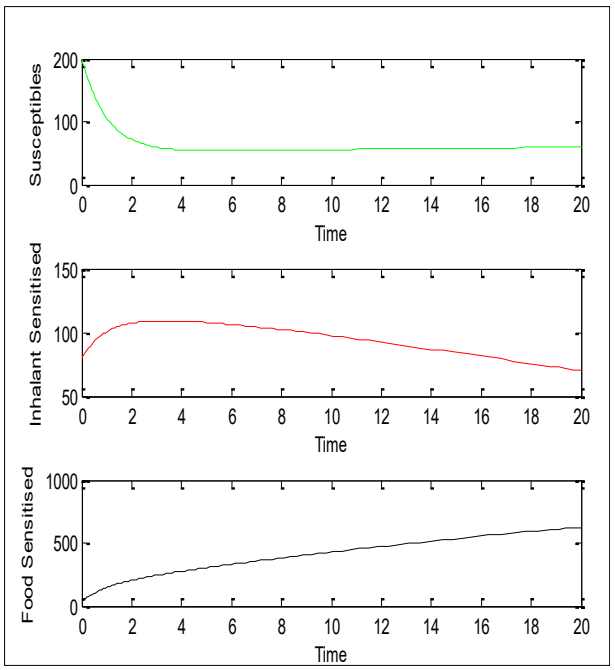

Fig. 3 Simulations over 20 year period with $\wedge_h = 50$, $\wedge_f = 0.053$, $\wedge_i = 0.071$

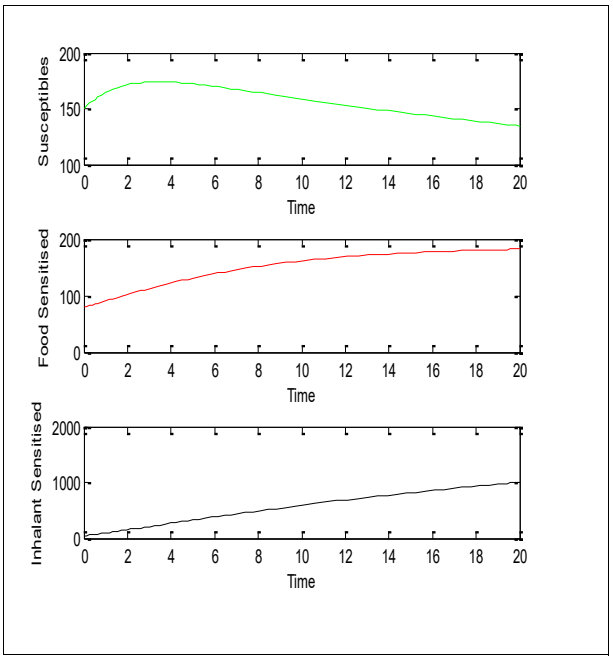

Fig. 4 Simulations over 20 year period with $\wedge_h = 100$, $\wedge_f = 0.106$, $\wedge_i = 0.142$

who get sensitised to the allergens. This means that stakeholders need to put in place measures to manage the disease for it will affect a greater proportion of the population in the near future.

In order to understand the trajectory of food and inhalant allergies, there is need to understand the mechanism of acquiring allergies. Many non-communicable diseases have been modelled using differential equations. The purpose of this study was to examine the mechanism for acquiring allergies and the trend of the prevalence of allergies and solve using differential equations.

The model presented in this paper is unquestionably very simple and can be improved in many ways. First, the model only focusses on 2 types of allergen groups when in actual fact there are more than 2. Second, the model fitting can be expanded to include the goodness of fit as observed in Fig. 2; the data have high statistical variance. The paper could be improved by including statistical measures reflecting the degree of fit. Despite these shortcomings, the model presents some interesting results on how mathematical models can be used to track the changes in the prevalence of allergens.

The model pointed out that allergy will be endemic in Zimbabwe with the prevalence of food allergy increasing and the prevalence of inhalant allergies tending to decrease in the next 2 decades. People should be educated to create awareness of allergy so that the community is cognisant of this chronic disease.

Abbreviations

SER; Susceptible, Exposed, Recovered, SEIR; Susceptible, Exposed, Infected, Recovered, IgE; immunoglobulin E.

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Authors' consent for publication

All authors consent to the publication of this manuscript.

Author contributions

MC, FN and EC were involved in the generation of the model extensively contributed to writing the paper, reviewed and approved the final draft for publication. SR supervised the student, guided statistical analyses, read and approved the manuscript for publication. LP and HP were involved in the data collection, reviewed the manuscript and approved the submission of the manuscript. ES was responsible for the clinical care of all the patients included in the model, supervised the laboratory testing, revised, reviewed and approved the submission of the manuscript.

Availability of data and materials

All data and materials from which the data are derived are available from the corresponding author and are accessible on request.

Ethics approval

Institutional approval was obtained. The anonymised analysis of these data is not applicable.

Declaration of competing interest

The authors declare no competing interests with respect to this manuscript.

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REFERENCES

1. Sibanda EN. Increasing trend of sensitization to food and inhalant allergen 343 sources in Zimbabwe. *Curr Allergy Clin Immunol*. 2013;26:214–219.
2. Pawankar R, Canonica GW, Holgate ST, Lockey RF. *World Allergy Organization (WAO) White Book on Allergy*. Wisconsin: World Allergy Organisation; 2011.
3. Gray CL. Food allergy in South Africa. *Curr Allergy Asthma Rep*. 2017;17(6):35.
4. Oluwole O, Arinola O, Falade G, et al. Allergy sensitization and asthma among 13-14 year old school children in Nigeria. *Afr Health Sci*. 2013;13(1). https://doi.org/10.4314/ahs.v13i1.20.
5. Kung S-J, Mazhani L, Steenhoff AP. Original research article. *Curr Allergy Clin Immunol*. 2013;26:8.
6. Hethcote HW. The mathematics of infectious diseases. *SIAM Rev*. 2000;42(4):599–653. https://doi.org/10.1137/S0036144500371907.
7. Lanzas C, Davies K, Erwin S, Dawson D. *On Modelling Environmentally Transmitted Pathogens*. 2020. https://doi.org/10.1098/rsfs.2019.0056. Interface, Focus.1020190056.
8. Naresha R, Tripathi A. A nonlinear mathematical model for asthma: effect of environmental pollution. *Iran J Optim*. 2009;1(1):24–56.
9. Sibanda EN. Inhalant allergies in Zimbabwe: a common problem. *Int Arch Allergy Immunol*. 2003;130(1):2–9.