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Effectivity of the Vaccination Strategy for a Fractional-Order Discrete-Time SIC Epidemic Model

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Abstract. Indirect disease transmission is modeled via a fractional-order discrete-time Susceptible-Infected-Contaminant (SIC) model vaccination as a control strategy. Two control actions are considered, giving rise to two different models: the vaccine efficacy model and the vaccination impact model. In the first model, the effectiveness of the vaccine is analyzed by introducing a new parameter, while in the second model, the impact of the vaccine is studied incorporating a new variable into the model. Both models are studied giving population thresholds to ensure the eradication of the disease. In addition, a sensitivity analysis of the Basic Reproduction Number has been carried out with respect to the effectiveness of the vaccine, the fractional order, the vaccinated population rate and the exposure rate. This analysis has been undertaken to study its effect on the dynamics of the models. Finally, the obtained results are illustrated and discussed with a simulation example related to the evolution of the disease in a pig farm.

Keywords: epidemic process, discrete fractional-order, indirect transmission, vaccination, sensitivity analysis.

AMS Subject Classification: 39A60; 92D30; 93B35.

1 Introduction

Epidemic processes are usually described using continuous and discrete time mathematical models based on differential or difference equations [12]. Nowadays, fractional order derivative approximations have been incorporated into this kind of model [3,11,15,18,22,25] to achieve better simulations of the real dynamical system since they consider the effect of the memory in the process. Several continuous models apply the Caputo's fractional derivative [8,10,17,18,21] or a modified Riemann-Liouville derivative [25]. Other works use difference fractional operators on a discretized continuous model [4, 20]. Also, starting directly from a discrete time model, some works use the Riemann-Liouville, the Caputo or the Grünwald-Letnikov fractional difference operators [9, 14, 15].

An important feature to be considered in epidemiological studies is the implementation of control strategies to improve the eradication of the disease. Different strategies exist to do this, such as quarantine, isolation, vaccination, etc. [1, 13, 23]. When vaccination is considered, determining the number of vaccinated individuals to eradicate the disease is one of the questions to be answered. Moreover, the effectiveness of the vaccine is also something to be considered in the model to better describe the process. Several mathematical models have been developed with vaccination strategies [26]. Some of them study the effectiveness of the vaccine [16], and other ones consider a new variable in the model representing the vaccinated population [24].

The different control strategies used in the mathematical modelling of epidemic processes involve several parameters, such as the effectiveness of the vaccine, the prevalence of the disease, the vaccination rate, etc. A sensitivity analysis of the effect on the basic reproduction number of the model produced by variations of the involved parameters can help to define the direction of the control strategies. The sensitivity index for the reproduction number with respect to a chosen parameter shows the effect of this parameter in the dynamical behavior of the system [24, 27, 28] and allows us to determine the importance of each parameter in the prevalence of the disease. It gives the necessary ratio of change in the parameter to get the desired change in the basic reproduction number.

Animal production is considered an essential source of food for humans. The way animals are raised has changed in last decades, and the stress of intense confinement can amplify the number and type of pathogens they harbor. This makes anyone who eats its meat susceptible to carrying a disease. And if an infectious, transmissible pathogen spreads from animals to people, as Covid-19 did, every person on this planet could be at risk. That is why it is so important to eradicate zoonotic diseases, such as salmonellosis, brucellosis, or hepatitis E, on farms. So, preventing them from the beginning helps human well-being and health.

The elaboration of a mathematical model is necessary to understand the spread of diseases and to establish reliable measures of prevention and control. Indirect transmission of diseases like Salmonella encourages us to use epidemic models that consider the amount of contaminant found in the environment as a variable together with susceptible and infected individuals [7,12]. This kind of

disease has been studied in the literature in different situations. For example, in [19], the authors experimentally studied the case of Salmonella transmission in organic farms, where the pig production is less than 200 pigs per year. In [5], a semi-stochastic model has been analyzed for Salmonella infection in the UK.

In this paper, we have analyzed a model of indirect disease transmission between animals, intending to simulate the conditions present in herds. The specific objective of this work is to examine how vaccination, as a control strategy, affects the eradication of a pathogen at the herd level. Starting from a fractional-order discrete-time SIC epidemic model given in [11], we construct two models applying two different vaccination control actions. One focuses on the effectiveness of the vaccine, while the other is worried about its impact. For that, the first model incorporates the effect parameter and the vaccination population rate, and the second one has a vaccination rate parameter and a new variable that accounts for the vaccinated individuals together with an exposition rate of the vaccinated population. For both models, a sensitivity analysis is done for the corresponding basic reproduction numbers with respect to some of the parameters involved, including the fractional order one. Also, bounds on the population are obtained to assure that the disease is erradicated. The implemented models are applied to a pig production farm as an example of the application of this kind of epidemic systems.

This paper is organized as follows. Section 2 presents the problem statement, explaining the two vaccination models analyzed in this paper, the *vaccine effectiveness model* and the *vaccination impact model*. These two vaccination actions are control strategies incorporated to the mathematical model defined in [11] to stabilize the system at the disease-free equilibrium point. Section 3 studies the *vaccine effectiveness model*, making a sensitivity analysis of the basic reproduction number for the parameters involved in the model (the effectiveness, the vaccination rate, and the fractional order). In Section 4, the *vaccination impact model* is analyzed, and some sensitivity analyses for the basic reproduction number are also done. In Section 5, a discussion of the obtained results and some numerical examples to illustrate the behavior of the models are presented. Some comparisons of the different behavior between the two models are also shown in this section. Finally, in Section 6, the main conclusions of the paper are summarized.

2 Problem statement

Consider an epidemic process with indirect transmission under the assumption of constant population size so that the infectious disease is not transmitted from individual to individual but through the contaminant. The population is epidemiologically divided into susceptible $S(t)$ and infectious $I(t)$ classes. So, we assume that the number of susceptible individuals plus the number of infected individuals is the total of the population, $P = S(t) + I(t)$, $t \geq 0$. Moreover, we consider that the transmission of this disease occurs by contact with the environmental contaminant $C(t)$. Infected individuals remain in the environment, and the contaminant produced by these individuals can infect others. Therefore, the evolution of individual classes is represented by means

of a Susceptible-Infected-Contaminant (SIC) model [12]

$$\begin{aligned} S(t+1) &= pS(t) - \sigma C(t)S(t) + \mu(t)P, \\ I(t+1) &= qI(t) + \sigma C(t)S(t) \\ C(t+1) &= sC(t) + \beta I(t), t \geq 0. \end{aligned} \tag{2.1}$$

In Table 1, we summarize the biological meaning of the natural parameters involved in this epidemic model.

Table 1. Parameters of the model.

Parameter	Description
σ	Exposition or infection rate of susceptible individuals.
$\mu(t)P$	Replacement rate of dead individuals.
p/q	Survival rate of the susceptible/ infected population, respectively.
s	Survival rate of the contaminant.
β	The amount of contaminant produced by each infected individual.

Note that the involved parameters must meet certain conditions to ensure that the model has a biological sense. That is, $0 < p,\ q,\ s < 1,\ q < p$, $0 < \sigma < p < 1$ and $\beta > 0$.

In [11], a discrete-time mathematical model was presented to study the evolution of disease through indirect transmission using a fractional order discrete model with k memory steps. To construct the model, we use the following truncated discrete-time fractional order (DTFO) operator [11, 12, 15],

$$\Delta_k^\alpha x(t) = \sum_{j=0}^{k} a_j^\alpha x(t-j), \tag{2.2}$$

where the fractional order α satisfies $0 < \alpha \leq 1$ and

$$a_j^\alpha = (-1)^j \binom{\alpha}{j},\ j \geq 0, \text{ with } \binom{\alpha}{j} = \begin{cases} 1, & j = 0 \\ \dfrac{\alpha(\alpha-1)...(\alpha-j+1)}{j!}, & j > 0. \end{cases}$$

The epidemic model (2.1) can be rewritten using the notation

$$x(t) = (x_1(t)\ x_2(t)\ x_3(t))^T = (S(t)\ I(t)\ C(t))^T,$$

with $x_1(t)$ representing the susceptible individuals, $x_2(t)$ the infected individuals and $x_3(t)$ the contaminant. From this and using the DTFO operator (2.2), we define the discrete-time fractional order (DTFO) model with k memory steps as

$$x(t+1) = Ax(t) - \sum_{j=2}^{k} a_j^\alpha x(t+1-j) + f(x(t)) + B\mu(t)P,\ t \geq k-1, \tag{2.3}$$

where

$$A = \begin{pmatrix} p-1+\alpha & 0 & 0 \\ 0 & q-1+\alpha & 0 \\ 0 & \beta & s-1+\alpha \end{pmatrix}, \; B = e_1,$$

with e_1 the first vector of the canonical basis for the three-dimensional space $\mathbb{R}^3$ and $f(x(t)) = \sigma x_1(t)x_3(t)v_1$ being $v_1 = (-1\ 1\ 0)^T$. Note that, in order to keep a constant population P at any time t, the replacement rate of dead individuals has to satisfy

$$\mu(t)P = (p-q)x_1(t) + P(1-q+\Sigma_k^\alpha), \quad t \geq k-1,$$

with $\Sigma_k^\alpha = \sum_{j=0}^{k} a_j^\alpha$, $k \geq 0$, $\quad 0 \leq 1-\alpha < \min\{q, s\}$.

In [11], the authors obtained a threshold for the population size P in order to assure a non-negativity trajectory of the solution of the model. This non-negativity bound, denoted by $\mathcal{C}_+$ is

$$\mathcal{C}_+ = \omega_\alpha K_\alpha, \text{ with } \omega_\alpha = \frac{q-1+\alpha}{\Theta_q} \text{ and } K_\alpha = \frac{\Theta_q \Theta_s}{\beta\sigma}, \tag{2.4}$$

where $\Theta_\theta = 1-\theta+\Sigma_k^\alpha$, $\theta = p,\ q$ or s. That is, if $P < \mathcal{C}_+$, the solution $x(t)$ of the model is non-negative. Moreover, for model (2.3), the disease-free equilibrium point, $x_f = (P, 0, 0)$, and the basic reproduction (BR) number,

$$R_0(\alpha, k) = \sqrt{\frac{\beta\sigma P}{\Theta_q \Theta_s}} \tag{2.5}$$

were obtained. Therefore, if $R_0(\alpha, k) < 1$, we can ensure that the solution tends toward the disease-free equilibrium point. Taking into account the expression of $R_0(\alpha, k)$, if the size of the population meets $P < K_\alpha$, then the disease is eradicated.

From now on, we assume $P < \mathcal{C}_+$ to assure the non-negativity of the solution. If $\omega_\alpha < 1$ we have $P < \omega_\alpha K_\alpha < K_\alpha$. Then the disease tends to disappear. On the other hand, if $\omega_\alpha > 1$ and the population satisfies $K_\alpha < P < \mathcal{C}_+$ the system does not reach the disease-free equilibrium point, and the disease remains. Then, it is interesting to apply control actions to achieve the eradication of the disease. In particular, we consider vaccination as such a control action.

In this paper, we consider how vaccination affects the transmission of the disease in this infectious process. We have made two assumptions:

- In our first assumption, the vaccine has a rate of effectiveness over the vaccinated individuals in such a way that when it is effective in an individual, it immunizes him completely. The individuals where the vaccine is effective are no longer susceptible to infection.

- In the second assumption, we have introduced a new variable $V(t)$ that corresponds to the vaccinated individuals. In this case, the vaccine does not fully immunize but reduces the risk of infection.

In both cases, we are interested in finding a lower bound on the vaccinated population rate to get that the disease tends to disappear. Also, we want to study how the size of the population affects the disease transmission process and to carry out a sensitivity analysis on some parameters related to the transmission of the disease. This analysis will be made from the explicit expression of the basic reproduction number of the models.

From now on, we consider an epidemic process modelled by the system (2.3) assuming that $\omega_\alpha > 1$ and $K_\alpha < P < \mathcal{C}_+$, or equivalently, $R_0(\alpha, k) > 1$. Remind, in this case, the disease remains since the trajectory of the system does not tend toward the disease-free equilibrium point. To ensure that the disease tends to disappear, we propose the control action of vaccinating a subset of the susceptible population using one of the two assumptions exposed above. The vaccinated population rate is denoted by v, and a reasonable assumption is to consider this rate is less or equal to the survival rate, $0 \leq v \leq p$.

3 Vaccine effectiveness model

In this section, we propose to study the effectiveness of the vaccine in a certain population. In this model, we assume that the vaccine either produces no effect or produces complete immunity. Since we want to study the protection provided by the vaccine in the vaccinated population, in addition to the parameter v, $0 \leq v \leq p$, we introduce a new parameter ϵ, $0 < \epsilon < v$. This parameter, ϵ, measures the percentage of vaccinated individuals who continue being susceptible to infection. Therefore, the factor $(1-\epsilon)$ represents the vaccine effectiveness.

Based on the DFTO model (2.3), we propose a new discrete-time fractional-order epidemic model with vaccination introducing the vaccinated population rate, v, and percentage of those vaccinated who continue to be susceptible to infection, ϵ. This model is given by

$$x(t+1) = Ax(t) - \sum_{j=2}^{k} a_j^\alpha x(t+1-j) + f_e(x(t)) + B\mu(t)P, \quad t \geq k-1,$$

where $f_e(x(t)) = \sigma(1 - v(1-\epsilon))x_1(t)x_3(t)v_1$. This is a model with k steps of memory since the solution $x(t+1)$ depends on the states $x(t), x(t-1), \ldots, x(t-k+1)$, and in the same way as it is done in [11], from now on we only consider $\alpha \in \Lambda_e$, where

$$\Lambda_e = \{\alpha \in]0,1] \ / \ 0 \leq 1-\alpha < q, \ 0 \leq 1-\alpha < s\}.$$

The condition of constant population P at any time $t \geq 0$ implies that the addition of the two first equations gives $\mu(t)P = (q-p)x_1(t) + P\Theta_q, \quad t \geq k-1$. So, applying this condition to our model, we have

$$x(t+1) = \tilde{A}_e \ x(t) - \sum_{j=2}^{k} a_j^\alpha x(t+1-j) + f_e(x(t)) + B\Theta_q P, \quad t \geq k-1, \ (3.1)$$

where

$$\tilde{A}_e = \begin{pmatrix} q-1+\alpha & 0 & 0 \\ 0 & q-1+\alpha & 0 \\ 0 & \beta & s-1+\alpha \end{pmatrix}.$$

We can observe that, in this model, the survival rate of the susceptible population does not appear explicitly in the equations.

Now, to have a model with biological sense, the solution of the system must be non-negative. Following an analogous reasoning to the one used in [11], we have that this is achieved if

$$P < \frac{1}{1-v(1-\epsilon)}\mathcal{C}_+,$$

with $\mathcal{C}_+$ given in (2.4).

Note that, this non-negativity bound, denoted by $\mathcal{C}_{e+}$, is lower than $\mathcal{C}_+$ and, by the expression of $\mathcal{C}_+$, it is given by

$$\mathcal{C}_{e+} = \frac{q-1+\alpha}{(1-v(1-\epsilon))\Theta_q}K_\alpha = \frac{\omega_\alpha}{1-v(1-\epsilon)}K_\alpha. \tag{3.2}$$

Furthermore, we have that

$$R_{e0}(\alpha,k,v,\epsilon) = \sqrt{\frac{\beta\sigma(1-v(1-\epsilon))P}{\Theta_q\Theta_s}} \tag{3.3}$$

is an explicit expression of the basic reproduction (BR_e) number associated with this *vaccine effectiveness model*.

It is easy to find the relationship between the basic reproduction numbers corresponding to the model with and without vaccination. From (2.5) and (3.3),

$$R_{e0}^2(\alpha,k,v,\epsilon) = R_0^2(\alpha,k)(1-v(1-\epsilon)). \tag{3.4}$$

In the following result, we study how vaccination can improve the disease transmission process. In particular, we give a lower bound for the vaccination rate of our *vaccine effectiveness model* that makes the disease tends to disappear, and we determine the minimum percentage of vaccinated individuals that allows the eradication of the disease.

Proposition 1. *Consider the model given in (2.3) with $R_0(\alpha,k) > 1$ and the vaccine effectiveness model given in (3.1). If the parameters ϵ and v satisfy $\epsilon < \dfrac{1}{R_0^2(\alpha,k)}$ and $v > \dfrac{1}{1-\epsilon}\left(1-\dfrac{1}{R_0^2(\alpha,k)}\right)$, then $R_{e0}(\alpha,k,v,\epsilon) < 1$.*

Proof. If $R_0(\alpha,k) > 1$ then $0 < 1 - \dfrac{1}{R_0^2(\alpha,k)} < 1$. From the biological meaning of ϵ we have that $0 < 1-\epsilon < 1$. By (3.4), $0 < 1-v(1-\epsilon) < 1$ and

$$R_{e0}^2(\alpha,k,v,\epsilon) < R_0^2(\alpha,k)\left(1-\frac{1}{1-\epsilon}\left(1-\frac{1}{R_0^2(\alpha,k)}\right)(1-\epsilon)\right) = 1.$$

□

Remark 1. Let us observe that, taking the restrictions on ϵ and v given in the previous result and taking into account the expression of $R_0(\alpha, k)$ given in (2.5) together with $\omega_\alpha > 1$ and the limits on the population size, that is $K_\alpha < P < \mathcal{C}_{e+}$, with $\mathcal{C}_{e+}$ given in (3.2), we can achieve that the disease is eradicated if

$$K_\alpha < P < \min\left\{\frac{1}{\epsilon},\ \frac{1}{1-v(1-\epsilon)}\right\} K_\alpha,$$

and the solution of *vaccine effectiveness model* is non-negative.

3.1 Sensitivity analysis

Sensitivity analysis helps to determine the most influential parameters of the model and to optimize its structure. In particular, we are interested in using this analysis to estimate how the corresponding parameter should be modified to decrease the basic reproduction number by a desired percentage. A decrease in infected individuals reflects a reduction in the BR number value. If the BR number is less than one, a reduction in its value corresponds to a faster approach to equilibrium, that is, a faster disappearance of the disease. And, in the case of a BR number greater than one, its reduction means that the disease does not spread with such virulence. That is, if we manage to reduce it to less than one, the disease will tend to die out.

We use the normalized sensitivity index for a quantity Q with respect to a parameter h defined as, [27],

$$\Phi(Q/h) = \frac{h}{Q}\frac{\partial Q}{\partial h} = h\frac{\partial}{\partial h}\log(Q). \tag{3.5}$$

In this part, we compute the sensitivity indices of the basic reproduction number of the *vaccine effectiveness model*. We are interested in studying the effect of vaccination on the transmission of the disease and the behavior of the model using a fractional approximation. We use the method of direct differentiation to obtain the explicit expressions for the indices associated with the BR_e number, $R_{e0}(\alpha, k, v, \epsilon)$, with respect to parameters v, ϵ and α, respectively, since, generally, the survival rates, p, q, and s, are hard to change.

Proposition 2. *Consider the BR_e number of the vaccine effectiveness model (3.1) given (3.3). Then,*

(i) $\Phi(R_{e0}(\alpha, k, v, \epsilon)/\epsilon) = \dfrac{\epsilon v}{2(1-(1-\epsilon)v)}$;

(ii) $\Phi(R_{e0}(\alpha, k, v, \epsilon)/v) = -\dfrac{v(1-\epsilon)}{2(1-(1-\epsilon)v)}$;

(iii) $\Phi(R_{e0}(\alpha, k, v, \epsilon)/\alpha) = \dfrac{\alpha}{2k!}\left(\dfrac{1}{\Theta_q} + \dfrac{1}{\Theta_p}\right)\displaystyle\sum_{j=1}^{k}\prod_{\substack{i=1\\ i\neq j}}^{k}(i-\alpha).$

With these results, we can analyze how small variations of the parameters v, ϵ, and α affect the BR_e number of the *vaccine effectiveness model*. They will be used in Section 5 to perform some numerical sensitivity analyses.

4 Vaccination impact model

In this section, we propose to study the impact of the vaccine on a specific population. To do this, we apply the control action of vaccinating to our initial model given (2.3) by adding a new variable, $V(t)$, containing the vaccinated individuals and denoting by v, $0 \leq v \leq p$, the vaccinated population rate. Of course, it is assumed that the infection rate is different for vaccinated and unvaccinated individuals, being lower for vaccinated individuals. In addition, we take that the response to the vaccine is the same for all vaccinated individuals. Let η be the parameter representing the exposition or infection rate of vaccinated individuals. So, for a vaccinated individual, the exposition rate satisfies $0 < \eta < \sigma$. Thus, as the population is assumed to be constant, we have $S(t) + V(t) + I(t) = P$, $t \geq 0$.

To use a similar notation to the previous section, we consider $x(t) = (x_j(t))_{j=1,2,3,4}^T$ where $x_1(t)$ denotes the susceptible individuals, $x_2(t)$ the vaccinated individuals, $x_3(t)$ the infected individuals and $x_4(t)$ the contaminant. This new discrete-time fractional order model with k memory steps is given by

$$x(t+1) = A_i x(t) - \sum_{j=2}^{k} a_j^{\alpha} x(t+1-j) + f_i(x(t)) + B\mu(t)P,\ t \geq k-1,$$

where

$$A_i = \begin{pmatrix} p-v-1+\alpha & 0 & 0 & 0 \\ v & p-1+\alpha & 0 & 0 \\ 0 & 0 & q-1+\alpha & 0 \\ 0 & 0 & \beta & s-1+\alpha \end{pmatrix}, \quad B = e_1,$$

with e_1 the first vector of the canonical basis for the four-dimensional space $\mathbb{R}^4$ and $f_i(x(t)) = \sigma x_1(t)x_4(t)u_1 + \eta x_2(t)x_4(t)u_2$, with $u_1 = (-1\ 0\ 1\ 0)^T$ and $u_2 = (0\ -1\ 1\ 0)^T$.

The condition of constant population P at any time $t \geq 0$ implies that the addition of the first three equations gives $\mu(t)P = (q-p)(x_1(t)+x_2(t)) + P\Theta_q$. So, applying this condition to our new model, we have

$$x(t+1) = \tilde{A}_i\ x(t) - \sum_{j=2}^{k} a_j^{\alpha} x(t+1-j) + f_i(x(t)) + B\Theta_q P,\ t \geq k-1, \quad (4.1)$$

where

$$\tilde{A}_i = \begin{pmatrix} q-v-1+\alpha & q-p & 0 & 0 \\ v & p-1+\alpha & 0 & 0 \\ 0 & 0 & q-1+\alpha & 0 \\ 0 & 0 & \beta & s-1+\alpha \end{pmatrix}.$$

In the same way as the previous section, from now on, we only consider $\alpha \in \Lambda_i$, where

$$\Lambda_i = \{\alpha \in]0,1] \ /\ 0 \leq 1-\alpha+v < q,\ 0 \leq 1-\alpha < p,\ 0 \leq 1-\alpha < s\}.$$

Under this assumption, the eigenvalues of matrix $\tilde{A}_i$ have a modulus less than 1. This assures the extinction of the population when no individual replacement is considered.

In the following result, we look for a sufficient condition to ensure that the model trajectory remains non-negative.

Proposition 3. *Let* $\mathcal{C}_{i+} = \dfrac{q-v-1+\alpha}{\Theta_q} K_\alpha$ *and* $\alpha \in \Lambda_i$. *If* $P < \mathcal{C}_{i+}$, *then the solution of system (4.1) is non-negative.*

Proof. From the system equation (4.1) and $x_3(t) \leq P$, for all $t \geq 0$, we have that

$$x_4(t+1) \leq (s-1+\alpha)x_4(t) + \beta P - \sum_{j=2}^{k} a_j^\alpha x_4(t+1-j).$$

Then, we can construct the system

$$y(t+1) = Hy(t) + \beta P e_1$$

with $y(t) = (x_4(t)\ x_4(t-1)\ \ldots\ x_4(t+1-k))^T$, e_1 the first vector of the canonical basis of $\mathbb{R}^{1-k}$ and H a block matrix given by

$$H = \begin{pmatrix} s-1+\alpha & -a_2 & -a_3 & \cdots & -a_{k-1} & -a_k \\ 1 & O & O & \cdots & O & O \\ O & 1 & O & \cdots & O & O \\ \vdots & \vdots & \vdots & & \vdots & \vdots \\ O & O & O & \cdots & 1 & O \end{pmatrix}$$

and whose solution is $y(t) = H^t y(0) + \beta P \sum_{i=0}^{t-1} H^j e_1$.

By using an analogous reasoning to the one given in [11], we prove that this matrix satisfies $\rho(H) < 1$ and $(I-H)^{-1} = \sum_{j=0}^{\infty} H^j$. Then,

$$y(t) \leq \beta P (I-H)^{-1} e_1 = \frac{\beta P}{\Theta_s}(1\ \ldots 1)^T$$

and consequently $x_4(t) \leq \dfrac{\beta P}{\Theta_s}$. Since $P < \mathcal{C}_{i+}$, we obtain $x_4(t) < \dfrac{q-v-1+\alpha}{\sigma}$, so $q - v - 1 + \alpha - \sigma x_4(t) > 0$. As $\alpha \in \Lambda_i$, $q < p$ and $\sigma > \eta$, we have that

$$0 < q - v - 1 + \alpha - \sigma x_4(t) \leq p - 1 + \alpha - \eta x_4(t).$$

On the other hand, $(q-p)x_2(t) + \Theta_q P \geq (q - p + \Theta_q)P \geq 0$. From these non-negative expressions and using the equations of the system (4.1) it follows directly that $x_j(t) \geq 0,\ j = 1,\ 2,\ 3,\ 4,\ t \geq 0$. □

From now on, the expression $\mathcal{C}_{i+} = \dfrac{q-v-1+\alpha}{\Theta_q} K_\alpha$ is called the non-negativity bound for the *vaccination impact model*.

Now, our objective is to obtain the basic reproduction (BR_i) number of this *vaccination impact model* using the spectral radius of the next-generation matrix, [2]. This matrix will be obtained from a linear approximation of the model (4.1) around the disease-free equilibrium point $x^* = (x_1^*\ x_2^*\ x_3^*\ x_4^*)^T$. In this equilibrium point, the variable corresponding to the infection and the variable representing the contaminant must be zero. By definition of the equilibrium point, it is fulfilled that

$$x^* = \tilde{A}_i\ x^* - \sum_{j=2}^{k} a_j^{\alpha} x^* + f_i(x^*) + B\Theta_q P.$$

So, through a direct computation, it is obtained that the disease-free equilibrium point of the model (4.1) is given by

$$x_1^* = \frac{\Theta_p P}{\Theta_p + v}, \qquad x_2^* = \frac{Pv}{\Theta_p + v}, \qquad x_3^* = 0, \qquad x_4^* = 0,$$

for any value $\alpha \in \Lambda_i$ and $k \geq 1$. Next, we are going to consider a linear approximation of the system (4.1) around this disease-free equilibrium point x^* taking $x_1(t)x_4(t) \approx x_1^*\ x_4(t)$ and $x_2(t)x_4(t) \approx x_2^*\ x_4(t)$. Then, we have that

$$\hat{x}(t+1) = E\ \hat{x}(t) - \sum_{j=2}^{k} a_j^{\alpha}\ \hat{x}(t+1-j),$$

where $\hat{x}(t) = x(t) - x^*$ and the coefficient matrix E is given by

$$E = \begin{pmatrix} q - v - 1 + \alpha & q - p & 0 & -\sigma x_1^* \\ v & p - 1 + \alpha & 0 & -\eta x_2^* \\ 0 & 0 & q - 1 + \alpha & \sigma x_1^* + \eta x_2^* \\ 0 & 0 & \beta & s - 1 + \alpha \end{pmatrix}.$$

We can analyze the evolution of this system considering the variables associated with the infected individuals, $\hat{x}_3(t)$ and the contaminant, $\hat{x}_4(t)$. So, we have the following linear subsystem:

$$z(t+1) = \bar{E}\ z(t) - \sum_{j=2}^{k} a_j^{\alpha}\ z(t+1-j),$$

where $z(t) = (\hat{x}_3(t), \hat{x}_4(t))^T$ and $\bar{E} = \begin{pmatrix} q - 1 + \alpha & \sigma x_1^* + \eta x_2^* \\ \beta & s - 1 + \alpha \end{pmatrix}$. As this system is a $k-$delayed linear system we use the equivalent $k-$stacked linear system with the state vector $(z(t)\ z(t-1)\ \ldots\ z(t+1-k))^T$ and the matrix $\mathcal{E}$ given by

$$\mathcal{E} = \begin{pmatrix} \bar{E} & -a_2 I & -a_3 I & \cdots & -a_{k-1} I & -a_k I \\ I & O & O & \cdots & O & O \\ O & I & O & \cdots & O & O \\ \vdots & \vdots & \vdots & & \vdots & \vdots \\ O & O & O & \cdots & I & O \end{pmatrix}.$$

Decomposing the matrix $\bar{E}$ into $\bar{E} = T + F$, being T the diagonal matrix formed by the diagonal of matrix E and $F = \bar{E} - T$, we have $\mathcal{E}$ as the addition of two matrices, $\mathcal{E} = \mathcal{T} + \mathcal{F}$, where $\mathcal{T}$ is called transition matrix and $\mathcal{F}$ is called infection matrix, which are given by

$$\mathcal{T} = \begin{pmatrix} T & -a_2 I & -a_3 I & \cdots & -a_{k-1} I & -a_k I \\ I & O & O & \cdots & O & O \\ O & I & O & \cdots & O & O \\ \vdots & \vdots & \vdots & & \vdots & \vdots \\ O & O & O & \cdots & I & O \end{pmatrix} \text{ and } \mathcal{F} = \begin{pmatrix} F & O & \cdots & O \\ O & O & \cdots & O \\ O & O & \cdots & O \\ \vdots & \vdots & & \vdots \\ O & O & \cdots & O \end{pmatrix}.$$

From these matrices, we construct the next-generation matrix $\mathcal{F}(I-\mathcal{T})^{-1}$ and we define the basic reproduction (BR_i) number of the *vaccination impact model* as the spectral radius of this matrix, that is, $R_{i0}(\alpha, k, v, \eta) = \rho\left(\mathcal{F}(I - \mathcal{T})^{-1}\right)$, and its explicit expression is

$$R_{i0}(\alpha, k, v, \eta) = \sqrt{\frac{\beta(\sigma x_1^* + \eta x_2^*)}{\Theta_q \Theta_s}}. \tag{4.2}$$

Taking into account the expression of $R_{i0}(\alpha, k, v, \eta)$, we can find a relationship with the basic reproduction number associated with the model without vaccination, $R_0(\alpha, k)$. By (2.5) and (4.2) we obtain

$$R_{i0}^2(\alpha, k, v, \eta) = \frac{\beta(\sigma\Theta_p + \eta v)P}{\Theta_q \Theta_s(\Theta_p + v)} = \frac{\beta\sigma P}{\Theta_p \Theta_s} \frac{\Theta_p + \frac{\eta}{\sigma} v}{\Theta_p + v} = R_0^2(\alpha, k) \frac{\Theta_p + \frac{\eta}{\sigma}}{\Theta_p + v}.$$

In the following result we give conditions in order to get $R_{i0}(\alpha, k, v, \eta) < 1$.

Proposition 4. *Consider the model given in (2.3) with $R_0(\alpha, k) > 1$ and the vaccination impact model given in (4.1). If the parameters η and v satisfy that $\eta < \dfrac{\sigma}{R_0^2(\alpha, k)}$ and $v > \Theta_p \dfrac{R_0^2(\alpha, k) - 1}{1 - \frac{\eta}{\sigma} R_0^2(\alpha, k)}$, then $R_{i0}(\alpha, k, v, \eta) < 1$.*

Proof. From the assumptions we have that $\left(1 - \dfrac{\eta}{\sigma} R_0^2(\alpha, k)\right) v > \Theta_p (R_0^2(\alpha, k) - 1) > 0$ and

$$\Theta_p + v > R_0^2\left(\alpha, k)(\Theta_p + \frac{\eta}{\sigma} v\right) \implies 1 > R_0^2(\alpha, k) \frac{\Theta_p + \frac{\eta}{\sigma} v}{\Theta_p + v} = R_{i0}^2(\alpha, k, v, \eta).$$

□

Note that we have established a lower limit for the number of vaccinated individuals,

$$\mathcal{C}_v = \Theta_p \frac{R_0^2(\alpha, k) - 1}{1 - \frac{\eta}{\sigma} R_0^2(\alpha, k)},$$

in such a way, we can get that the infected population tends to disappear by vaccinating a percentage of the population greater than $\mathcal{C}_v$.

Remark 2. Note that using the expression of $R_0(\alpha, k)$ given in (2.5) the restrictions assumed in Proposition 4 are equivalent to $P < \dfrac{\sigma}{\eta}K_\alpha$ and $P < \dfrac{\Theta_p + v}{\Theta_p + \frac{\eta}{\sigma}v}K_\alpha$, respectively. Moreover, $\dfrac{\Theta_p + v}{\Theta_p + \frac{\eta}{\sigma}v} < \dfrac{\sigma}{\eta}$, since $0 < \eta < \sigma$. These conditions, together with the non-negativity bound $\mathcal{C}_{i+}$ given in Proposition 3, allow us to assure that if the size of the population satisfies

$$K_\alpha < P < \min\left\{ \frac{\Theta_p + v}{\Theta_p + \frac{\eta}{\sigma}v}, \frac{q - v - 1 + \alpha}{\Theta_q} \right\} K_\alpha,$$

then the solution of the *vaccination impact model* is non-negative, and the vaccination strategy leads to eradicating the disease.

4.1 Sensitivity analysis

As we have done for the *vaccine effectiveness model*, in this part, we also analyze the sensitivity of the BR_i number of the *vaccination impact model*. So, we use the normalized sensitivity index for a quantity Q with respect to a parameter h, $\Phi(Q/h)$, introduced in (3.5), and we show explicit expressions for the indices. In particular, we analyze how small parameter variations η, v, and α affect the BR_i number of the *vaccination impact model*.

Proposition 5. *Consider the BR_i number of the vaccination impact model (4.1) given in (4.2). Then,*

(i) $\Phi(R_{i0}(\alpha, k, v, \eta)/\eta) = \dfrac{v\eta}{2(v\eta + \sigma\Theta_p)}$;

(ii) $\Phi(R_{i0}(\alpha, k, v, \eta)/v) = \dfrac{v\Theta_p(\eta - \sigma)}{2(v\eta + \sigma\Theta_p)(\Theta_p + v)}$;

(iii) $\Phi(R_{i0}(\alpha, k, v, \eta)/\alpha) = \dfrac{\alpha}{2k!}\left(\dfrac{1}{\Theta_q} + \dfrac{1}{\Theta_s} + \dfrac{1}{\Theta_p + v} - \dfrac{\sigma}{v\eta + \sigma\Theta_p}\right)\displaystyle\sum_{j=1}^{k}\prod_{\substack{i=1 \\ i\neq j}}^{k}(i - \alpha).$

In the next section, we perform some numerical sensitivity analyses, seeing how the values of certain parameters affect the value of the basic reproduction number for both models.

5 Discussion: numerical examples

In this section, we compare the behavior of the different discrete-time fractional order models studied above, we begin with the original DTFO model (2.3).

To carry out our analysis, we assume that the models describe a pig farm where an infectious outbreak is transmitted indirectly by contact or ingestion of the contaminant. To evaluate the parameters of our models, we have used some of the data given in [5] and [6] and the references therein, which correspond to the particular case of a Salmonella infection. Thus, $p = 0.9995$, $q = 0.99$, $s = 0.98$, $\sigma = 0.24 \times 10^{-9}$ Bacteria^{-1}, $\beta = 2.25 \times 10^4$ Bacteria.Indiv^{-1} colony-forming unit (c.f.u.).

5.1 Qualitative behavior of the discrete-time fractional-order models

First, we study the dependence on the fractional derivative order α and the memory steps k of the non-negativity bound $\mathcal{C}_+ = \omega_\alpha K_\alpha$ given in (2.4). If $\omega_\alpha < 1$, the size of the population satisfies $P < \mathcal{C}_+ < K_\alpha$ and we can assure that the trajectory of the system is non-negative and it tends to the disease-free equilibrium point.

Otherwise, if $\omega_\alpha > 1$ and $K_\alpha < P < \mathcal{C}_+$, the disease remains, and we will propose some vaccination actions. In Table 2, for different values of α and k, we show the lower and upper bounds of P such that the trajectory is non-negative, but it does not tend to the disease-free equilibrium point. We have considered values of α and k providing $\omega_\alpha > 1$.

Table 2. Variation of K_α and $\mathcal{C}_+$ in terms of α and k.

k \ α	0.97		0.98		0.99	
2	167.075	6302.22	112.97	5424.81	69.8153	4546.11
4	92.647	4963.43	70.8237	4524.03	52.3824	4091.7
6	72.0675	4506.85	58.6683	4219.15	47.0478	3939.06
8	62.579	4275.67	52.9439	4065.43	44.4681	3862.42
10	57.1399	4135.73	49.6203	3972.65	42.9475	3816.3
	K_α	$\mathcal{C}_+$	K_α	$\mathcal{C}_+$	K_α	$\mathcal{C}_+$

Now, we focus our attention on the worst case. That is, we consider the smallest bound K_α for P, which ensures the disappearance of the disease. We can observe that this occurs when $\alpha = 0.99$ and $k = 10$. From now on, we will consider these values to study both vaccination models.

If the *vaccine effectiveness model* is considered, according to Remark 1, the upper bound to assure that the vaccine allows eradicating the disease is a function of the parameters v and ϵ, which is shown in Table 3.

Table 3. Upper bound on P, in terms of v and ϵ, to assure that the solution of the *vaccine effectiveness model* ($\alpha = 0.99$ and $k = 10$) is non-negative and the vaccination strategy leads to the eradication of the disease.

v \ ϵ	0.2	0.4	0.6
0.564	78	64	55
0.8	119	82	63
0.96	185	101	69

If the *vaccination impact model* is considered, according to Remark 2, the upper bound to assure that the vaccine allows leading the solution of the system toward the disease-free equilibrium point is a function of the parameters v and η, see Table 4.

Table 4. Upper bound on P, in terms of v and η, to assure that the solution of the *vaccination impact model* ($\alpha = 0.99$ and $k = 10$) is non-negative and the vaccination strategy leads to the eradication of the disease.

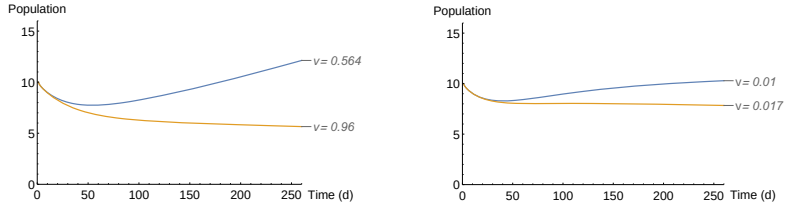
v \ η	6×10^{-11}	7×10^{-11}	8×10^{-11}	$\boxed{9 \times 10^{-11}}$
0.01	122	111	101	93
0.013	130	117	106	97
$\boxed{0.017}$	137	122	110	$\boxed{100}$

Looking at both models, we focus on a population size equal to $P = 100$. If we take a DTFO model for $\alpha = 0.99$ truncated in 10 steps, the disease remains without vaccination since $R_0(0.99, 10) = 1.52$.

On the one hand, in the *vaccine effectiveness model*, we can observe that taking $\epsilon = 0.4$ we have assured that the vaccination strategy leads to the disappearance of the disease since $P = 100 < 101$. It is necessary to vaccinate the 96% of the population in order to get the disease disappears, see Table 3. Note that, in this case, the BR_e number (3.3) is $R_{e0}(0.99, 10, 0.96, 0.4) = 0.99$.

On the other hand, in the *vaccination impact model*, taking $\eta = 9 \times 10^{-11}$, we have that the population size $P = 100$ is equal to the bound given in Table 4. In this case, we only need to vaccinate the 1.7% of the population. Moreover, we have that the BR_i number (4.2) also reduces to the same value, $R_{i0}(0.99, 10, 0.017, 9 \times 10^{-11}) = 0.99$.

Once the values of the parameters indicated in the previous paragraphs have been fixed, we consider a variation in the vaccinated population rate. For instance, we observe a different behavior when reducing v by about 41% in both models. In the *vaccine effectiveness model*, when we reduce from $v = 0.96$ to $v = 0.564$, the new BR_e number is $R_{e0}(0.99, 10, 0.564, 0.4) = 1.241$; while, in the *vaccination impact model*, when we pass from $v = 0.017$ to $v = 0.01$ we have that the new BR_i number is $R_{i0}(0.99, 10, 0.01, 9 \times 10^{-11}) = 1.032$. The same happens with the trajectory of the infected population. Figure 1 shows the evolution of the infected population in both models for both vaccinated population rate. We observe more significant growth of the infected population in the *vaccine effectiveness model* than in the *vaccination impact model*.



(a) *Vaccine effectiveness model* with $\epsilon = 0.4$. Cases: $v = 0.564$ and $v = 0.96$.

(b) *Vaccination impact model* with $\eta = 9 \times 10^{-11}$. Cases: $v = 0.01$ and $v = 0.017$.

Figure 1. Evolution of infected population when the vaccinated population rate is reduced to the same percentage in both fractional-order models with 10 memory steps and $\alpha = 0.99$.

5.2 Sensitivity analysis

Next, we make a sensitivity analysis for the two discrete-time fractional order models developed previously. Depending on the model considered, the most effective method to reduce the BR number will be different. For the *vaccine effectiveness model*, a reduction in BR_e number is achieved with an increase in vaccine effectiveness. In contrast, for the *vaccination impact model*, the BR_i number reduces with a decrease in the exposure rate of vaccinated individuals. In both models, the reduction of the BR number is also achieved by increasing the vaccination rate. In addition, we have also studied the influence of the fractional order, α, of the model on the reduction of the BR number.

Sensitivity analysis: Vaccine effectiveness model

We aim to reduce the BR_e number by about 10% using the new value for every parameter from its corresponding sensitivity index whose expression is given in Proposition 2. In Tables 5–7, the sensitivity index with respect to v, ϵ and α, respectively, and the variation achieved by the BR_e number are shown.

Table 5. Variation of v and R_{e0} from $\Phi(R_{e0}/v)$ in order to get a decrease of the BR_e number by 10% in the *vaccine effectiveness model* with $\alpha = 0.99$, $k = 10$ and $\epsilon = 0.4$.

v	$R_{e0}(0.99, 10, v, 0.4)$	$\Phi(R_{e0}/v)$	$\hat{v}$	$R_{e0}(0.99, 10, \hat{v}, 0.4)$
0.564	1.241	-0.255	0.784	1.11
0.604	1.218	-0.284	0.84	1.074
0.644	1.195	-0.314	0.89	1.037
0.684	1.171	-0.348	0.95	0.999

Table 6. Variation of ϵ and R_{e0} from $\Phi(R_{e0}/\epsilon)$ in order to get a decrease of the BR_e number by 10% in the *vaccine effectiveness model* with $\alpha = 0.99$, $k = 10$ and $v = 0.564$.

ϵ	$R_{e0}(0.99, 10, 0.564, \epsilon)$	$\Phi(R_{e0}/\epsilon)$	$\hat{\epsilon}$	$R_0(0.99, 10, 0.564, \hat{\epsilon})$
0.25	1.159	0.122	0.045	1.036
0.3	1.187	0.139	0.085	1.061
0.35	1.214	0.155	0.125	1.086
0.4	1.241	0.17	0.165	1.11

Table 7. Variation of α and R_{e0} from $\Phi(R_{e0}/\alpha)$ in order to get a decrease of the BR_e number by 10% in the *vaccine effectiveness model* with $k = 10$, $v = 0.564$ and $\epsilon = 0.4$.

α	$R_{e0}(\alpha, 10, 0.564, 0.4)$	$\Phi(R_{e0}/\alpha)$	$\hat{\alpha}$	$R_{e0}(\hat{\alpha}, 10, 0.564, 0.4)$
0.99	1.241	7.236	0.976	1.124
0.996	1.297	7.391	0.982	1.173
1.	1.336	7.5	0.986	1.207

We consider the *vaccine effectiveness model* with 10 memory steps and $\alpha = 0.99$, $v = 0.564$ and $\epsilon = 0.4$. With these values of the parameters, the disease remains since the corresponding BR_e is greater than 1. In Figure 2, the evolution of the infected population is shown when one of these parameters is changed according to the new value proposed in Tables 5-6-7.

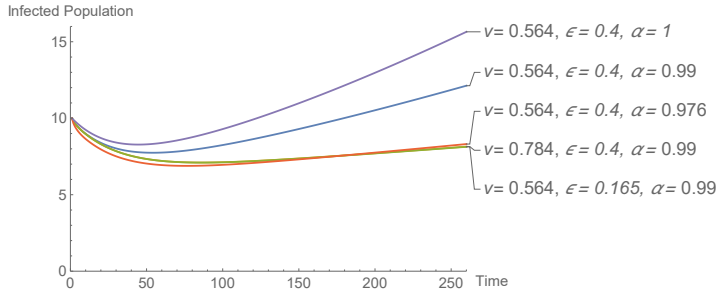


Figure 2. *Vaccine effectiveness model*: Evolution of infected population in the different cases provided from the sensitivity analysis.

It is observed that the new values of the parameters provide similar decreasing evolution of the infected population. The initial BR_e number,

$$R_{e0}(0.99, 10, 0.564, 0.4) = 1.241,$$

transforms into new BR_e numbers, given by

$$\begin{aligned} R_{e0}(0.99, 10, 0.784, 0.4) &= 1.11, \\ R_{e0}(0.99, 10, 0.564, 0.165) &= 1.11, \\ R_{e0}(0.976, 10, 0.564, 0.4) &= 1.124, \end{aligned}$$

depending on the parameter, we vary. In the three cases, the disease still remains, but we have reduced the BR_e number.

Sensitivity analysis: Vaccination impact model

Analogously to the above case, we propose to reduce the BR_i number by about 10% varying one of the parameters. The variation of the BR_i number when the parameters v, η, and α are changed according to its corresponding sensitivity index, given in Proposition 5, is shown in Tables 8–10.

Table 8. Variation of v and R_{i0} from $\Phi(R_{i0}/v)$ in order to get a decrease of the BR_i number by 10% in the *vaccination impact model* with $\alpha = 0.99$, $k = 10$ and $\eta = 9 \times 10^{-11}$.

v	$R_{i0}(0.99, 10, v, 9 \times 10^{-11})$	$\Phi(R_{i0}/v)$	$\hat{v}$	$R_{i0}(0.99, 10, \hat{v}, 9 \times 10^{-11})$
0.01	1.032	-0.078	0.023	0.982
0.013	1.013	-0.067	0.029	0.972
0.015	1.004	-0.061	0.034	0.967
0.017	0.997	-0.055	0.038	0.963

Table 9. Variation of η and R_{i0} from $\Phi(R_{i0}/\eta)$ in order to get a decrease of the BR_i number by 10% in the *vaccination impact model* with $\alpha = 0.99$, $k = 10$ and $v = 0.01$.

η	$R_{i0}(0.99, 10, 0.01, \eta)$	$\Phi(R_{i0}/\eta)$	$\hat{\eta}$	$R_{i0}(0.99, 10, 0.01, \hat{\eta})$
7×10^{-11}	0.947	0.328	5.03×10^{-11}	0.855
8×10^{-11}	0.99	0.342	5.74×10^{-11}	0.89
$\boxed{9 \times 10^{-11}}$	1.032	0.355	$\boxed{6.466 \times 10^{-11}}$	0.923

Table 10. Variation of α and R_{i0} from $\Phi(R_{i0}/\alpha)$ in order to get a decrease of the BR_i number by 10% in the *vaccination impact model* with $k = 10$, $v = 0.01$ and $\eta = 9 \times 10^{-11}$.

α	$R_{i0}(\alpha, 10, 0.01, 9 \times 10^{-11})$	$\Phi(R_{i0}/\alpha)$	$\hat{\alpha}$	$R_{i0}(\hat{\alpha}, 10, 0.01, 9 \times 10^{-11})$
$\boxed{0.99}$	1.032	1.859	$\boxed{0.936}$	0.854
0.996	1.041	1.119	0.907	0.743
1.	1.045	0.497	0.798	0.437

Now, we consider the *vaccination impact model* with $k = 10$ steps of memory and parameters: $\alpha = 0.99$, $v = 0.01$ and $\eta = 9 \times 10^{-11}$ and we fix our attention on the new values of these parameters shown in Tables 8–10. In Figure 3, the evolution of the infected population in each one of these cases is plotted.

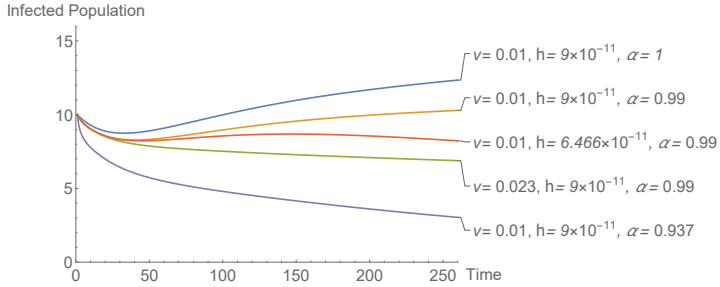


Figure 3. *Vaccination impact model*: Evolution of infected population in the different cases provided from the sensitivity analysis.

It is observed that the new values of the parameters provide different decreasing evolution of the infected population. In fact, from the initial BR_i number $R_{i0}(0.99, 10, 0.01, 9 \times 10^{-11}) = 1.032$, the new BR_i numbers obtained are

$$\begin{aligned} R_{i0}(0.99, 10, 0.023, 9 \times 10^{-11}) &= 0.982, \\ R_{i0}(0.99, 10, 0.1, 6.466 \times 10^{-11}) &= 0.923, \\ R_{i0}(0.936, 10, 0.01, 9 \times 10^{-11}) &= 0.854. \end{aligned}$$

With these initial values of the parameters, the disease tends to be eradicated since, in the three cases, we get the BR_i number less than 1. Here, we also

observe that the reduction of the BR_i number is more significant when the parameter α is modified.

6 Conclusions

Although mathematical models provide critical information about infectious diseases, these models could be more robust in analyzing the advantages of using certain control measures and the effects that small variations in model parameters can have on the extinction of an infection.

In this work, we focus on vaccination as a control action on an epidemic model, and we have considered the study of the sensitivity of the value of the basic reproduction number with respect to certain parameters. For that, we have applied a vaccination control strategy to a fractional-order discrete-time SIC epidemic model and consider how vaccination affects disease transmission in two scenarios: for a *vaccine effectiveness model* and for a *vaccination impact model*. We have done a sensitivity analysis for the BR number with respect to some parameters. The structure followed in both cases is similar. First, upper bounds have been determined to ensure the biological meaning, that is, the non-negativity of the solution. Lower bounds have also been sought to determine the vaccinated population rate that ensures the eradication of the disease, obtaining information on the size of the population that ensures this behavior. This fact is important when taking into account the economic factor in the use of certain control. In addition, it has been studied how the variation of certain parameters affects the value of the basic reproduction number, obtaining an explicit expression for the sensitivity indices. This analysis allows us to use the data to mark the increase or decrease in the considered parameters to reduce the value of the basic reproduction number.

Finally, we have illustrated the theoretical results using our models applied to a pig farm. In the *vaccine effectiveness model* analyzed in this example, we observe that the dependence of the BR_e number with respect to each one of the parameters (v, ϵ, and α) is very similar. It has been reduced approximately to the 10% target in all three cases.

In the *vaccination impact model*, the dependence of the BR_i number with respect to each parameter is not the same. Specifically, the sensitivity index associated with α provides a more significant reduction of the BR_i number than the index associated with v and η.

As future work, more general models can be considered with discrete fractional order operators, which include recovered individuals or even that the disease transmission is directly from infected individuals and indirectly by the contaminant. Also, different discrete fractional order operators, as Caputo's or Riemann-Liouville ones, can be considered for a SIC model.

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