A unified model for wild resistance dynamics and weed control using herbicide

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Abstract

A major issue in the modelling of weed resistance to herbicide lies in effectively handling the wild dynamics, that is, the allele frequency prior to the herbicide application, and in particular when starting to use the herbicide. The wild allele frequency is a key variable as the resistance evolution is highly sensitive to it, moreover it is extremely difficult to measure in the agricultural field. In this paper we propose a model for weed control that handles the allele frequency in a direct manner, grounded on the very dynamics of the weed life cycle, with no need of a priori distributions nor the use of the Hardy-Weinberg equilibrium. The proposed model is individual based, stochastic, and considers some phenomena like the relative fitnesses and mutation that are prominent in the resistance dynamics without herbicide. A case study is presented for the herbicide nicosulfuron in a field with the weed \textit{Bidens pilosa}. Another two models having a standard deterministic dynamics are compared with ours in terms of the initial allele frequency, its time evolution, and the resistance visualization in the field, indicating that the proposed model is effective to provide more realistic simulations for the weed resistance.

Keywords: stochastic model, individual-based model, herbicide resistance, allele frequency dynamics, weed control.

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1. Introduction

Weed resistance to herbicide is a key issue in food production as it promotes efficiency loss of herbicide, one of the most worldwide used strategy to control weeds [1]. Due to the difficulty and high cost in the development of new pesticides, much effort has been made in understanding the multiple factors surrounding this phenomenon. In this context, it is well recognized that computer simulation modelling can help understanding the relationship between the use of herbicide and its resistance evolution [2]. During the last decades many models have been proposed to describe and predict the herbicide resistance evolution [2, 3].

The available dynamic models are, to our knowledge, concerned with the resistance evolution in short term, typically in a time interval \([0, T]\), being the order of magnitude of \(T\) less than 2. A notorious difficulty that arises when dealing with these models is to accurately estimate the resistant allele frequency at \(t = 0\), and in a more general context, the allele frequency dynamics for pre-herbicide application periods, which we refer to as the \textit{wild resistant allele frequency dynamics}, or \textit{wild frequency} for short. According to [4], the wild frequency is critical for studying the resistance risk. Similar discussions were also presented in [5, 6]. Most of simulation results assume this quantity as deterministic, see e.g. [6, 7, 8, 9, 10, 11, 12]. However, as the natural evolution of resistance is mainly driven by both mutation and natural selection (specifically, the relative fitnesses between resistant and susceptible individuals), two phenomena that are largely known as random ones, it is important to consider the wild frequency as a random variable, as in [4, 5, 13, 14]. The main difficulty in this case is to estimate its probability distribution, once it is extremely hard to measure this variable in agricultural fields to collect the necessary data [2, 3, 8, 11].

In this paper we propose a stochastic and individual-based model for the resistance evolution that handles the wild frequency issue in a simple, unified manner. In fact, we use a single dynamic model in a large time interval \([-T_0, T]\) with \(T_0, T > 0\), and \(T_0\) large enough such that the initial condition at time \(t = -T_0\) has almost no relation with the seed bank state at \(t = 0\), when the application of herbicide starts. The main benefit in using this strategy is that there is no need to define a priori distribution for the wild resistant allele frequency and also for the use of the Hardy-Weinberg equilibrium [15, Chapter 3]. Using the model in the interval \([-T_0, 0]\) it is also possible to estimate the distribution of wild frequency, called \textit{wild}
distribution, as performed in Section 3, see Figure 3. Moreover, the model allows us to study the allele frequency distributions for the post-herbicide application period \([0, T]\), also performed in Section 3.

The proposed model is based on the life cycle of a weed population considering a post-emergent herbicide \([16,17]\), involving the germination in soil, emergence, herbicide application, flowering, seed production, mortality of seeds in soil and again germination. We employ a dose response function obtained from greenhouse experiments, relating the dose response of susceptible and resistant *Bidens pilosa* to the application of the nicosulfuron herbicide. The models also allow to emulate competition with other species and other natural factors that prevent a booming population, in a simple way, which can be interpreted as if an “alternative herbicide” were employed for \(t < 0\). The stochastic model employs binomial distributions \([18]\) in the most random processes involved in the life cycle of weeds, including the mutation phenomena and relative fitnesses. It is worth noting that the use of binomial distributions is suggested by \([2]\) and is also employed in \([11]\). A contribution of this paper is to combine all the above ingredients in a single model, which involved integration of existing equations and development of some new ones, such as the stochastic reproduction equation in \([3]\), and to present the complete equations in a detailed manner.

Another contribution is to compare the proposed model with a deterministic model and also with a simple extension of it, which we refer to as the hybrid model, defined by a deterministic dynamics for \(t \geq 0\) and a random initial condition at \(t = 0\). The motivation behind the deterministic model lies in the fact that it is able to estimate averages of the main variables, has low computational burden and is very popular \([2]\), so it is important to assess the quality of its estimates. As for the hybrid model, we check how the deterministic dynamics act on the random initial condition, by simulating the distributions and comparing with the ones given by the stochastic model. The simulation results presented here focus on the resistance risk in terms of visualization of resistance in the field, and also on the evolution of the wild distribution when the application of herbicide starts.

The text is organized as follows. In Section 2 we present the stochastic, hybrid and deterministic models. In Section 3 we present estimates for the wild frequency and for the allele distributions in the post herbicide application period. We also present a comparison between the models in terms of resistance visualization index. At the end of this section we carry out a discussion about the results. Finally, some conclusion are made in Section 5.
Throughout the paper capital letters indicate a vector or some of its elements and lowercase letters indicate scalar variables or parameters. We reserve boldface letters for stochastic variables and standard letters to deterministic ones. The symbols $N$ and $\mathbb{R}$ represent the set of natural and real numbers, respectively. We use $E\{\cdot\}$ for the expected value of a stochastic variable, $\mathcal{B}(n, p)$ for a binomial distribution \cite{18} with $n$ being the number of experiments and $p$ its probability of success; $\mathcal{B}^M(n, P)$ is employed for the multinomial distribution \cite{18}, with $n$ already been defined and $P$ the probability vector, and $\mathcal{N}(m, s^2)$ for the normal distribution \cite{18} with $m$ being the mean and $s$ the standard deviation.

2. Weed population models

The considered models are based on the life cycle of a weed population and post-emergent herbicides. We assume that the population is closed, that is, there is no gene exchange from surrounding agricultural fields. The Figure 1 presents all variables employed by the models and indicates how they are related to each other. Note that the time sequence is indexed to the weed life cycle. We focus on the resistance evolution of a certain herbicide referred to as the target herbicide, denoted $u_t$. It is worth noting that a second herbicide is included in the models, denoted $v_t$, providing more flexibility, for instance, allowing to simulate the use of other herbicides or even emulate natural population control; we refer to this herbicide as the alternate herbicide, and assume it causes no selective pressure regarding resistance to the target one.

2.1. Stochastic model

Following papers in weed resistance such as \cite{8, 9, 13}, we suppose that the weed resistance to the target herbicide is associated to a gene arranged at a single genetic locus. We also suppose that the weed resistance is due to dominant alleles which are denoted by the capital letter $A$. Therefore, the list of all possible genotypes in the weed population is given by $\mathcal{G} = \{AA, Aa, aa\}$. For each genotype, we denote the quantity of seeds in the seed bank at instant $t$ as $\mathbf{X}_t = [X_{1,t} \quad X_{2,t} \quad X_{3,t}]'$, where $\mathbf{X}_t \in \mathbb{N}^3$ and $X_{i,t}$ the amount the $i$-th genotype seed in list $\mathcal{G}$. As an example, $X_{2,5}$ is the amount of seed type $Aa$ at instant 5.
Figure 1: Flow chart of weed life cycle considered by the models. The disks indicate the employed variables. The value or distribution of the variables at the head of an arrow is a function of the variables at the tail. The green disks highlight the state variables in two consecutive cycles, \(t\) and \(t+1\).
Assuming that each seed in the area has an uniform probability in time to germinate, referred here as constant probability, the amount of seedling is a random variable with the following probability,

\[ Y_{i,t}^{\delta} \sim B(X_{i,t}, \delta) \]  

where \( Y_{i,t}^{\delta} \in \mathbb{N} \) is the amount of seed of genotype \( i \) germinated in the \( t-th \) cycle and \( \delta \) is the germination probability of each seed. We mention that parameter uncertainty and time variation can be incorporated in the model, e.g. by considering \( \delta \) as a time varying parameter in the above probability distribution.

Assuming that the number of mature weeds remaining from the previous cycle is negligible\(^1\), we have that the total of mature weeds observed at time instant \( t \), denoted by \( Y^a_t \in \mathbb{N} \), obeys the following distribution,

\[ Y^a_{i,t} \sim B \left( Y_{i,t}^{\delta}, \xi_i M_i(u_t, v_t) \right), \]  

where \( \xi_i M_i(u_t, v_t) \) is the probability that a germinated weed survives until the mature stage, addressed as follows. We assume that the herbicides are not employed together in a same cycle, so that the survival probability to the herbicide can be modelled by the functions \( M_i(u, v) = (1 - \rho^R(u))(1 - \rho(v)) \), for \( i = 1, 2 \) and \( M_3(u, v) = (1 - \rho^S(u))(1 - \rho(v)) \), where \( \rho^R(u) \) and \( \rho^S(u) \) are the expected mortality of resistant and susceptible seedlings, respectively, due to the target herbicide application, and \( \rho(v) \) is the expected mortality due to the alternative herbicide. Note that the uses of \( v \) doesn’t cause selective pressure. Now, to account for the natural mortality we define \( \xi_i = \gamma(1 - c) \), for \( i = 1, 2 \) and \( \xi_3 = \gamma \), where \( \gamma \) is the natural survival of weeds and \( c \) is the fitness cost parameter, considered in several papers as in [19, 20, 21]. We have included the fitness cost because there is usually a metabolic penalty associated with the resistance to a certain herbicide [20], leading to decrease in the reproductive success.

The total generated seeds can be obtained by \( y^a_t x_{t}^{spp} \), where \( y^a_t \) is the number of total adult plants, that is, \( y^a_t = \sum_{i=1}^{3} Y^a_{i,t} \), and \( x^{spp}_t \in \mathbb{R} \) is the mean of seeds produced per plant at \( t \). Since we do not have the probability

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\(^1\)This is valid for instance when the production cycle is sufficiently short so that there is only one generation of mature weed per cycle. If this hypothesis is void, the remaining weeds can be added to \( Y^a_{i,t} \) in (2).
distribution for \( x_t^{\text{spp}} \in \mathbb{R} \) (and have found no literature on this subject), we adopt a normal distribution
\[
y_t^a x_t^{\text{spp}} \sim N \left( y_t^a x_t^{\text{spp}}, (\varphi y_t^a x_t^{\text{spp}})^2 \right),
\]
where \( x_t^{\text{spp}} := \mathcal{E} \{ x_t^{\text{spp}} | y_t^a \} \) and \( \varphi \) is a parameter to adjust the standard deviation of the distribution. To calculate \( x_t^{\text{spp}} \) we use the following expression,
\[
x_t^{\text{spp}} = \begin{cases} \frac{F_g}{F_g + g} & : y_t^a > 0 \\ 0 & : y_t^a = 0 \end{cases},
\]
where \( F \) is the agriculture field area in square meters, and \( g \) and \( G \) are bounds related to \( x_t^{\text{spp}} \) in the sense that \( x_t^{\text{spp}} \) converges to \( g \) when \( y_t^a \to 0 \) and \( \frac{y_t^a x_t^{\text{spp}}}{F} \) converges to \( G \) when \( y_t^a \to \infty \). Equation (3) was adapted from [5, 10], and takes into account the intra-specific competition in such a manner that \( x_t^{\text{spp}} \) decreases with the increase of the adult plants. It is worth to highlight that in the cited papers the equation is slightly different, its output is the amount of seeds produced per square meter. Now, rounding down the values generated by the normal distribution, we obtain the total produced seeds as \( x_t^r = \lfloor y_t^a x_t^{\text{spp}} \rfloor \). Also, considering that each seed has a probability \( \kappa \) of becoming a viable seed, we get
\[
x_t^\kappa \sim B \left( x_t^r, \kappa \right),
\]
where \( x_t^\kappa \in \mathbb{N} \) is the total viable seeds produced. Finally, we determine the number of viable seeds of each genotype, denoted by \( X_i^g \in \mathbb{N}^3 \), employing the following distribution,
\[
X_i^g \sim B^M \left( x_i^\kappa, P(G_i) \right),
\]
where \( P(G_i) = [P(G_{1,i}) \, P(G_{2,i}) \, P(G_{3,i})] \in \mathbb{R}^3 \), with \( P(G_{i,t}) \) being the probability of a generated seed to have genotype \( i \). The calculation of \( P(G_{i,t}) \), presented in Appendix B, depends on the quantity of mature plant of each genotype \( Y_i^{a,t} \), on the probability of mutation in the gametogenesis, denoted \( P(m) \), and on the probabilities of a seed be generated by self or of cross-fertilization, denoted \( P(F_a) \) and \( P(\bar{F}_a) \), respectively.

To model the mortality of dormant seeds we use the following distribution,
\[
X_i^{\psi,t} \sim B \left( X_{i,t} - Y_i^{a,t}, \psi \right).
\]
where $\psi$ is the seed death probability. Finally, using balance of seeds, we obtain the characterization of the seed bank for all $t \geq t_0$ using the following recursive equation,

$$
\begin{align*}
X_{i,t+1} &= X_{i,t} - Y_{i,t}^\delta - X_{i,t}^\psi + X_{i,t}^g, \\
X_{i,t_0} &= \ddot{X}_i,
\end{align*}
$$

(8)

where $t_0$ is a predetermined starting time and $\ddot{X}_i$ is a stochastic initial condition for the seed bank.

### 2.2. Deterministic model

In this section, we describe the deterministic model. This model takes into account the same stages of weed life cycle considered for the stochastic model. For conciseness, we present the equations directly, as following

$$
\begin{align*}
Y_{i,t}^\delta &= \delta X_{i,t}, \\
Y_{i,t}^a &= \xi_i M_i(u_t, v_t)Y_{i,t}^\delta, \\
x_{t}^{\text{supp}} &= \begin{cases} 
\frac{F_g}{F+gy_t^a/G} & : y_t^a > 0, \\
0 & : y_t^a = 0, 
\end{cases} \\
x_t^r &= y_t^a x_{t}^{\text{supp}}, \\
x_t^\kappa &= \kappa x_t^r, \\
X_t^g &= x_t^\kappa P(G_t), \\
X_{i,t}^\psi &= \psi \left( X_{i,t} - Y_{i,t}^\delta \right).
\end{align*}
$$

(9) (10) (11) (12) (13) (14) (15)

Using the seed balance, we obtain

$$
\begin{align*}
\begin{cases} 
X_{i,t+1} &= (1 - \delta - \psi + \psi \delta) X_{i,t} + X_{i,t}^g, \\
X_{i,t_0} &= \ddot{X}_i,
\end{cases}
\end{align*}
$$

(16)

where $\ddot{X}_i$ is a deterministic initial condition for the seed bank.

The stochastic and deterministic models are similar only in terms of number of variables and equations, as they are derived from the same life cycle stages of a weed population; the parameter $\varphi$, in Equation (3), is the only additional one in the stochastic model. Apart from this aspect, the models are quite different. One of the major difference lies in the domains of variables. The state variables of the stochastic model take values in the set of integer
numbers, which seems more realistic than the real variables of the deterministic model. Also, the interpretation of some parameters is different; e.g. $\delta$ is the probability that a seed in the bank germinate at a cycle in the stochastic model, while it is the fraction of seeds that germinate in the deterministic one, leading to a exact (real) number of $\delta X_{i,t}$ germinated seeds, which seems less consistent with the real-world application. Of particular relevance is the case, when the number of seeds is small, as frequently found when handling small areas, the number of seeds of resistant genotype turns out to be smaller than in the deterministic model. These fractions of seeds propagate in this model as if they were able to germinate and produce new plants.

It is worth to note that the deterministic equations cannot be obtained by taking expectation of the stochastic ones. In fact, as the expectation does not commute with some operators, we have that the expectation does not “move freely” between equations and inside them. To illustrate, while in the deterministic model we assumed that $x_t^r = y_t^a x_{t}^{SPP}$, from (3), we find

$$E\{y_t^a x_t^{SPP}\} \neq E\{y_t^a\} E\{x_t^{SPP}\},$$

(17)
because the number of mature weeds and mean of seeds produced per plant are correlated.

2.3. Hybrid model

Simplicity is one of the main features of the deterministic model, perhaps explaining its popularity in the weed resistance literature. On the other hand, the simplicity affects the comparisons with the stochastic model; for instance, the distribution of the allele frequency is trivial (concentrated in a point) making superficial the comparison with the relatively complex distribution given by the stochastic model (see e.g. Figure 3). With this motivation, we introduce a third model, which we refer to as the hybrid model, which preserves the deterministic dynamic, (9) - (15), but with a random initial condition

$$\begin{cases} X_{i,t+1} = (1 - \delta - \psi + \psi \delta) X_{i,t} + X_{i,t}^g, & \text{for } t \geq t_s \\ X_{i,t_s} = \bar{X}_i, \end{cases}$$

(18)

where $t_s$ is the starting time and $\bar{X}_i$ is a stochastic initial condition.
3. Simulation results

We start with the simulation results for the pre target herbicide application period, \([-T_0, 0]\], with emphasis on the wild frequency and its distribution according to the stochastic model. Then we turn our attention to the herbicide application period \([0, T]\). Recall that the application of target herbicide is zero \((u_t = 0)\) in the interval \(-T_0 \leq t \leq -1\), and the alternative herbicide is applied at fixed doses \(v_t = 60 \text{ g ha}^{-1}\) (please, see Appendix C for motivation) to emulate the usage of any other herbicide, competition with other species and other factors preventing a booming weed infestation prior to the first usage of the target herbicide. Conversely, we set \(v_t = 0\) and \(u_t = 60 \text{ g ha}^{-1}\) for \(0 \leq t \leq T\).

The parameters adopted in all simulations are given in Appendix C. We denote the seed trajectory generated by the deterministic model by \(\{X_t, -T_0 \leq t \leq T\}\) and, for the \(k\)-th realization of a Monte Carlo simulation we denote the seed trajectories of the stochastic and hybrid models by \(\{X^\omega_k, -T_0 \leq t \leq T\}\) and \(\{X^\star_k, 0 \leq t \leq T\}\), respectively. The Monte Carlo simulation was performed with \(10^4\) realizations. The initial seed bank (at \(t = -T_0\)) for the stochastic model is set to 2200 seeds \(m^{-2}\), with no resistant seeds. We recall that we set the initial allele frequency of hybrid model using the stochastic model outputs, \(\{X^\omega_0 = X^\star_0, 1 \leq k \leq 10^4\}\). It made possible to compare the stochastic and hybrid model output on the evolution and visualization of resistance for the post herbicide application period.

Pre target herbicide application period and wild distribution

Trajectories of the resistant allele frequency and the seed bank have been computed based on \(X^\omega_k\) and \(X_t\) using the equations given in Appendix A and they are illustrated in Figure 1. We plot only the estimated average and standard deviation linked with the stochastic model as plotting \(10^4\) realizations is useless. The allele frequency estimated by the Mutation-Selection Theory, given by \(P(m)/c\) ([13, Chapter 4]), is also included in Figure 1. Note that \(\mu_{X_t}\) and \(\pi_{X_t}\) at \(t = 0\) are close to \(P(m)/c\), as expected. These results strongly suggest that the stochastic model is stable, as unstable dynamics usually leads to divergence of the average or the standard deviation.
Figure 2: Allele frequency and seed bank given by the deterministic model (red line), the average and standard deviation given by the stochastic model (solid green and dashed green, respectively). The allele frequency given by the Mutation-Selection Theory is expressed by blue line.

From the same simulation results we have estimated the histogram of allele frequency at time $t = 0$, as illustrated on the left of Figure 3. Note that the number of occurrences of null resistant alleles is displayed aside, on the left of the plot. This histogram give us an estimation for the wild distribution, and indicate a high probability of finding no resistant allele in the considered field. For comparison purposes, we also plot on the right of Figure 3 an histogram obtained from the log-normal distribution considered in [4, 5, 14]. To make the comparison meaningful we keep the same mean obtained from the stochastic model (around $3.05 \times 10^{-8}$) and the standard deviation as in [4, 5, 14], set as $10^{-7}$, and we also employ the same upper bound frequency given by the stochastic model to calculate the null resistant alleles probability, that is, we set $P(\mu_0 = 0) = P(\mu_0 \leq 6.58 \times 10^{-10})$ for the histogram of the log-normal distribution.

**Target herbicide application period**

Figure 4 shows some resistant allele frequency and seed bank trajectories. In each figure we plot the deterministic trajectory and the stochastic and hybrid models trajectories associated to the realizations $k = 1, \ldots, 50$. Every trajectory has an initial “phase” where it remains close to zero, and a second one in which it quickly gets far from zero, referred as the *resistance emergence*. 
period. We say that this period starts at the emergence time. Figure 4 indicates that the emergence time is rather variable for the stochastic model, or more precisely, it is a random variable with large variance. We note a smaller variance according to the hybrid model, and the simulations suggest that the emergence time is mostly constrained to a bounded time interval $t \in [20 \ 30]$.

Distributions of the resistant allele frequencies estimated by the stochastic and hybrid models for $t = 1, 5, 10, 20, 50, 100$ are shown in Figures 5 and 6, respectively. These figures illustrate how both models predict the evolution of the wild distribution (Figure 3) along the time. It is interesting to note that for $t \geq 5$, every histogram of the stochastic model (Figure 5) shows a portion of low occurrences around the interval $[10^{-8} \ 10^{-7}]$, which we refer to as the few occurrences region (FOR). We also note to the left of the FOR, around $[10^{-9} \ 10^{-8}]$, a region with relatively many occurrences, which we call left peak for short. We believe that the left peaks are maintained almost exclusively by the mutation phenomenon (a $\rightarrow$ A), and not by seeds generated by the reproduction of resistant mature plants. Note that the bar corresponding to the null resistant allele occurrences remains high even after one hundred cycles of herbicide application. Note also that for $t \geq 20$ the deterministic model outputs differs considerably from the expected value calculated from the stochastic model. Regarding the hybrid model (Figure 6) we see how the deterministic dynamics after $t = 0$ modifies the wild distribution: it constantly shifts the distribution to the right towards the bound $\mu = 10^0$. 

![Figure 3: Allele frequency obtained by the stochastic model at $t = 0$ (on the left) and by a log-normal distribution (on the right); histograms (green bar) and averages (green triangle). The output of deterministic model is also displayed (red cross).](image)
Figure 4: Allele frequency and seed bank trajectories given by the deterministic (red line), stochastic (green lines) and hybrid (yellow lines) models.

We have obtained the resistance visualization from the simulated trajectories $X_t$, $\bar{X}_t$ and $X_t$ according to (A.2). The accumulated number of occurrences of weed visualization (A.3) illustrated in Figure 7 shows a booming visualization at time $t = 14$ and similar results in the interval $15 \leq t \leq 26$ using the hybrid and stochastic models. From $t = 27$ on the occurrence estimated by the hybrid model increases considerably faster than the one given by the stochastic model, such that at $t = 32$ we have $f^*_X = 10^4$, i.e., we have visualization of resistance in all realizations. The deterministic model exhibits a rather simple behaviour: there is no visualization of resistance prior to $t = 24$, and 100% of visualization at $t = 24$.

4. Discussion

The stochastic model takes advantage of the inherent stability of the dynamics to weaken the dependence of the simulation results for $t \geq 0$ on the initial guess for the allele frequency at time $-T_0$; this allowed us to use the strategy of setting $-T_0$ in a distant past and to overcome the difficulty in finding a meaningful distribution for $\mu_0$. It is unfruitful, for instance, to use the same strategy for the deterministic model, as the distribution of $\mu_0$ turns out to be very concentrated around the value given by the Mutation-Selection Theory.
Figure 5: Histogram of allele frequency obtained from the stochastic model for $t = 1, 5, 10, 20, 50, 100$. The green triangles indicate the average of distributions. The red crosses give the values obtained from the deterministic model.
Figure 6: Histogram of allele frequency obtained from the hybrid model for $t = 1, 5, 10, 20, 50, 100$. The yellow triangles indicate the average of distributions. The red crosses give the values obtained from the deterministic model.
The distribution of $\mu_0$ obtained with our model and the log-normal distribution given in Figure 2 disagree. Of particular relevance is that the probability of having zero resistant alleles is small in the log-normal model, around 5%, versus approximately 55% in our model. We believe that the latter is better suited to the considered experiment, which consists of a field of small area $F = 20$ha (the smaller is the area, the larger is the probability of zero resistant allele) and having no direct nor indirect effect of the target herbicide e.g. via seed interchange with other areas subjected to herbicide.

Turning our attention to the comparison between the stochastic and the hybrid models (recall $\mu_0$ is identical in both models), we consider the period of target herbicide application $t \geq 0$. The hybrid model indicates the existence of deterministic bounds to the emergence time and visualization index (in fact, $14 \leq t_X \leq 32$ for all realizations), which seems implausible. A possible way around this is to “introduce randomness” in the parameters of the hybrid model, similar to [4, 5, 13, 14]. Even so, we have noted that the allele distribution given by the hybrid model evolves in a rather simple fashion along the time (see Figure 6), in such a manner that the variance remains close to a fixed value for a large time horizon, while it changes dynamically in the stochastic model in a fashion that cannot be reproduced by the hybrid model. As for the deterministic model, we note that it is more severely affected by a lack of randomness, moreover its estimates for the average
resistant allele frequency coincide with the stochastic model in a portion of the time interval only (they do not agree in the last three plots of Figure 5).

5. Conclusions

We have proposed a stochastic model for weed control and wild resistance dynamics, which can be used for estimating the wild resistant allele frequency as well as its evolution along time, which plays a fundamental role in the weed control. The model is individual based, stochastic, and considers some phenomena like the relative fitnesses and mutation that are prominent in the wild resistance dynamics.

The proposed model brings new insights on the resistance evolution along time as presented in Section 3. The stochastic model features the largest, dynamic variability of the resistant allele frequency estimates and their distributions among all considered models, as discussed in more detail in Section 4. The visualization index as defined in (A.2) presents an abrupt behaviour along time when using the other models, whereas it is smooth according our model, which seems more adherent to the case study considered in the simulations. This is a relevant index as it may be easily observed in the real-world, allowing to test the proposed model in future work.

Appendix A. Auxiliary equations and weed model parameters

Appendix A.1. Allele frequency and seed bank - $\mu, \eta$

The frequency of allele $A$ is calculated using

$$
\mu(Z_t) = \frac{2Z_{1,t} + Z_{2,t}}{\eta(Z_t)},
$$

(A.1)

for $Z_t \in \mathbb{R}^3$, where $\eta(Z_t) = Z_{1,t} + Z_{2,t} + Z_{3,t}$. Note that $\mu(X_t)$ gives the frequency of seed bank, $\mu(Y_t^a)$ the frequency of mature plants and $\eta(X_t)$ the total amount of seeds at seed bank at time instant $t$.

We define below the mean ($\mu_{X_t}$) and standard deviation ($\tilde{\mu}_{X_t}$) for the allele frequency generated by the stochastic model simulation. We use anal-
ogous equations for the seed bank, defined by $\overline{\eta}_{X_t}$ and $\widehat{\eta}_{X_t}$.

$$\overline{\mu}_{X_t} = N^{-1} \sum_{k=1}^{N} \mu(X_{t}^{\omega_k}),$$

$$\widehat{\mu}_{X_t} = \overline{\mu}_{X_t} + \sqrt{N^{-1} \sum_{k=1}^{N} \left( \mu(X_{t}^{\omega_k}) - \overline{\mu}_{X_t} \right)^2}.$$ 

Appendix A.2. Resistance visualization - $t_X, f^t_X$

We assume that when the number of resistant individuals is greater than 30% of total mature plants, the resistance can be visualized in the field [22]. So, we define the first time of resistance visualization index by

$$t_X = \min_{0 \leq t \leq T} \{ t : J(Y_t^a) = 1 \}, \quad (A.2)$$

where $J(Y) = \text{sgn} \left\{ \frac{Y_1 + Y_2}{\eta(Y)} - 0.3 \right\}$, with $\text{sgn}$ being the signal function, defined as

$$\text{sgn}(x) = \begin{cases} 1, & \text{when } x \geq 0 \\ 0, & \text{otherwise.} \end{cases}$$

Hence, the accumulated occurrence of resistance visualization can be calculated as following. For instance, for the stochastic model, we have

$$f^t_X = \sum_{k=1}^{N} 1_{t \geq t_X}, \quad (A.3)$$

where $1_{\Omega}$ is the indicator function (that is 1 if $\Omega$ is true and 0 if it is false). Analogous equations are used to obtain the accumulated occurrences hybrid ($f^t_X$) and deterministic ($f^t_X$) processes.

Appendix B. Breeding seeds genotype probability - $P(G_i)$

To obtain the genotype of the seeds produced by breeding, equation 6, we need to calculate the probability that a generated seed has genotype $i$, denoted by $P(G_{i,t})$. First, we calculate $P(G_{i,t})$ for a generic weed population $W$, that only make cross-fertilization. To this end, consider the probability of pick randomly a gamete with allele of type $A$ produced by this population,
denoted by \( P(A|W) \). Assuming that the mutation may occurs only once per allele during the gametogenesis, we have

\[
P(A|W) = P(A^-|\bar{m},W)P(\bar{m}|W) + P(a^-|m,W)P(m|W),
\]

where \( A^- \) and \( a^- \) are the events of take at random an allele at the beginning of gametogenesis, and \( m \) and \( \bar{m} \) are, respectively, the mutation and non-mutation events. Considering the complementarity of the events \( A^-/a^- \) and \( \bar{m}/m \), and assuming the independence of mutation event with allele genotype and \( W \), we can write

\[
P(A|W) = P(A^-|W)(1 - 2P(m)) + P(m).
\]

(B.1)

Then, assuming that the gametes combine randomly to form a seed, consider to pick at random a seed produced by this subpopulation for inspection. Denoting by \( G_i \) the event of finding a seed with genotype \( i \), we have

\[
P(G_1|W) = (P(A|W))^2,
\]

(B.2)

\[
P(G_2|W) = 2P(A|W)(1 - P(A|W)),
\]

(B.3)

\[
P(G_3|W) = (1 - P(A|W))^2.
\]

(B.4)

Now we consider a more general population, in which two fertilization mechanism is possible: self and cross-fertilization. To calculate \( P(G_{i,t}) \) for this population, we may assume that seeds are produced by two distinct subpopulations, where each employs only one fertilization mechanism. Hence, using the total probability rule, we get

\[
P(G_{i,t}) = P(G_{i,t}|F_a)P(F_a) + P(G_{i,t}|\bar{F}_a)P(\bar{F}_a),
\]

(B.5)

where \( F_a \) is the event of the seed be generated by self-fertilization and \( \bar{F}_a \) by cross-fertilization, and \( P(F_a) \) and \( P(\bar{F}_a) \) are, respectively, its probability of occurrence.

The self-fertilizing subpopulation may be also divided in 3 subpopulations, where each has only mature weeds with one genotype of \( G \). So, using the total probability rule again, we get

\[
P(G_{i,t}|F_a) = \sum_{\ell \in G} P(G_{i,t}|Y^a_\ell, F_a)P(Y^a_\ell),
\]

(B.6)

where \( Y^a_\ell \) refers to the event of the weed parent have the genotype \( \ell \). Note that it does not matter to assume either cross or self-fertilizing in a sub
population with the same genotype. What means that we can also use (B.2)-(B.4) for $P(G_{i,t}|Y^a_{i,t}, F_a)$.

Assuming $P(m)$, $P(F_a)$ and $P(F_a)$ time invariant and given, to calculate $P(G_{i,t})$, we only need to know $P(Y^a_{i,t})$, and $P(G_{i,t}|F_a)$ and $P(G_{i,t}|Y^a_{i,t}, F_a)$ for all $i, l \in G$. First, note that the former can be easily obtained doing $P(Y^a_{i,t}) = Y^a_{i,t}/\eta(Y^a_{i,t})$ (please, see Appendix A for definition of $\eta$). Now, recalling that $W$ was defined as a general weed population, is sufficient to employ $P(A^-|F_a) = \mu(Y^a_{i,t})$ in (B.1) to obtain $P(G_{i,t}|F_a)$, and $P(A^-|Y^a_{1,t}, F_a) = 1$, $P(A^-|Y^a_{2,t}, F_a) = 0.5$ and $P(A^-|Y^a_{3,t}, F_a) = 0$, to obtain $P(G_{i,t}|Y^a_{i,t}, F_a)$, for all $i, l \in G$.

Appendix C. Weed model parameters

All parameters employed in simulations are presented in Table [C.1]. Some parameters (mortality induced by the target herbicide, reproduction, self and cross-fertilization probabilities) were obtained for the herbicide nicosulfuron and the life cycle of B. pilosa, an aggressive and rather preeminent weed in annual and perennial crops in Brazil. According to greenhouse experiments carried out by the Brazilian Agricultural Research Agency Embrapa Milho e Sorgo, the recommended dose of nicosulfuron (60 g ha$^{-1}$ for maize cultures) causes 84.50% of phytotoxicity in susceptible B. pilosa individuals and 22.75% in resistant individuals. According to [23], we assume a 92% of self-fertilization probability. We set $g = 1846.2$ in order to obtain $x^{sp} = 1500$ when the density of mature plants is $1m^{-2}$, and $G = 70000$, the maximum expected seed production density (please, see comments below Equation (4)). We consider a probability of $10^{-9}$ for an allele mutation at gametogenesis and an adaptive cost of 3% following [21] and [20], respectively. For the remaining parameters we adopt the suggestions of specialists.
Table C.1: Parameters used in the numeric simulations

| Parameter | Value       | Parameter | Value       |
|-----------|-------------|-----------|-------------|
| $F$       | $2 \times 10^5$ | $P(m)$    | $10^{-9}$   |
| $\delta$  | 0.10        | $P(F_a)$  | 0.92        |
| $\gamma$  | 0.10        | $g$       | 1846.2      |
| $\psi$    | 0.14        | $G$       | 70000       |
| $c$       | 0.03        | $\rho^S(u^*)$ | 0.8450   |
| $\kappa$  | 0.10        | $\rho^R(u^*)$ | 0.2275 |
| $\varphi$ | 0.20        | $\rho(v^*)$ | 0.8450   |

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Frequência do Alelo

Ocorrências

Frequência do Alelo

ALEXXX
LDX
Frequência do Alelo

Ocorrências

LMXXX
LDX
