SUPPLEMENTARY MATERIAL

To: *A model for the dynamics of a protozoan parasite within and between successive host populations*, by D. Klinkenberg and J. A. P. Heesterbeek

The model (1) was simplified in 2 steps: first, the within-cohort dynamics was described by subsequent infection generations, and second, approximations were made for the separate generations. We start with a few simplifying assumptions:

- $\alpha=1$: waning of immunity is irrelevant due to continuous re-infection
- $\gamma=\beta$: precise estimates for γ are not available and the order of magnitude is correct
- a new immune variable $\tilde{y}_k$ is defined by the approximation $1+y_k^m \approx (1+y_k)^m=\tilde{y}_k^m$. Note that $\tilde{y}_0=1$.

Then, by regarding only the first ingested dose of each infection generation k (in original time: $w_t=a_0 v_t$ only for $t=0$, 5, 10, etc, otherwise $w_t=0$), the dynamics are fully described by v and $\tilde{y}$ only. This results in the following model:

$$v_{k+1}=\phi v_k+\frac{r v_k}{\left(\tilde{y}_k^2(1+p v_k)\right)^m} \qquad (A1.1)$$

$$\tilde{y}_{k+1}=\tilde{y}_k(1+p v_k)\left(1+\frac{p\lambda_1 v_k}{\tilde{y}_k^m}\right) \qquad (A1.2)$$

The new parameters are $\phi=(\sigma(1-a_0))^5$, the oocyst survival probability per infection generation, i.e. per 5 original time steps; $r=a_0 a_1 \lambda_1 \lambda_2$, the maximum oocyst multiplication rate; and $p=\beta a_0 a_1$, the immunity growth rate per oocyst per unit $\tilde{y}$. Each broiler cohort consists of four infection generations. Because it is the gamont stage that causes most of the damage, damage can be described by $d_k=v_k-\phi v_{k-1}$, the amount of oocysts excreted in generation k.

Figure A1 shows the functions V and D (defined in the main text) and the bifurcation diagram for model (A1). It appears that all typical features of the original model (1) as shown in Fig. 3 are present in the new model: the wavelike Fig. A1a and A1b, and the unstable dynamics with some cleaning rates in Fig. A1c; only the chaotic dynamics has disappeared. However, with other parameter sets (e.g. the *E. brunetti* parameter set) the simplified model is still capable of displaying chaotic dynamics.

The next step was to define functions for the damage level and the final oocyst level due to each infection generation, D_k and V_k (like D and V, these are defined on the log scale). We observe that $D_k=V_k-(4-k)\log\phi$, $k\geqslant 1$, because damage is determined by the number of gamonts which is equal to the number of excreted oocysts. This leaves us to find expressions for all V_k, starting with $k=0$ and $k=1$:

$$V_0=\log v_0+4\log\phi \qquad (A2.1)$$

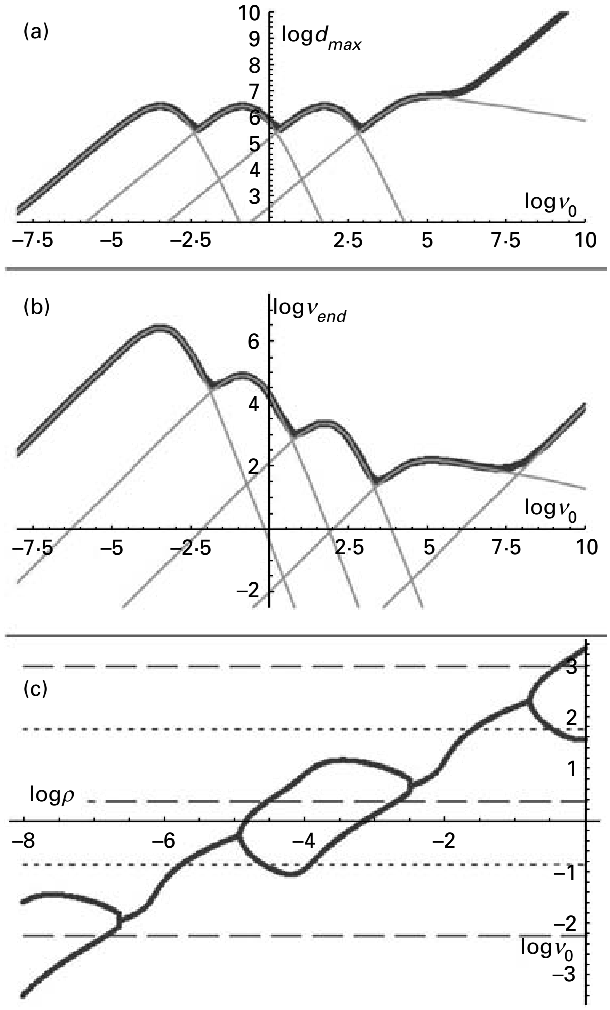

Figure A1. Results of the simplified models (equations A1 in thick black lines and A2 in thin grey lines) for the default parameter set; (a) relation D between initial oocyst level $\log v_0$ and maximum damage $\log d_{max}$ during a single chicken cohort; (b) relation V between initial oocyst level $\log v_0$ and final oocyst level $\log v_{end}$; (c) bifurcation diagram for the between-cohort dynamics of $\log v_0$ as a function of the cleaning efficiency $\log\rho$.

$$V_1=\log v_0+\log r-m\log(1+p v_0)+3\log\phi \qquad (A2.2)$$

For excretion in generations $k=2$, 3, and 4 to be relevant for the final oocyst level, immunity should still be small in generation $k-1$. As long as the oocyst level and immunity are small in the early generations ($v\approx 0$ and $y\approx 1$), model (2) can be approximated by $v_{k+1}=r v_k$ and $y_{k+1}=1+p\lambda_1 v_k$. Therefore, the level of immunity can be described in terms of the oocyst level, $y_k=1+p\lambda_1 v_k/r$, which leads to the following function for v_{k+1}:

$$v_{k+1}=\phi v_k+\frac{r v_k}{(1+p\lambda_1 v_k/r)^{2m}(1+p v_k)^m} \approx \frac{r^{k+1} v_0}{(1+p\lambda_1 r^k v_0/r)^{2m}(1+p r^k v_0)^m}$$

Thus, V_k, $k>1$, is equal to:

$$V_k = \log v_0 + k \log r - 2m \log(1 + p\lambda_1 r^{k-2} v_0) - m \log(1 + pr^{k-1} v_0) + (4-k) \log \phi \qquad (A2.3)$$

All functions D_k and V_k are plotted in Fig. A1 (thin grey lines).

As indicated in the main text, unpredictable damage hinges on unstable dynamics and large differences between minima and maxima in D. The condensed model (A2) gives insight into the role of the different parameters:

- the distance between the peaks of D_k and D_{k+1} (and between V_k and V_{k+1}), $k>1$, in the $\log v_0$ direction is equal to $\log r$. The distance between the peaks of D_1 and D_2 (and between V_1 and V_2) also increases with increasing $\log r$. Therefore, an increase in $\log r$ results in larger differences between damage minima and maxima, and it results in a larger domain in V with negative slopes <-1.
- the distance between the peaks of V_k and V_{k+1}, $k>1$, in the $\log v_{end}$ direction is equal to $\log \phi$. There is no effect on D, but the domain in V with possibly unstable between-cohort dynamics increases if $\log \phi$ decreases.
- the slopes left of the peaks of all V_k is 1, the slope right of the peak of V_1 is $1-m$, and the slopes right of the peaks of all V_k, $k>1$, is $1-3m$. Thus, the minimum slopes for realistic values of m are smaller than -1 and these are always close to the minima in D. A larger m will result in smaller minima in D and possibly less stable between-cohort dynamics.
- the minimum slopes $1-3m$ are due to two terms in V_k, $k>1$: $-2m\log(\ldots)$ and $-m\log(\ldots)$, which contribute to V_k only if $p\lambda_1 r^{k-2} v_0$ and $pr^{k-1} v_0$ are notably larger than 0. The peak between increase (slope = 1) and decrease (slope = $1-3m$) will be sharpest if both terms are of the same order of magnitude, that is if $\lambda_1 \approx r$, because that will cause the most prominent transition between maximum and minimum slope. Sharper peaks will result in a sharper profile in D, and in a larger domain in V with unstable dynamics.
- an increase in $\log p$ causes a translation of the series of peaks in the $(-1, -1)$ direction. This does not affect the qualitative behaviour of the between-cohort dynamics.