

A Mathematical Model Investigating the Impact of an Environmental Reservoir on the Prevalence and Control of Vancomycin-Resistant Enterococci

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CMV DNA in CSF from patients with GBS will have to be elucidated further in future studies. Even more importantly, the clinical significance of CMV DNA present in CSF has to be investigated, particularly to clarify whether these patients should be offered antiviral therapy.

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A Mathematical Model Investigating the Impact of an Environmental Reservoir on the Prevalence and Control of Vancomycin-Resistant Enterococci

To the Editor—In an article recently published in the *Journal of Infectious Diseases*, D'Agata et al. [1] presented a mathematical model of the transmission of vancomycin-resistant enterococci (VRE). We developed an extension of that model that incorporates an environmental reservoir for VRE. Although our model (which we call the “environment model”) supports many of the findings of D'Agata et al., we predict different outcomes for some infection-control interventions.

VRE is known to contaminate environmental surfaces [2, 3], and case-control studies have suggested that such contamination can contribute to the acquisition of VRE [4]. This had led many researchers to speculate that the environment plays an important role in patient acquisition of VRE.

Mathematical models can give insight into the likely consequences of infection-control practices, such as hand hygiene [5, 6], patient cohorting [5, 6], staff-to-patient ratios [7], and antibiotic restriction [1]. However, mathematical models will only deliver results based on the assumptions underlying them. All models that have been published to date on the nosocomial transmission of VRE have assumed that there is no transmission due to environmental contamination. It is important to estimate how environmental contamination could influence the outcomes of infection-control interventions.

The environment model uses the structure and assumptions of the model presented by D'Agata et al. and adds a new environment compartment. It is assumed that the environment is saturable and that colonized patients and health-care workers contribute to environmental contamination. In turn, the contaminated environment can cause the contamination of health-care workers, indirectly leading

to patient colonization. The new model requires the addition of 4 parameters: β_{eh} (0.15), transmission from health-care workers to the environment; β_{eo} (0.4), transmission from colonized patients not exposed to antibiotics to the environment; β_{ei} (4), transmission from colonized, antibiotic-exposed patients to the environment; and κ (0.1), the rate of decontamination of the environment. Parameters were chosen so that the rate of environmental contamination was 25% that of patient or health-care worker contamination. In accordance with the findings of Wendt et al. [8] and Noskin et al. [9] that VRE persists in the environment for at least 1 week, we assumed that VRE persists for an average of 10 days in the environment. To make the equilibrium colonization prevalence the same as that of D'Agata et al., the “fitted” parameter, β_{pi} , was 0.0074 in our model. All other parameters were those of D'Agata et al.

Our model predicted that, in the presence of an environmental reservoir, the direction of the impact of infection-control interventions is the same as the predictions in the model of D'Agata et al. (which we call the “original model”) but that the magnitude is altered. The environment model predicts that improving hand-hygiene compliance from 40% to 60% leads

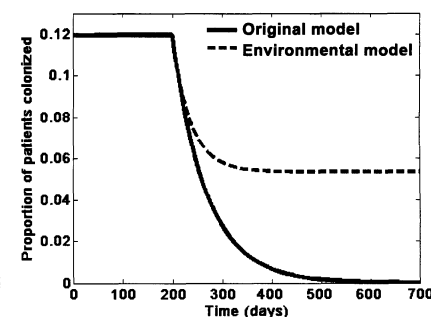


Figure 1. Model predictions of the prevalence of vancomycin-resistant enterococci (VRE) over time. Both the environment and the original model begin with VRE at an endemic steady-state level of 12%. On day 200, further colonized patients are prevented from entering the ward. In the environment model, a new equilibrium is established at 5.3%.

Equations

$$\begin{aligned}\frac{dP_{u0}}{dt} &= \Delta_{u0} + \sigma_{u1}P_{u1} - (\tau_{u0} + \gamma_{u0})P_{u0}, \\ \frac{dP_{u1}}{dt} &= \Delta_{u1} - (\sigma_{u1} + \gamma_{u1})P_{u1} + \tau_{u0}P_{u0} - \left[\alpha_{p1}\beta_{p1}(1 - \eta) \frac{H_c}{N_h} \right] P_{u1}, \\ \frac{dP_{c0}}{dt} &= \Delta_{c0} + \sigma_{c1}P_{c1} - (\tau_{c0} + \gamma_{c0})P_{c0}, \\ \frac{dP_{c1}}{dt} &= \Delta_{c1} - (\sigma_{c1} + \gamma_{c1})P_{c1} + \tau_{c0}P_{c0} + \left[\alpha_{p1}\beta_{p1}(1 - \eta) \frac{H_c}{N_h} \right] P_{c1}, \\ \frac{dH_u}{dt} &= \left[-\alpha_{p0}\rho\beta_{h0} \frac{P_{c0}}{N_p} - \alpha_{p1}\rho\beta_{h1} \left(\frac{P_{c1}}{N_p} + E \right) \right] (N_h - H_c) + \mu H_c, \\ \frac{dE}{dt} &= \left(\beta_{c0} \frac{P_{c0}}{N_p} + \beta_{c1} \frac{P_{c1}}{N_p} + \beta_{ch} \frac{H_c}{N_h} \right) (1 - E) - \kappa E.\end{aligned}$$

to a reduction in admission prevalence of 17% for the environment model, compared with 23% for the original model. Increasing staff-to-patient ratios from 1:4 to 1:2 leads to a reduction in colonization prevalence of 24% for the environment model, compared with 32% for the original model. Reducing the length of stay of colonized patients from 28 to 14 days led to a reduction in colonization prevalence of 51% for the environment model and 64% for the original model. The selective isolation of colonized patients (under the presumption of 80% efficacy) led to a predicted 44% reduction in colonization prevalence for the environment model, compared with 42% reduction for the original model.

A significant prediction of the environment model is that, even if colonized patients are prevented from entering the ward, VRE remains endemic at a rate of 5.3%, as illustrated in figure 1. This differs from the conclusion of D'Agata et al. The system of ordinary differential equations governing this model is as follows.

The model predicts that the presence of an environmental reservoir reduces the predicted efficacy of some interventions (e.g., compliance with hand hygiene, increased staffing levels, and reduced length of stay) yet increases the predicted efficacy of others (selective isolation). The results of the model with regard to different effects due to interventions are not intuitively obvious. We show that, under certain circumstances, an environmental reservoir for VRE could lead to endemic VRE transmission even if the admission of

VRE-colonized patients ceases. These results suggest that, in the presence of an environmental reservoir, VRE may be harder to eradicate and infection-control interventions less effective, with the exception of patient isolation, which remains effective, as predicted by this model.

The system of ordinary differential equations describing the environment model is shown. Please see D'Agata et al. [1] for an explanation of parameters not given in the text.

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