

A SYSTEM DYNAMICS MODEL FOR ANALYSING POPULATION RISK IN CASE OF A VOLUNTARY VACCINATION PROGRAM AGAINST MENINGOCOCCAL MENINGITIS

Yolanda Álvarez

Martín Caicoya

Rafael García

Begoña González-Busto

Summary

Meningococcal meningitis outbreak is a complicated public health problem. Control measures include prompt treatment of cases, vaccination and contacts chemoprophylaxis. The treatment of an index case cuts the spread of bacteria, the treatment of contacts avoids the disease in those that might be infected and cuts the spread of the disease by carriers and the vaccination lowers the risk of disease. Vaccination has a limited value because it does not protect children less than two years, only confers partial immunity against certain serogroups, the duration of immunity is short, it does not provide herd immunity and revaccination has conflicting results. When the incidence is high, mass immunization is recommended. If the incidence is not high, in some situation where public pressure is high, an offer of voluntary vaccination has been done. We hypothesise that in this situation the risk of nonvaccinate might be increased. In effect, those that decide to be vaccinated will have the same risks and benefits than those that did so under massive vaccination. However, those that decide no to vaccinate might be an increased risk. This is so because vaccination has avoided the disease in those that would have become cases if no vaccination; but it does not avoid their infection: they will be spreading virulent bacteria, and as no treatment is imposed to cut their infection, they would do so as long as it lasts the natural history of this situation. Those contacts that are vaccinated are at low

risk but non-vaccinated contacts are at high risk because they are not to benefit from chemoprophylaxis and they might be exposed for longer periods of times to the aborted index case.

Introduction

Meningococcal meningitis outbreak is a complicated public health problem. Most of the cases and the deaths occur among children and teenagers. The industrialised countries are not used to loose children because of infectious diseases. The presence of a temporal or geographical cluster of meningitis produces an alarm in the society and usually a mass hysteria (1). Media and population pressure over the Public Health Authorities might be one of the most important components in the decision to vaccinate.

In Spain the incidence of Meningococcal disease had a peak in 1979 with a rate of 17.9 cases per 100000 inhabitants. In the last 10 years the rates varied between 1 and 3 cases per 100000, however there are interregional differences (2). Rates similar are found in England and Wales (3). In 1996 we experience in Spain an increment meningococcal disease (2). This prompted vaccination in several regions (4) and a consensus conference to evaluate and produce guidelines for the management of the problem (5).

The strategies to control meningococcal disease are aimed to reduce the risk of death among cases and reduce the risk of disease among contacts. In order to reduce the mortality rate the diagnosis and treatment of suspected cases should be rapid as well as the admission to hospital. Here, the public health authorities and the media have a role in the dissemination of the information to enable the public to recognise the early symptoms of the disease. The strategies to reduce the dissemination of the disease include good housing and vaccination and treatment of close contacts. Most of the cases are sporadic, i.e. the

source is not known, therefore the usefulness of chemoprophylaxis in close contacts is limited. However, serogroup C causes epidemic diseases (6). The attack rate for the people who live in the same household as a case of meningococcal disease is increased by about 500 to 1200 times, representing a risk of 1% for household (7). For serogroup C the attack rate may be as high as 1000/100000 in schools while it is only between 5 and 20/100000 in the community (6). The estimated secondary attack rate in American schools is 2.5/100000, while the annual incidence of meningococcal disease is 1.08/100000 for the same age group (8). The risk of a secondary case is mainly reduced to the first week since the case is diagnosed. Vaccination against serogroup C meningococci is recommended whenever the risk of meningococcal disease is high enough as to render benefits. It has been recommended to vaccinate when the rate is 10/100000 of this serogroup, considering the denominator the population that is subject of vaccination, or the occurrence of at least three in three months, in an organisation or a community (9).

The treatment of an index case cuts the spread of bacteria, the treatment of contacts avoids the disease in those that might be infected and cuts the spread of the disease by carriers and the vaccination lowers the risk of disease. Vaccination has a limited value because it does not protect children under two years, only confers partial immunity against certain serogroup, the duration of immunity is short, it does not provide herd immunity and revaccination has conflicting results (8). We hypothesise that the risk of nonvaccinate might be increased. In effect, those that decide to be vaccinated will have the same risks and benefits than those that did so under massive vaccination. However, those that decide no to vaccinate might be an increased risk. This is so because vaccination has avoided the disease in those that would have become cases if no vaccination; but it does not avoid their infection: they will be spreading virulent bacteria, and as no treatment is imposed to cut

their infection, they would do so as long as it lasts the natural history of this situation. Those contacts that are vaccinated are at low risk but non-vaccinated contacts are at high risk because they are not to benefit from chemoprophylaxis and they might be exposed for longer periods of times to the aborted index case.

The experience of Galicia (Spain) might serve as reference. There, in 1996 the incidence of meningitis went up to 14.5/100000 person-years from a previous rate in the 80' and 90' lower than 4/100000-person year's (4). The incidence of serogroup C meningitis was calculated to be 12/100000 person-year. This situation precipitated a massive vaccination starting in December 1996. We have estimated from the data published that In 1996 the peak incidence for children 2 to 4 years was 4,5/100000 person-week for serogroup C. In the first 16 weeks of 1997 the incidence of serogroup C meningitis among non-vaccinated (5% of the population) for children 3 to 5 years was 8/100000 person-week, an incidence that is twice that of the worst week in 1996, when there was no vaccination. From this perspective, the risk of the non-vaccinated in a population which high vaccination coverage might be twice that of non-vaccinated when the vaccination coverage is absent or low. In the other hand, the incidence among those vaccinated (95% of the population) went down to 0,2 /100000 person-week, with an estimated protection as compared to the incidence among nonvaccinated in the same weeks of 97,5%.

The experience of Australia is similar. An outbreak was declared for a rate before vaccination was 17.55/1000. After a massive vaccination and chemoprophylaxis for serogroup C outbreak, 6 cases occurred among 483 vaccinated children and 2 among 47 non-vaccinated children, for rates of 12.92/1000 and 42.55/1000. Therefore the rate among non-vaccinated was 2.5 times that of the rate that occurred when the outbreak was declared (10). In Asturias the vaccination campaign took place in 1997-1998. Among the 145599

vaccinated persons a case of meningococcal meningitis C occurred in the first weeks following the vaccine campaign, and none among the 59522 non-vaccinated. Therefore, there is no evidence that the risk is higher among non-vaccinated (11)

From this analysis it might be concluded that vaccination is able to diminish the rate of meningococcal disease among vaccinated but it might increase its risk among non-vaccinated. Therefore, if vaccination is offered, it might be necessary to inform the population that the risk of disease among those that decide not to vaccinate might be increased because of the vaccine. The purpose of this study is to examine the hypothesis by means of a system dynamics model. The hypothesis is that the nonvaccinated population is at higher risk in a community where the immunisation rate is high than that where the immunisation rate is low.

Material and methods

We built a system dynamics model with Vensim with the following assumptions:

1-Cases are infectious 4 days before starting treatment.

2-Each case makes 0.05 contacts a day.

3-10% of the contacts becomes infected

4-10% of the infected becomes cases

5-Infected cases remain infected for 1 month if they had not developed the disease and they develop immunity.

6-20% of the cases dies.

7-We introduce an exogenous infective rate of $5/100000$ in time=25

8-We assume that the efficacy of the vaccine is 100% given this is not the subject of our enquire.

8-We compare the index of cases in the two situation: when a mass vaccination is introduced (in our case the coverage is 85%) and when no vaccination occurs. The index is defined as the number of cases occurred among the nonvaccinated population.

In the next figures we depict the flow structure of the model, the definition of infection rate, and the used indexes.

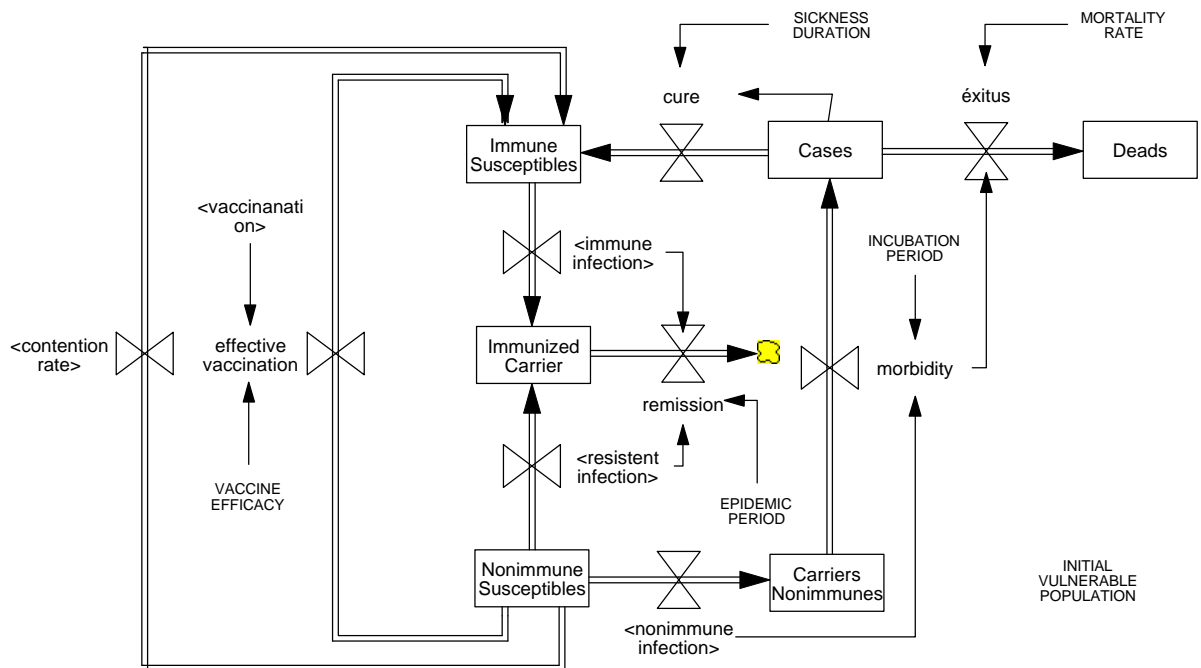


Figure 1. Flow diagram of the model

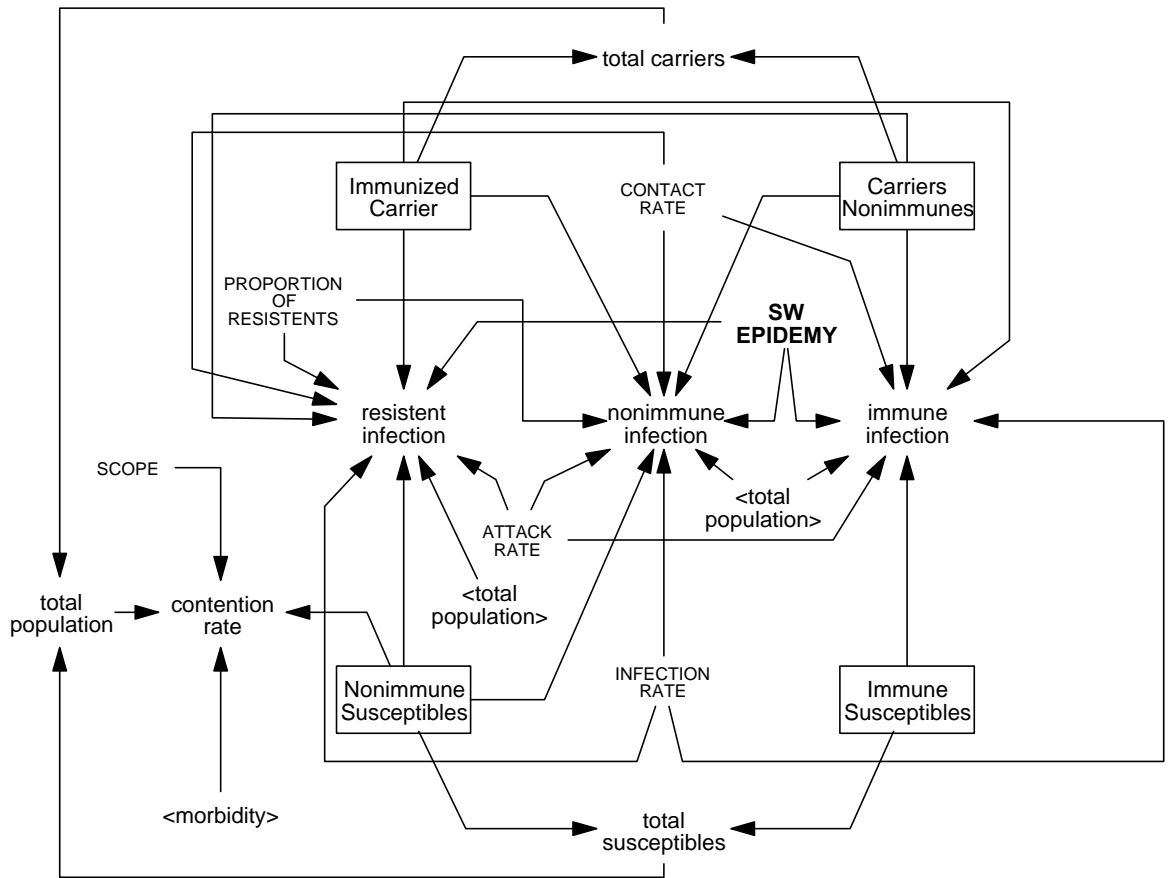


Figure 1. Diagram of the infection rates

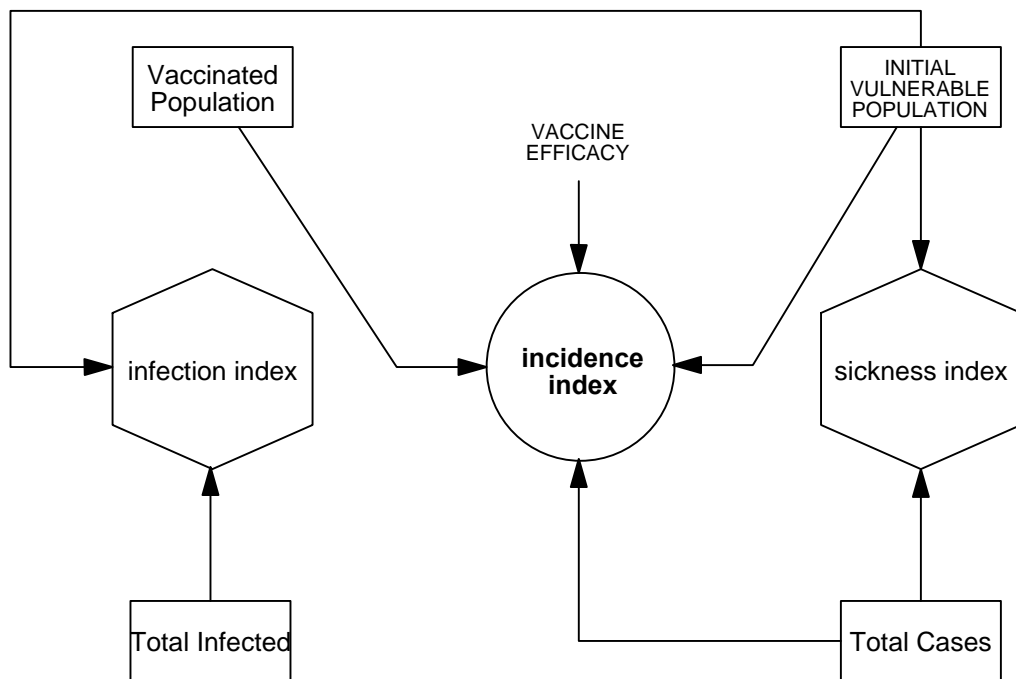
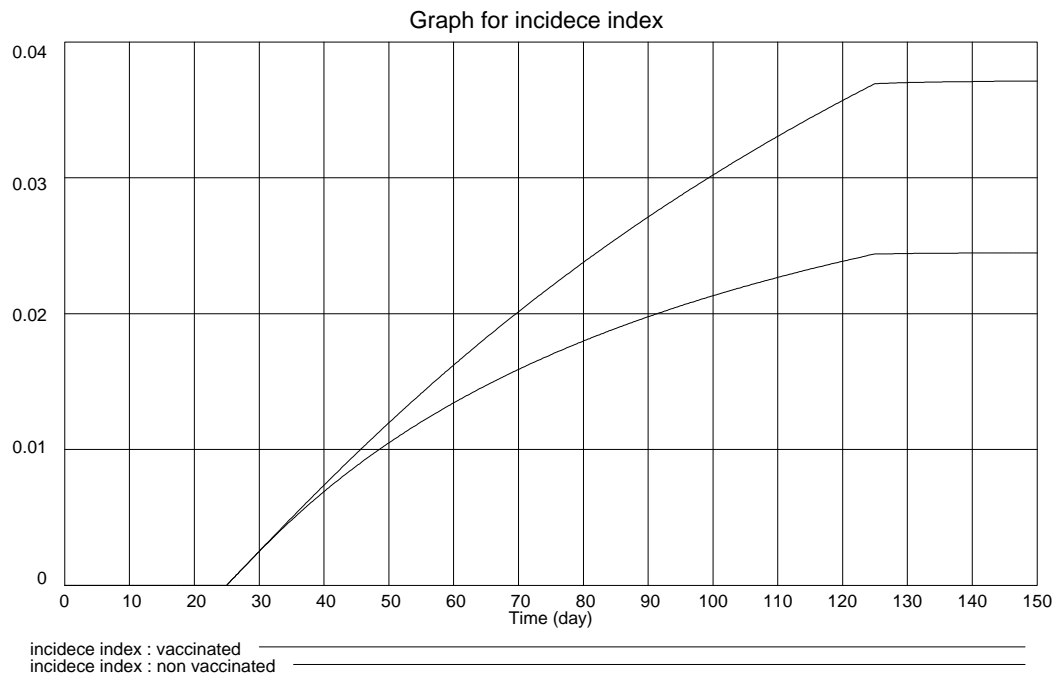


Figure 3. Infection index

Results

In figure 3 we show the result o the simulation. Among nonvaccinated cases in a population with a 85% coverage of vaccine the rate of meningococcal disease is double than that occurring in a population where no vaccination has been introduced. This proportion are maintained for contact rate up to 0.5, duration of carrier state up to 10months, epidemic attack of 1 per day per 100 days.



Conclusion

With the assumption taken in this model we find that the risk of those that do not accept vaccination or are not reached by the vaccination campaign doubles when the vaccination coverage is high. In the examination of the data from two experiences of massive vaccination we found similar results. This renders robustness to our findings. However, epidemics models are very complicated to reproduce. The model of diffusion produces a number of cases way over the reality even for a very low contact rate. This is because of the mechanics of contact and the ecology of the bacteria. Modelling the last is complicated.

The decision to perform vaccination against meningococcal disease must be presided by an analysis of cost/benefit . The public health authorities must decide how much they want to spend for each year of life saved. The benefits will be higher when the rate of disease is higher. Once vaccination is adopted as a public health measure, the objective should be to accomplish a high coverage, not only in order to benefit the most and in this way to control the epidemic, but also not to increase the risk of those that do not get to be vaccinated. We think that in case the vaccination strategy is adopted, the public health authorities should inform of all the risk and benefits of complying and not complying with the recommendation. With most of vaccines, herd immunity protects those not vaccinated, therefore they are at lower risk that if no vaccination campaign has taken place. But with meningococcal vaccine the risk of non vaccinated is increased. In this sense, an emphasis should be putted in informing the population of the risk of non accepting vaccination when the coverage is high.

Reference

1-Hume SE, Mass Voluntary Immunization Campaign for Meningococcal Disease in Canada: Media Hysteria.

2-Boletín Epidemiológico Semanal. Instituto Carlos III. Ministerio de Sanidad y Consumo 1987-1998.

3-Jones DM. Meningococcal infection in England and Wales 1994. Communicable Disease Report/CDR Review 1995;5:125-137

4-La campaña de vacunación frente al meningococoC: resultados y primera evaluación.en Galicia.Boletín Epidemiológico Semanal. 1996;46:393-396.

5-Resumen de la Conferencia Nacional de la Enfermedad Meningococica . Oviedo 17-18 Junio de 1997. Boletín Epidemiológico Semanal. 1996;46:396-398

6-Jackson LA, Schuchat A, Reeves MW, Wenger JD. Serogroup C Meningococcal Outbreaks in the United States. JAMA 1995;273:383-389.

7- Meningococcal Infectious Working Group and Public Health Medicine. Environmental Group. Control of Meningococcal Disease. A Guidance for Consultants in Communicable Disease Control. CDR, Review 1995;5:189-198.

8- Zanwill KM, Schuchat A, Riedo FX, Pinner RW, Koo DT, Reeves MW et al. School -Based Cluster of Meningococcal Disease in the United States. JAMA 1997;277:389-395.

9-Control and Prevention of Meningococcal Disease and Control and Prevention of Serogroup C Meningococcal Disease: Evaluation and management of Suspected Outbreaks. MMWR 1997; 46:Nº RR-5.

10-Pearce MC, Sheridan JW, Jones DM, Lawrence GW, Murphy DM, Massutty B et al. Control of group C meningococcal disease in Australia Aboriginal children by mass rifampicin chemoprophylaxis and vaccination Lancet 1995;346:20-23.

11-Huerta I . Servicio de Salud Pública. Sección de Vigilancia Epidemiológica Personal communication.