Foodborne transmission of norovirus: mechanism modelling, seasonality and policy implications (2020 System Dynamics Applications Award paper)

FAST TRACK

Foodborne transmission of norovirus: mechanism modelling, seasonality and policy implications (2020 System Dynamics Applications Award paper)

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Abstract

The article describes a study of the foodborne transmission mechanisms for norovirus. It was undertaken for the U.K. Food Standards Agency and received the System Dynamics Society's 2020 "System Dynamics Applications Award". The article opens with descriptions of norovirus, the organisational context and the aims of the study. The first phase involved the construction of a large, fully formulated SD simulation model which included person-to-person mechanisms and, newly built, food-related mechanisms for norovirus transmission. The group modelling process and the model structure are described. The model's existence demonstrated that enough was known about foodborne mechanisms to create an explicit and carefully documented representation that specialists recognised, understood, and accepted. Additionally, a framework for analysing the model's parameters—some currently unknown—helped organise FSA thinking on future research and potential policy levers. A second phase used mathematical analysis of a simplified SD model to assess the relative scale of the foodborne effects. In terms of contributions, this generated insights into possible sources of seasonality and insights into whether the most effective leverage points in the system lay solely within the remit of the FSA or were also within the remits of other government departments. The article closes by summarising the findings and then exploring their policy implications and recording the client's reactions to them.

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Introduction

This article describes a model-based study of the different transmission mechanisms for norovirus. It was undertaken for the Food Standards Agency in the United Kingdom. The FSA's objective was to use system dynamics modelling to increase and record understanding of the foodborne transmissions mechanisms of the virus and to use that modelling to explore ways of reducing the spread of norovirus in humans

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Norovirus after COVID

At its core, this work concerns the epidemiological modelling of a virus which causes disease in humans. Many of the features of the modelling, of how the virus is transmitted, of how transmission can be reduced, how one tests for it, etc. seemed very specific to the study when it was being undertaken. However, readers looking at this work today will find themselves nodding with recognition at many points. Quite simply, this is because the work was done before the appearance of SARS-CoV-2.

Whilst norovirus is a different problem in many ways, the connections with SARS-CoV-2 are notable. As is the case with applied work, we had to learn about the particular detail and sometimes arcane terminology of the norovirus application domain. Yet much of this has ceased to be as specialised as when we encountered it. Today, we all live in a world suffused with concern about fomites and aerosol transmission. Discussions regarding the benefits of hand-washing and mask-wearing, and debates about the effects of immunity are daily experiences. Terms such as "exponential growth" and "PCR test" are heard regularly in news sources and in conversations. None of this was the case when our work was undertaken. However, perhaps the biggest development is the apparent growth in the use of modelling by governments across the world and its presence in public discourse.

System dynamicists believe in the contribution that modelling can make in the formulation of policy. In the case of the SARS-CoV-2 pandemic, in the United Kingdom, we are already seeing accounts of how this played out, with success stories (Gilbert and Green, 2021) and troubled descriptions of perceived failures (Farrar and Ahuja, 2021; Horton, 2022). However, it is clear that there is now more interest amongst academics in contributing to the public understanding of how quantitative approaches can help when formulating public policy (Spiegelhalter and Masters, 2021; Alba, 2022) and more engagement between academics and policymakers (Reicher, 2021).

In terms of the content of the models used, systems ideas aspire to generality, be it concepts from the broad systems science field (von Bertalanffy, 1950; Boulding, 1956), or system dynamics ideas about core mathematical structures (Forrester, 1968) or insights into the way that social systems behave (Forrester, 1969). Norovirus and SARS-CoV-2 provide a vivid example of that generality. A search of the materials published online quickly shows that the core structure at the heart of the SARS-CoV-2 models used around the world today is exactly the one that we used in our modelling. It is the structure used with polio by Forrester Award winners Thompson and Duintjer Tebbens (2007). It is the structure that one of the authors of this article (Lane) learned in Oxford in the 1980s (Murray, 1989). That structure is the "susceptible-infective-removed", or "SIR", model—and it is a century old (Kermack and McKendrick, 1927). Powerful systems ideas endure.

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The global pandemic of SARS-CoV-2 that we are still living with has produced much greater familiarity with the features discussed above. It therefore seemed appropriate to start by commenting on the suddenly changed circumstances in which this article will perforce be read.

The approach of this article

One description of our work has been published previously (Lane, *et al.*, 2019). The generosity of the Food Standards Agency means that this was published as Open Access and so is freely available to all. That article positions the work within the methodology of MS/OR and provides very detailed references relating to that positioning and other elements. Here we have chosen to emphasise different aspects of the work.

This article therefore proceeds as follows. In the next section we set the scene, giving a description of norovirus, describing the organisational context and outlining the aims of the study. We then describe what was done to model endogenously the range of mechanisms by which norovirus is transmitted via channels related to food. We explore the process used, outline the modelling, and record the contributions made. We go on to describe an unexpected second phase of the work which allowed us to make an assessment of the scale of such foodborne effects. This phase generated insights into possible sources of seasonality and questions about the most effective leverage points in the system. We close by summarising our findings and then exploring their policy implications and the client's reactions to them.

Setting the scene

In this section, we explain a little about norovirus, describe the client organisation and outline the initial aims of the project.

Norovirus

Norovirus—hereafter "NV"—is a virus; segments of RNA inside a protein coat or capsid. These are simple submicroscopic entities, about 30–40 nm in size and so only visible via electron microscopy. Based on the amino-acid sequence diversity, NV is classed into seven genogroups: GI—GVII. Of these, GII.4 is the most frequently occurring in humans. Viruses are hard to culture because they cannot replicate without a host cell but they can be detected by applying "reverse transcription-polymerase chain reaction" (RT-PCR) testing. More detail and references may be found in Lane *et al.* (2019).

Contracting NV has unpleasant consequences. In humans it produces infectious intestinal disease. If you are exposed to it, you are latent for about two days, and then most humans experience headache and/or a fever,

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nausea, stomach pain, sudden vomiting and diarrhoea. People can become dehydrated. As might be predicted, people are highly infectious when experiencing these symptoms. Most recover in about 48 hours, albeit a very unpleasant 48 hours. This morbidity is the main effect but there are rare cases of mortality. This tends to occur in the very young and the aged and may result from perforation of the bowel or dehydration and links with other conditions. Consequently, the cause of death can be hard to ascribe to NV.

NV has been the cause of a number of well-publicised outbreaks amongst passengers on cruise ships and restaurants. Amongst the general population, cases have a strongly seasonal pattern, which is why it is also known as the "winter vomiting bug" (Figure 1).

The main vector for NV is person-to-person contact—"P2P." This can occur directly, via bodily contact. For example, if someone is caring for a sick person, changing a soiled nappy, or merely shaking hands with someone carrying the virus, and they do not wash their hands carefully before touching their mouth, then transmission and illness can result. There are also vectors for P2P that are indirect. Airborne transmission occurs from sick individuals via aerosol effects. NV can also be contracted from fomites, inanimate objects such as door handles, toys, clothing, or kitchen surfaces. If fomites are contaminated with NV, then the virus can be transmitted indirectly from one person to another.

However, P2P effects are not the whole story. NV can also be transmitted via a number of food-related mechanisms, for example, uncooked shellfish, contamination of certain foodstuffs at source or at the packaging stage, and via infected food preparers. Concerning shellfish, there are thought to be increases in NV after 14 February. This is associated with the increased consumption of raw oysters on Valentine's Day. Cooking removes transmission risk—but foodborne transmission is subtle. For example, a cluster of cases in a restaurant was found to result from diners who had tomato soup. On the surface this made no sense, since tomato soup is served hot. However, it was found that one of the chefs was sprinkling fresh basil on the soup. The chef had norovirus.

All communicable diseases cause just 9% of deaths in the West; consequently the associated risks are not well known amongst nonspecialist decision-makers since the focus tends to be on noncommunicable conditions such as cancers, strokes, and heart disease (Tooze, 2021). Even amongst infectious pathogens, the fact that NV leads to self-limiting illness and is very seldom fatal means that it has received comparatively little attention in term of public health and modelling. Nevertheless, NV is the most commonly identified cause of infectious intestinal and acute gastroenteritis in Western Europe and in the United States. In the United Kingdom, there are three million cases per year. This has an economic burden but it also means six million pretty horrible days being endured. NV deserves some attention.

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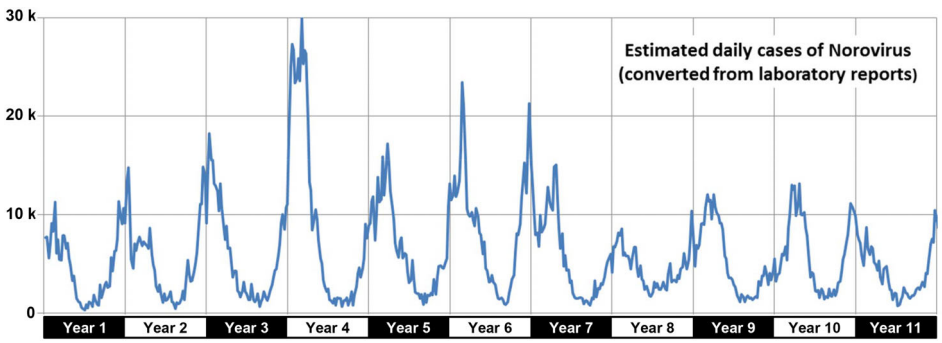

Fig. 1. Daily norovirus incidence in England and Wales for an 11-year period. Calculated from original laboratory report data provided by Public Health England (now U.K. Health Security Agency) by adjusting for estimated under-reporting and demography [Color figure can be viewed at wileyonlinelibrary.com]

The Food Standards Agency

The client for this work was the Food Standards Agency, a department of the U.K. government. Its remit is broad. The FSA is responsible, inter alia, for protecting public health from risks derived from the consumption of food including risks generated by how food is grown, picked, handled, packed, prepared, and consumed. Its food safety and food hygiene remit involves commissioning research, publishing consumer advice, and making recommendations for legislation.

Project aims

The project aims were as follows. To develop a simulation model that conceptualises, formulates, and parametrises the primary foodborne NV transmission mechanisms. It was believed that that modelling process and the resulting model would improve understanding of foodborne mechanisms and give insight into the relative importance of foodborne transmission and its effect on human cases. That, in turn, would give an indication of where FSA might concentrate its efforts to reduce foodborne transmission, allowing it to assess where risk reduction is most beneficial.

We should say that at the commissioning stage the FSA project lead (DH) observed "We think you won't be able to do this." The view was that although modelling might be possible, a lack of available parameters would probably make full completion of the aims unlikely. We all decided to give it a go anyway.

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Modelling the foodborne mechanisms as endogenous effects

This section describes Part 1 of our work. This material is what the FSA had in mind for the project when they commissioned it. Below we describe the existing work that we built on and then the process we used to construct our much larger model. We outline the structure of that model and then describe the contribution that it made to FSA thinking.

State of thinking to build on

We built on a previous study done for the FSA (Lawrence, *et al.*, 2004). Those authors were not system dynamicists and used the language of ordinary differential equations (ODEs) for their model. We therefore provide those equations before recasting them in SD terms:

$$\frac{\mathrm{dS}}{\mathrm{dt}} = -\left(\frac{\beta \mathrm{I}}{N} + \theta\right) S + \delta \mathrm{R} + \mu(N[1-\chi] - S)$$

$$\frac{\mathrm{dE}}{\mathrm{dt}} = \left(\frac{\beta \mathrm{I}}{N} + \theta\right) S - (\mu + \alpha) E$$

$$\mathrm{I} = \mathrm{Is} + \mathrm{Ia}$$

$$\frac{\mathrm{dIs}}{\mathrm{dt}} = \alpha(1-\kappa) E - (\gamma + \mu)\mathrm{Is}$$

$$\frac{\mathrm{dIa}}{\mathrm{dt}} = \alpha\kappa \mathrm{E} - (\gamma + \mu)\mathrm{Ia}$$

$$\frac{\mathrm{dR}}{\mathrm{dt}} = \gamma \mathrm{I} - (\mu + \delta) R$$

The state variables all represent groups of people: S – "Susceptible"; E – "Exposed"; I – "Infectious"; R – "Recovered." These are discussed further below. These equations are a slight development of the standard "SEIR" epidemiological structure. Here the "I" variable is split in two: Is – "Symptomatic" and Ia – "Asymptomatic." That structure is itself an extension of the "SIR" model, the almost 100-years-old formulation referred to in the Introduction and lying at the heart of SARS-CoV-2 work today.

There were two issues with this model. First, we discovered a flaw in its calibration. Second, it concentrates on P2P effects. These effects are represented endogenously by the term βIS/N. This is the standard formulation for infection resulting from people susceptible to the disease homogeneously mixing with those already infected with it. The total mixing population has

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size N. The parameter β is the number of social "encounters" per day that each person is involved in which might result in exposure. This term therefore creates a cross-link which threads together two of the ODEs in the model. Contrast this with the representation of foodborne transmission, the θS element. This is simply an exogenous forcing term which has susceptibles exposed to FB effects by a certain amount per time period, the θ parameter.

As the FSA requested, we rebuilt this model in SD software. The SFD—"stock/flow diagram"—is shown in Figure 2. Note that as with the ODEs above, the simple formulation for foodborne effects is shown—but only prior to its complete removal and replacement in what follows.

The underlying assumptions are straightforward. There is a stock of people who are "Permanently Protected" because they are immune to contracting NV (top). They still need to be represented because they are part of the population that is mixing together, and we need to account for that. There are then (left) "Susceptible Individuals," people free of NV for the moment but who could be infected if exposed to genomes. When these are exposed to infectives, or to foodborne sources of NV, an "Exposure Rate" flows them into a stock of "Exposed Individuals." After two days, they become infectious; either symptomatic (the top stock) or asymptomatic (the bottom one). After two days—very unpleasant for the symptomatic individuals—people recover, flowing into the "Recovered Immune" stock. They stay there for an average of six months before their immunity wanes, and they cycle back to become susceptible again. Births and natural deaths are also included.

Note that this model ignores the questions of whether the two infectious groups are socially mixing in the same way (unlikely, given the debilitating symptoms of NV), or whether they are equally infectious (a much more complex matter).

The SFD illustrates our task: based on what experts currently knew of foodborne infection routes, to turn θ, simply a number, into set of endogenised causal mechanisms using SD modelling.

Overview of process employed: Iterative modelling

The FSA arranged access to scientists who were expert in different aspects of NV. They were drawn from a range of organisations: the Animal Health and Veterinary Laboratories Agency; the Centre for Environment, Fisheries and Aquaculture Science; the Food and Environment Research Agency; the Health Protection Agency; the University of Liverpool; and the FSA itself. We built their endogenised model of the main foodborne contamination mechanisms of NV (see next subsection).

With that group of domain experts, we used an iterative modelling process. Vensim was the SD software we were asked to use. There was a literature review. We then did a series of, at a distance, expert interviews to elicit

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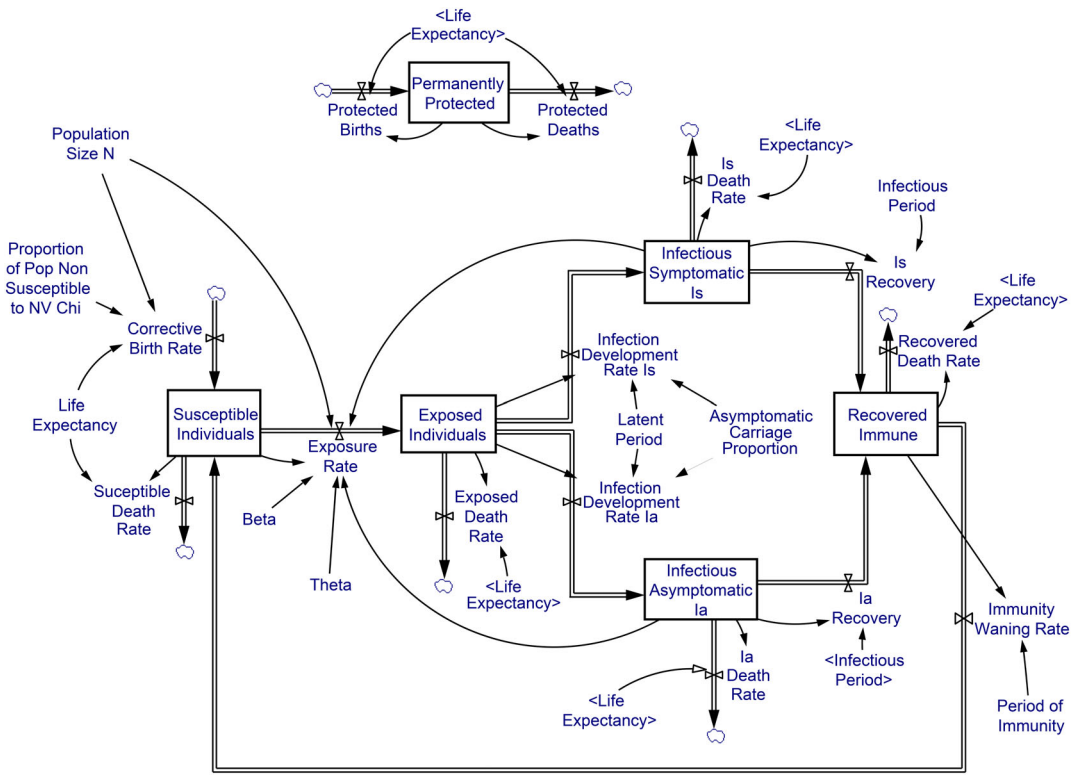

Fig. 2. Stock/flow diagram illustrating the model which was the point of departure for our work [Color figure can be viewed at wileyonlinelibrary.com]

information. We introduced people to SFDs and showed them pieces of model structure for comment and correction, all to draw them into the construction and use of the simulation model. There was further literature reviewing and interviews and, finally, a workshop that brought it all together.

The ideas of model ownership and the creation of an agreed description of a situation are central to system dynamics (Forrester, 1961) and have become increasingly important (Vennix, 1996, 2000; Lane, 1999; Lane and Husemann, 2008). We therefore comment here on some of the features of the process of engaging with the NV experts—at a distance and then in a final workshop.

We introduced stock/flow iconography. Normally we ask people to think in terms of bathtubs, taps and plugholes, then show the stock and flow symbols. In a striking departure, our point of reference was the unfriendly (to some) form of ordinary differential equations (see Figure 3). Why?

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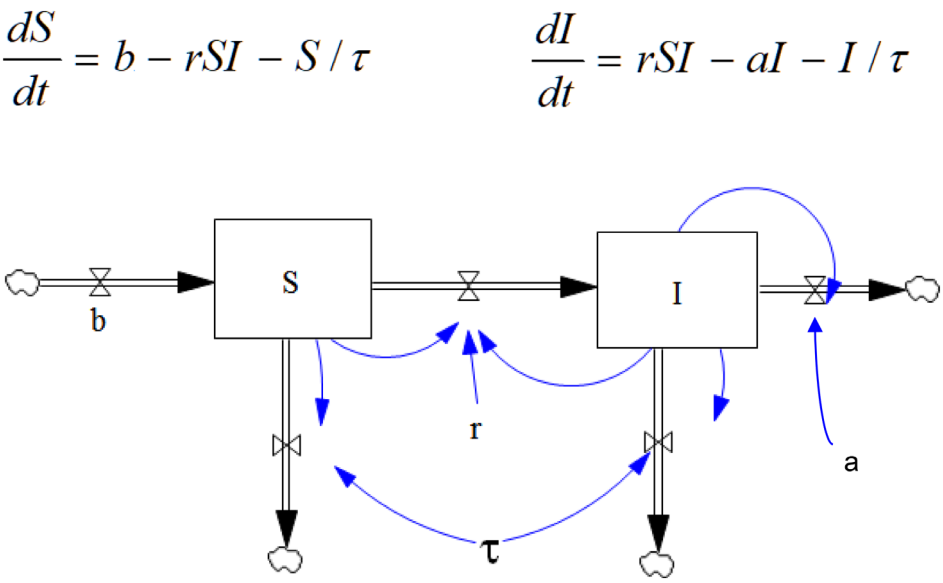

Fig. 3. Diagram introducing stock/flow iconography to the experts consulted during the study. Note that this is a reduced version of the SIR structure [Color figure can be viewed at wileyonlinelibrary.com]

Because the experts were familiar with the epidemiological modelling literature; there, ODEs are the standard usage. Our unusual task was therefore to relate the quite technical but familiar calculus concepts of differentials and state variables to stocks and inflows/outflows icons.

The language of SFDs was important to have at hand. Conceptualisation and formulation involved the iterative struggle to extract information, define the underlying processes, and model them. We first mapped out the pure causal logic. However, as we progressed, we introduced necessary stock and flow variables. For example, the initial version of shellfish transmission did not have the slow accumulation of NV genomes in shellfish, and we came to understand this mechanism and see that it had to be included. As a result, whilst the inputs and outputs remained the same, this model sector eventually evolved to include stocks and flows, two conserved systems, and a co-flow structure (Figures 4). Similarly, the food-handling sector eventually emerged as two complex supply-line structures. In both cases, despite the increased sophistication, this work remained fully comprehensible to all because we had introduced and explained the meaning of the symbols used.

The final workshop allowed the whole group to meet for the first time and discuss, challenge, and correct the model developed by that stage. The five experts all attended, along with members of the project team and an additional member of FSA. People who were highly expert in their particular area were able to come together and discuss mechanisms in what they knew were different areas—yet also knew were all to do with NV.

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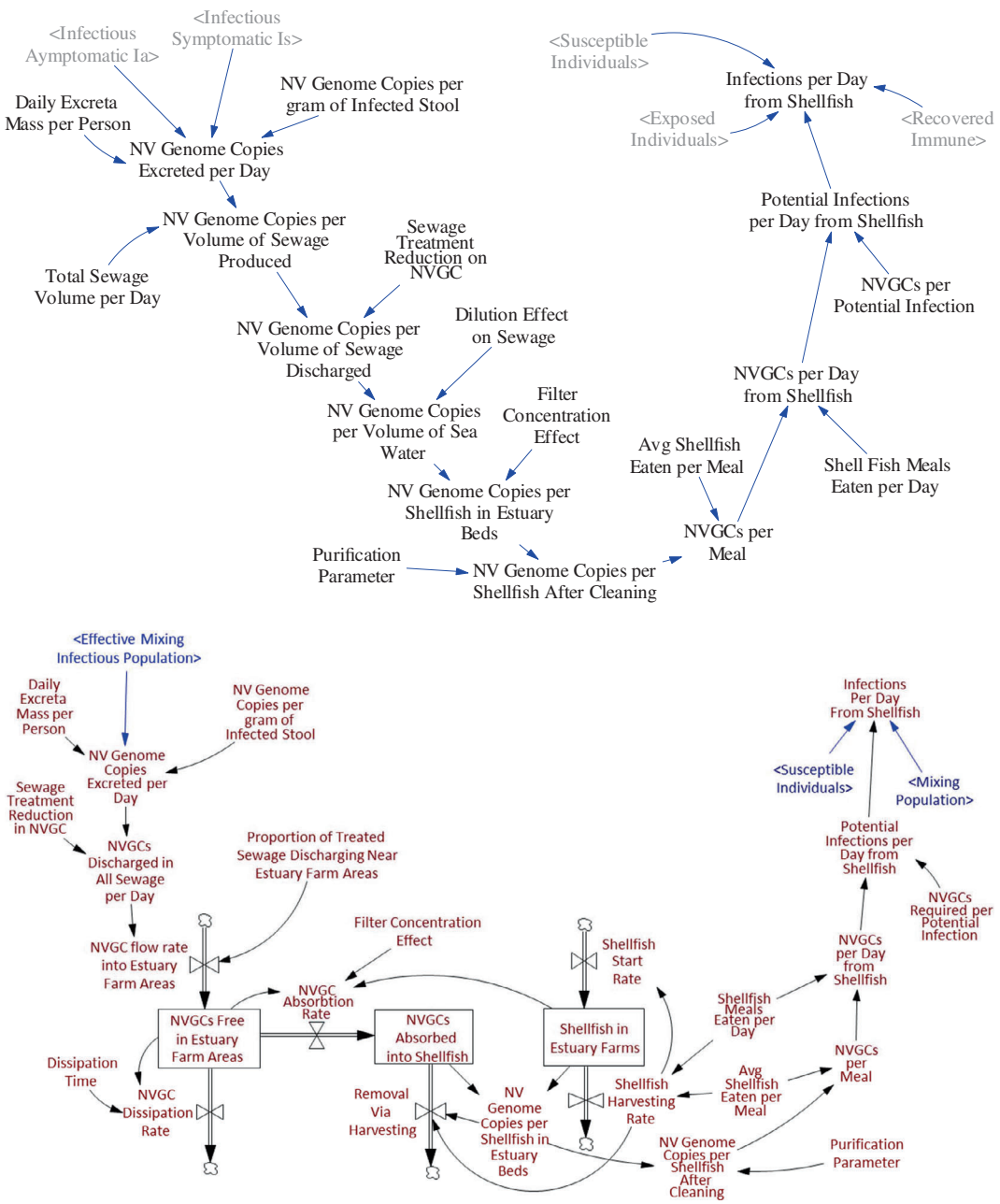

Fig. 4. Diagrams of the "Bivalve Shellfish" sector of the model. The early version (top) captures the pathway as simple causal links. The version in the final model (bottom) has stocks and flows. Note the use of colour to indicate cross-links between the different sectors [Color figure can be viewed at wileyonlinelibrary.com]

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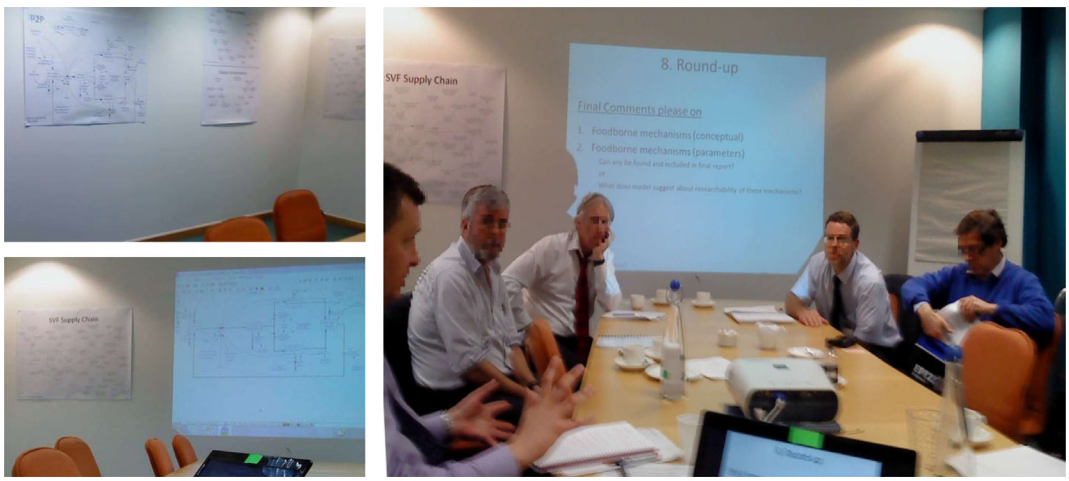

Fig. 5. Images from the final workshop, showing printouts of the model sectors on the walls and some of the group of experts in discussion [Color figure can be viewed at wileyonlinelibrary.com]

In that workshop, we projected the original P2P model onto a central screen and showed our new calibration and our new simulation runs. For the larger, endogenised model we had participants doing live correction via the Vensim model. Questions about some parameter values were raised, and this resulted in the 2 × 2 schema discussed below.

We had also "wallpapered" the room with SFDs of the different sectors (see Figures 5). This meant that although we had a computer projecting parts of the model as we chose to focus on each area, the whole model was available for scrutiny at all times. Additionally, part of the afternoon was spent in a "peripatetic mode": participants were encouraged to gather around the posters for the sectors, the nominal "owner" of each sector exploring and explaining it before they and others crossed out bits of the SFD, correcting what was there by drawing directly onto the posters for later inclusion in the Vensim model. Corrections were also made to parameter values and variable names. It was agreed to leave some known effects out of the model. Detailed minutes of the discussion were taken.

It was striking that this final session was not hard to run. We worried about it beforehand, and it was exhausting to do. However, on the day it was not difficult because participants rapidly "bought into the process." They did seem surprised that we were able to elicit their knowledge, articulate it in diagrams, and share it with them. However, they very quickly saw that they were getting something from the session and engaged strongly with the process. This aspect was a pleasure for us, as FSA had organised a group of people with an enormous amount of experience and knowledge related to NV

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and who were impressive to work with. System dynamics was able to bring something new, something that they had not anticipated but that they really liked. They enjoyed the process, and it succeeded in creating a model which all supported.

Endogenised model structure

To extend the earlier model to include endogenous representations of the foodborne mechanisms we created four entirely new model sectors representing the four main contamination mechanisms.

The first involves bivalve shellfish. NV genomes are excreted by infectious humans, pass into sewage which is treated, and then discharged into the ocean. Over time the remaining genomes are absorbed by bivalve shellfish, such as oysters. Humans eat the shellfish. Cooking normally removes any risk to humans but shellfish are frequently eaten raw and infections can result. Figure 4 (bottom) shows the stock/flow structure of the sector that we developed.

The second sector involves "sludge fertiliser" applied to soil. Usage is carefully regulated but, again, some NV genomes in treated sewage can pass into sludge which is then applied to stimulate crop growth. Some of the surrounding soil may then stick to crops when they are harvested and may remain after washing. The important types of food to consider here are berry fruits and leafy vegetables—BFLV—such as strawberries and lettuce; food that is not cooked. The model calculates a probability that a harvested berry fruit or leafy vegetable portion is contaminated.

A berry fruits and leafy vegetables supply chain is the third sector. This has three stages, each having the potential for transmission. At the harvesting stage, NV crop contamination from sludge, as modelled in the previous sector, can lead to infection of humans. However, genome transmission can also result from infected crop pickers transferring genomes during harvesting. At the food processing stage, contamination is also possible from infected food-processing workers. Lastly, at the use stage—meaning social catering and home preparation—infected food preparers can transmit the virus.

Whilst the previous sector dealt only with some foodstuffs, the final sector had a much broader scope. Other foodstuffs can serve to transmit NV. Many foods can be contaminated if they are prepared by infected people. This is true for home preparation or catering—restaurants, staff canteens, etc. We included such mechanisms in this sector of the model.

The first, third, and fourth sectors described above fold back into the people sector. The exogenous parameter θ is removed, replaced by a new set of infection-rate variables generated by the various foodborne mechanisms. Foodborne transmission is therefore fully endogenised in this model.

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Contributions

The result of this work is a large model—almost 150 variables, 14 of these stocks—of the now endogenised causal mechanisms of foodborne transmission. What contributions does it make to thinking at the FSA?

The first contribution is simply that the model exists, proof that it was possible to build it. Enough was known about the foodborne mechanisms to create a good model that specialists in the area recognise, understand, and accept; a model that is explicit, well documented, and understandable. It was useful for all of these reasons.

Conceptualisation was complete. All of the relationships had explanatory comments and appropriate references to the literature. Formulation was complete: all of the equations were specified. All of the parameters made conceptual sense. However, there were a lot of parameters. All meaningful and measureable in principle but, as the FSA warned us from the start, we could not get values or even estimates for all of the parameters. Consequently, we could not simulate. However, the model is still useful for one more thing.

Exactly because of all the detailed and careful conceptualisation, the formulation, the in-model documentation, and the dimension checking, we were able to think about the parameters and contribute to the NV research agenda and consider policy interventions. For all of the model parameters we asked two questions: do we know the parameter or do we need more research? And is the parameter fixed by nature, or could human behaviour alter it? That yields a straightforward 2×2 matrix (see Table 1). With the experts, we then went through the model and categorised all of the parameters into one of these four boxes. This helped the FSA to think about two things. First, what was still unknown but the FSA might want to find out with future research calls. Second, where policy parameters might be.

It is important to say that it was actually an expert participant who suggested that the model was useful for this. We had introduced the two questions in relation to some parameters of interest; he generalised and expanded upon the idea. This occurred in the final workshop and was immediately supported by the others present. We took this as an indicator of sound model ownership.

Table 1. Framework for the parameters in the endogenised model. This helped organise the FSA's thinking on future research agenda and potential policy levers

	Fixed	**Alterable in principle**
Value Known	Known, Fixed	Known, Alterable
Value Unknown but Researchable	Unknown, Fixed	Unknown, Alterable

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The scale of the foodborne effects: Explorations and implications

The work described in the previous section was supposed to be the substance of the project as commissioned by the FSA. They were very happy at this point, and we could have stopped. In fact, we then had an idea that there was more modelling that we could do. That created a whole new second half to the project, one benefit of which was that it helped us to get a sense of the scale of the foodborne transmission effects when compared with P2P. We describe that work in this section: how we created and calibrated a "compact model," how we explored the sensitivity of that model, and how that exploration gave us insights into seasonality and ideas about policy implications of the modelling.

Calibration of a compact model

We went back to the Lawrence *et al*. model. We made some extensions to that model and found new data values.

The main model extension was a handling of the effects relating to asymptomatic infectives: whether their social mixing rate and their infectivity are different from those of symptomatic infectives. We brought into the model the possibility of both being different using the new compound parameter ω and setting:

$$I = \mathrm{Is} + \varpi \mathrm{Ia}.$$

The analysis that followed was general, allowing ω to take any nonzero value.

Concerning data values, we got two new pieces of data from researchers based at the University of Liverpool. First, a new annual incidence rate of 2,905,278 (95% CI: 2,418,208–3,490,451). Converted to a daily value, we referred to this as the "Observed Incidence Rate." In the model, it is the flow "Infection Development Rate Is" (see Figure 2), although we labelled it Φ in our subsequent algebraic manipulations. Second, the number of cases attributable to foodborne transmission. This was hard to obtain precisely but a best estimate of 73,420 cases per year was now available for use in this study. This, in turn, implied that 0.02527 of all cases were foodborne. We called this the "Foodborne Proportion of Incidence Rate" and labelled it π.

We also obtained a new estimate for the proportion of people exposed to NV who show no symptoms (but are still infectious because they shed the virus). The "Asymptomatic Carriage Proportion", or κ, was 0.003 in the original work. A series of discussions with FSA staff about the different definitions used in the literature lead to an important clarification about quite what parameter was being described. This process was subsequently cited in

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a government report as an example of good practice in ensuring "data validity" (Government Office for Science, 2018). We were then able to identify the value 0.12 in existing literature.

To obtain an idea of the scale of foodborne effects required two steps. The first step involved taking that new, slightly extended P2P model—the compact model—and calibrating it.

We realised that we could use a version of the d'Alembert-Gauss theorem; simply put, we had enough parameters that it became possible to solve the steady-state equations for this model to find closed-form algebraic solutions. The algebra requires care but is straightforward. It produces expressions for the model's key—and previously unknown—parameters β and θ.

It is then possible to calibrate the compact model. For clarity, and to aid comparison with the second step described below, it is worth laying out the underlying logic of this first step. We are starting with "As Is World"; what is actually empirically observable. We are using the three new values, those for Φ, π, and κ, and "processing" them using the assumptions in the model; its stock/flow assumptions, and its other structural relationships as well as its other parameter values. Rigorously consistent with that combination of empirical reality and modelling assumptions, complete steady-state analysis follows. This gives you two things. First, the prevalence of NV: the values of $\overline{S}, \overline{E}$ … etc., that is, how many people would be in each stock in steady state. It also produces, using the above formulae, numerical values for the β and θ parameters that, based on the validity of the model, we take to exist in the world right now. We call these "base case" values β_0 and θ_0.

The compact model is therefore completely specified. Moreover, it is, we believe, the first empirically calibrated model of NV transmission.

However, a second step is possible.

Exploring scenarios and sensitivity

The second step involves reversing the above analysis to get an idea of the scale of the different transmission effects, how incidence depends on the foodborne mechanisms. This allows us to generate scenarios and to explore sensitivity to key parameters.

To do this, we move into the "What If World." We now assume values of β and θ. We "process" them using the compact model's assumptions about stocks/flows and other causal connections as well as established parameter values. Then, rigorously consistent with this combination of scenario assumptions, known parameters, and modelling assumptions, we can do complete steady-state analysis. These newly derived formulae give the prevalence of NV—the number of people in each stock at steady state. However, consistent with this we also obtain formulae which can be used to give numerical values of the incidence rate, Φ, and the foodborne percentage, π.

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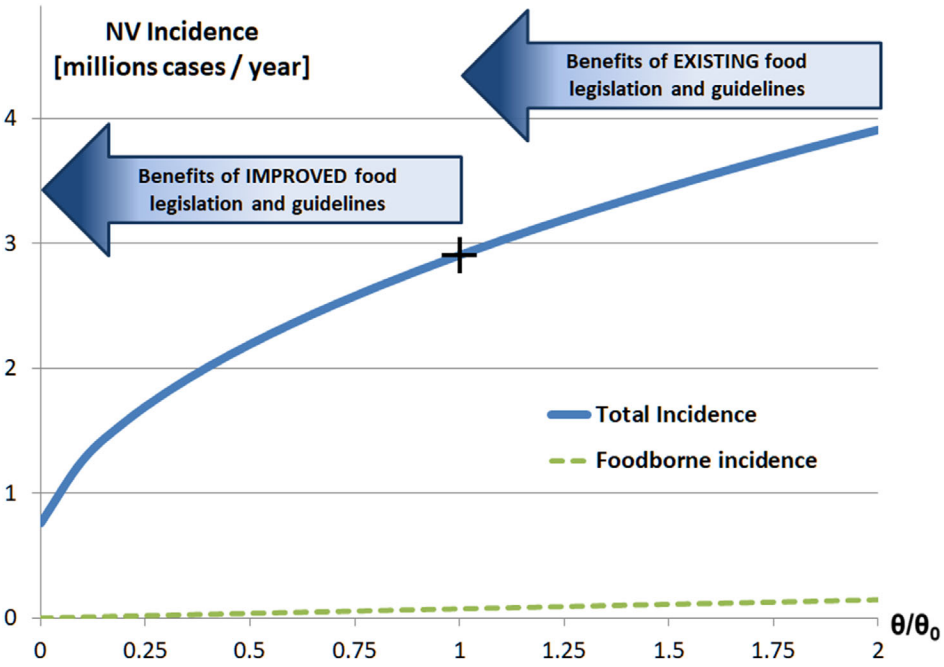

Fig. 6. Scenarios showing how foodborne NV incidence and total NV incidence vary with the FB effect parameter, θ. Presented as a semi-spiderplot [Color figure can be viewed at wileyonlinelibrary.com]

This version of the steady-state analysis involves even worse, complex algebra. Prof. James D. Murray FRS, would urge students confronted with such a situation to knuckle down to the effort needed for this stage and press on to the interesting results; "Ach, this is just tedious algebra."

The algebra, whilst tediously intricate, is technically straightforward. What it delivers is a sense of how prevalence ($\overline{S}$, $\overline{E}$ … etc.,) and incidence and its constitutive elements Φ and π depend on different βs and different θs. In other words, how they depend on P2P versus foodborne mechanisms.

Figure 6 shows how incidence changes with foodborne parameter θ. In this semi-spiderplot, the base case is represented by the point (1, 2.9), that is $\theta = \theta_0$ and total annual incidence is 2.9 million. As we would expect, examining the $X = 1$ values, most incidence results from P2P infection (upper line); only 2.5% result from foodborne transmission (lower line). However, the interest is in varying θ.

If θ is doubled, then NV incidence increases by a third. One way of thinking of this is to consider the interval $X > 1$, the right-hand side of the plot. This gives a sense of the benefits we have already gained, that is, reductions in norovirus transmission resulting from people following existing legislation on food preparation and also following guidelines on food harvesting, handling, and processing. Taken together, all of these have increased the hygiene associated with food and so reduced norovirus transmission to the

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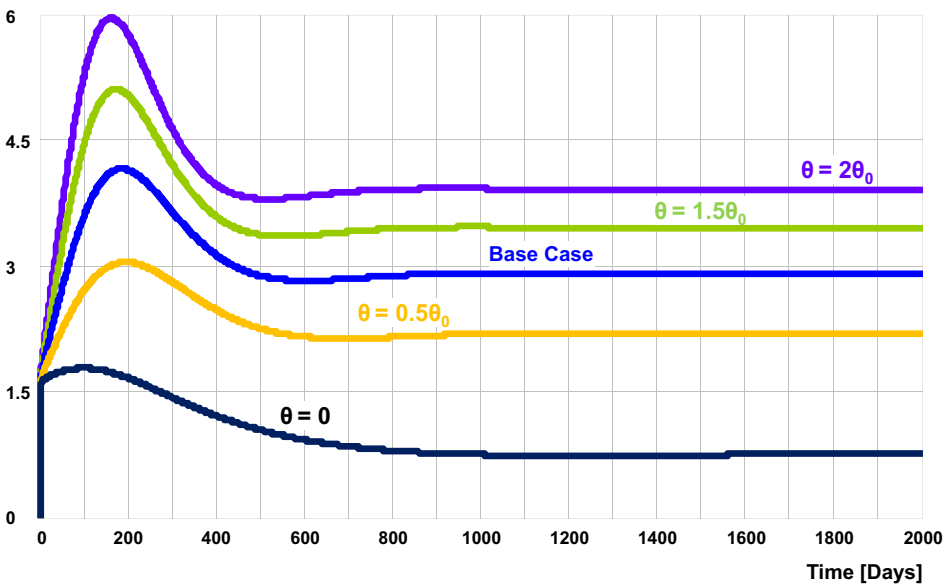

Fig. 7. Runs of the compact model exemplifying the sensitivity of NV incidence to θ, the foodborne infectivity parameter. The model is initialised with arbitrary stock values and the different θ values and then run until it settles to steady state. Note the response time [Color figure can be viewed at wileyonlinelibrary.com]

point we see today. Now look at the effect of dropping θ to 0 (so all remaining incidence is P2P). In this scenario, NV is reduced by 75%, a striking nonlinear response. The interval on the left, X∈[0,1), therefore gives an idea of what we could gain if we further improved food hygiene legislation and guidelines. The FSA are, therefore, quite right to look into reducing foodborne transmission effects; considerable benefits can be obtained.

We simulated the model to see this sensitivity in action. Figure 7 shows some exemplifications of the sensitivity of NV incidence to θ. Arguably these runs really just confirm the validity of the steady-state calculations. However, they give something new, the response time of the system. The responses to different values of θ all play out over 1 to 2 years. That is a short time for a (public) policy to achieve an effect.

However, from where we are now—at the point (1, 2.9) in Figure 6—this response curve is quite flat.

This is worth comparing with the curve of changes in P2P infectivity—Figure 8. Here the logarithmic Y-axis hints at the very different sensitivity to β. In this spiderplot, the base case is represented by the point (1, 0), that is $\beta = \beta_0$, and total annual incidence is its base case value of 2.9 million.

If β is doubled, NV increases to 15 times its base case value. Again, the right-hand side shows what we have already gained from getting people to wash and dry their hands after going to the toilet, after being around sick people and small children, etc. If β falls to 0, then NV incidence is reduced by 97.5%. All remaining incidence is foodborne. That region where $X < 1$

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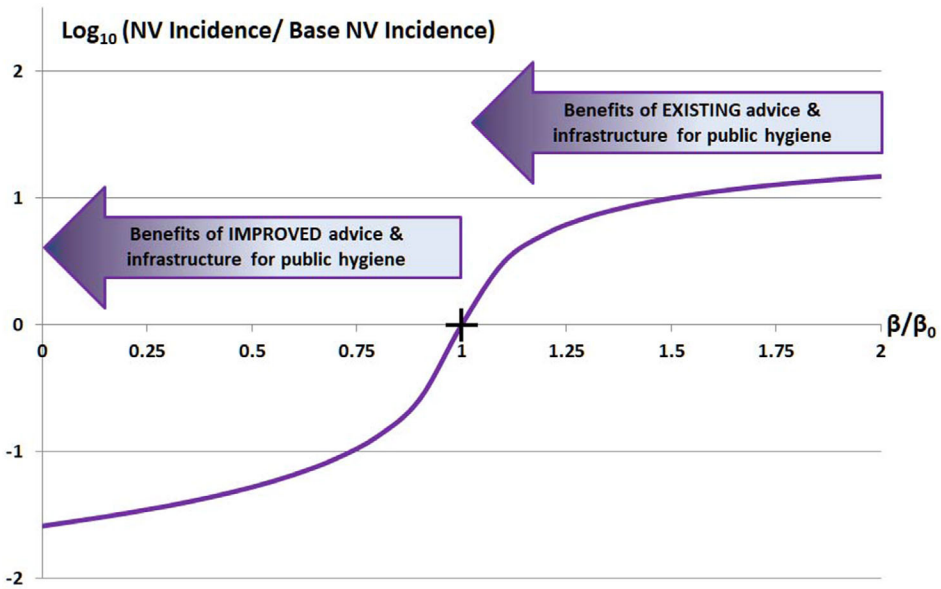

Fig. 8. Scenarios showing how total annual NV incidence varies with the relative size of the P2P effect parameter, β. Presented as a spiderplot with a logarithmic vertical scale [Color figure can be viewed at wileyonlinelibrary.com]

shows what we could gain if we improve personal hygiene even more. Note that from where we are now, this curve is very nonlinear. If you increase β by just 10% you triple the NV incidence. If you decrease β by 25%, incidence collapses to 10% of its current value. That is a huge reduction in misery and work lost. Figure 9 shows simulated exemplifications of the high sensitivity of NV incidence to β.

Having explored these scenarios and got a sense of the sensitivity of the compact model to β and θ, what can we learn? Two insights follow. These are considered below.

Gaining insights: Source of seasonality?

As we said at the start, NV incidence exhibits a strong seasonal variation (see Figure 1). To analyse this further, we examined 11 years of data. We found the average pattern over a year. We formulated this as a seasonal trend as follows. We set the seasonal trend as the average multiplied by a seasonal factor, a repeating pattern with average one. For the factor, we fitted a quintic polynomial. This preserved the repeating pattern, found the position of the turning points, and implemented the requirement that it integrate to unity. We found that the best-fit seasonal factor shows a tenfold variation between peak and trough: around an average of one, the trough is only $^1/_5$ whereas the peak is around $2^1/_3$ (Figure 10, left).

A factor of 10 is hard to explain in terms of changes in human behaviour over the year. However, prompted by the sensitivity of model output to values

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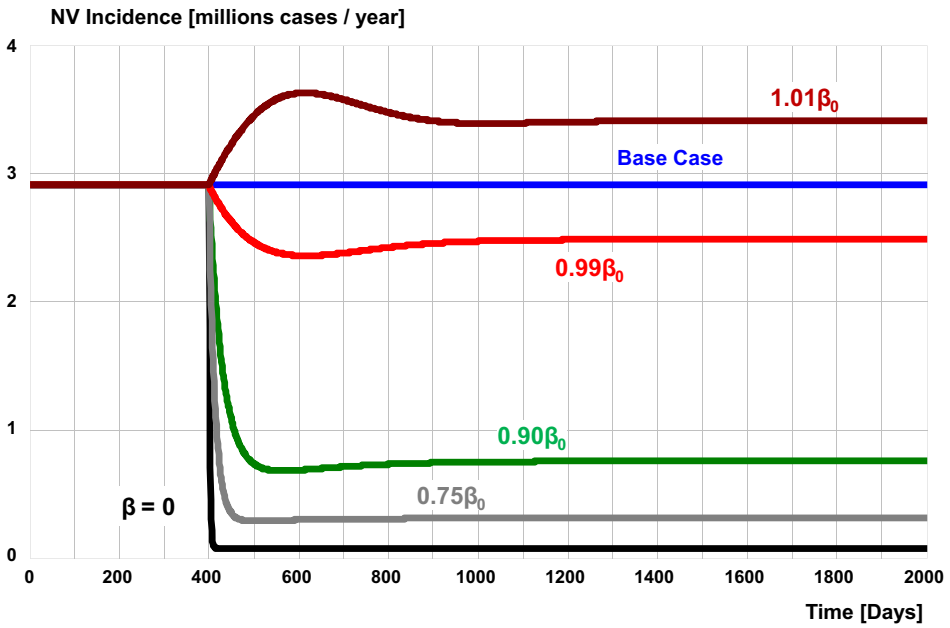

Fig. 9. Runs of the compact model exemplifying the sensitivity of NV incidence to β, the P2P infectivity parameter. The model runs with base case values until time $t = 400$, when the new values of β are implemented as a step-change [Color figure can be viewed at wileyonlinelibrary.com]

of β, we asked the question: what seasonal pattern of β would produce this amount of seasonality in NV incidence? The algebra from step two above gives the answer. The seasonal factor for P2P effects, the necessary multiplier for β, is also shown in Figure 10, left. Its scale is very different: consistent with the spiderplot in Figure 8, this analysis shows that β needs to rise by only 6%, or fall by only 14% of its base value to produce the observed seasonality.

To simulate this analysis, we used that seasonal pattern for β in the extended and recalibrated P2P model, the compact model. We added a small amount of pink noise for realistic stochastic variability and used the resulting time series for β as an exogenous input.

It should be said that this is a somewhat counterintuitive move for system dynamicists, who normally wish to see complex behaviour being generated endogenously. However, the aim here is to probe further the range of behaviours possible, to see what the compact model could do. Note also that the calculated values of β are only consistent with the NV seasonal trend in snapshot, or steady-state terms. Their effect when used to simulate a dynamic model involving integrations around the various stocks was expected to be somewhat different.

What we discovered is simply stated but nevertheless significant: the compact model reproduces the observed seasonal behaviour well (Figure 10, right). Note that this graph is in cases/day to encourage comparison with Figure 1. Here, then, we have a candidate explanation for the source of NV's very distinctive seasonality. NV incidence may change by an order of magnitude across a year

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but in human behaviour terms, you only need a 106% peak and an 86% trough to produce this. That is much more plausible and easier to explain (people staying indoors more in winter and so mixing a little more closely).

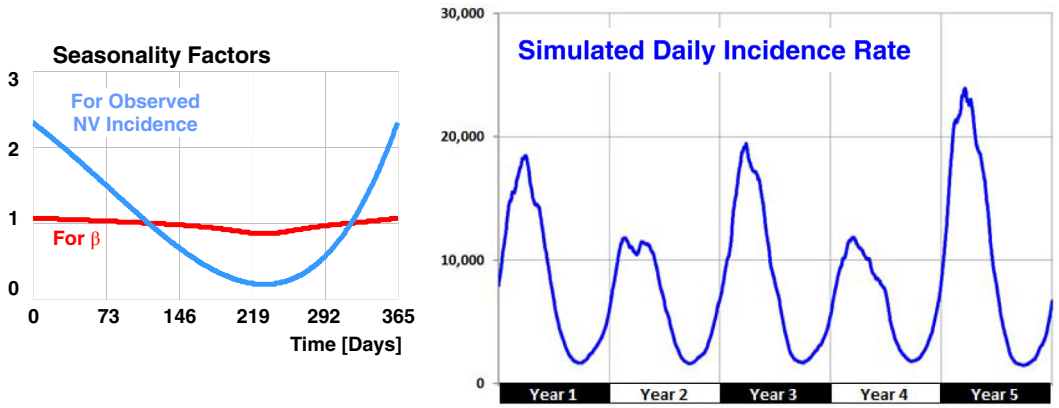

Fig. 10. Understanding NV seasonality. *Left: Seasonal factors for incidence (extracted from empirical data) and for β (established algebraically). Right: Run of the compact model showing daily simulated NV incidence generated by the β seasonality trend. Compare with Fig. 1.* [Color figure can be viewed at wileyonlinelibrary.com]

Gaining insights: Implications for policy focus

The sensitivity analysis generated a second insight. It is clear that θ and β have very different sensitivities. This really shows up when the two are put on the same graph—Figure 11. Both curves pass through the point (1, 2.9), representing the base case. With the linear vertical scale, the sharpness of the β response is very clear—but it is the difference in marginal gains around the status quo that is most striking. Of course, this horizontal axis is parameter change, it is not money, or difficulty, or effort; it is not the whole story about what has to be done in reality to bring about these changes. Nevertheless, it makes a challenging point to the FSA: it shows that the scale of the foodborne effects is small with respects to the P2P. That then raises the question of quite where the FSA's policy focus should be. We come back to this in the final section.

Summary of findings, implications, and reactions

We close with a summary of findings; these relate well to the initial aims. We also explore the implications of those findings, combining this with some client reactions.

To start with findings, the first is that modelling is possible. What is known about foodborne mechanisms can be elicited, shared, and represented in a large system dynamics simulation model. On the pages of this journal that

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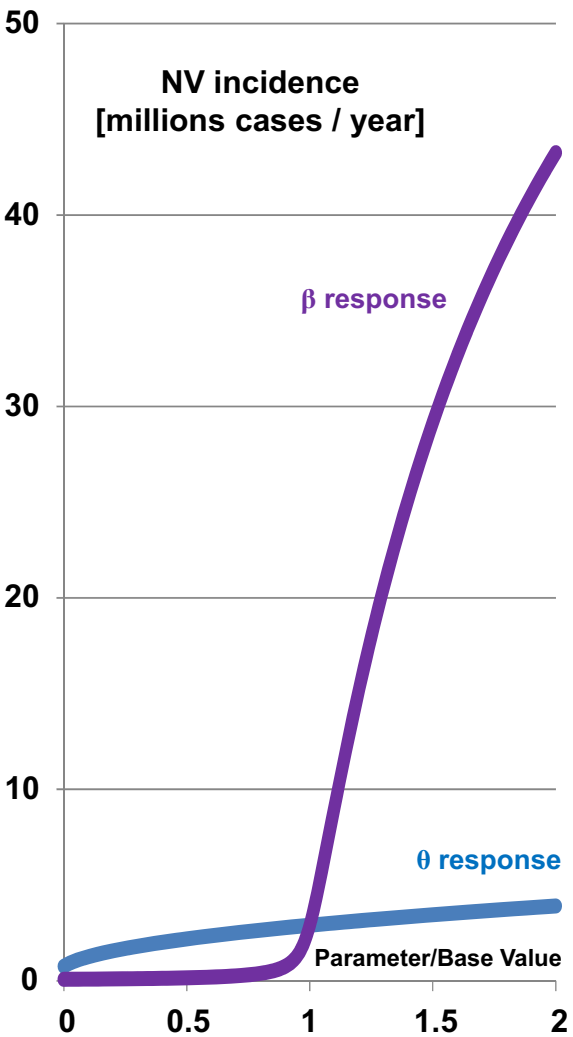

Fig. 11. Comparing how NV incidence varies with the P2P parameter β and the foodborne parameter θ. Presented as a semi-spiderplot. [Correction added on 26 October 2022, after first online publication: figure 11 has been corrected in this version.] [Color figure can be viewed at wileyonlinelibrary.com]

may seem obvious but it was not clear to the FSA at the start of the project that such pooling of knowledge would be possible. Second, modelling is useful. Workshop participants stated that it helped them to understand the various pathways of infection, filling knowledge gaps, and framing further research requirements. The third finding is that the modelling is "parameter hungry," that not all of the parameters are known and judgemental estimates resisted elicitation. However, a compact model can still give insight into sensitivities. This forth point merits a longer description.

The final, 100 pages, report was internally reviewed by anonymous FSA experts. One of them wrote "the modelling work identified important sensitivities,

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non-linear effects, and parameter uncertainties." Flowing from this finding, we showed that modelling offers a possible explanation for the strong seasonal pattern of NV. Additionally, we showed that a compact model can act as an organising, or prioritising, framework for discussions on where to focus policy—and that that framework suggests a shift, a broadening, in policy focus. The modelling offers the systems insight that we are at a point where we see the best gains not just from reducing foodborne effects but also from improving P2P hygiene. Remember, however, that foodborne effects are what the FSA is interested in: improved toilet facilities for fruit pickers, guidelines for shellfish safety, etc. This work challenges that. Our modelling work suggests that those things might possibly be less effective than P2P-related interventions, that in tackling NV, interventions related to food, whilst effective, may not be "where the action is." Instead, it might be better to include P2P effects, that is, to improve personal hygiene both in terms of advice and available infrastructure to reduce β on the ground. An example is hygiene-related infrastructure: getting in place more automatic taps encourages hand cleaning, and makes cleaning more effective.

The work's implications were also evaluated by Professor Rick Mumford, FSA Deputy Director of Science and Head of the Science, Evidence and Research Division.

First, he referred to the research contribution, including the 2×2 analysis: "as well as generating key outputs, the project identified a number of key knowledge gaps. These insights have fed into and helped to prioritise the FSA's NV research programme. In addition, some of the parameters populated in this model have since been used in other FSA projects." (R. Mumford, personal communication, July 7, 2020).

Second, he noted the creation of the system dynamics model and also the contribution of the "Process maps," the SFDs showing the pathways of infection. He described how the FSA, **"**plan[s] to revisit these maps to understand where effective interventions may be possible in the [leafy vegetable and catering] supply chains."

Third, he remarked on the dissemination of the work. He cited its presentation at the "FSA Conference on Foodborne Viruses Research," an international meeting held in London. He observed more generally that it "also forms part of a larger body of evidence on this pathogen, which is helping us to raise awareness of its importance."

Finally, he noted the broadening of policy focus that the work recommended and gave an example of a tangible consequence: "This project highlighted the benefit of a cross-government approach to tackling norovirus. Since the report, the FSA agency has produced a forecasting model for norovirus outbreaks using Twitter data. When an outbreak is forecast, the FSA contacts NHS Choices and together we run joint consumer messaging on hygiene practices covering both food and person to person sources. The model has helped us drive this collaborative approach." (NHS Choices is a National Health Service website which offers an A-Z guide on conditions and treatments.)

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This broadening of policy focus, the "cross-government approach," is a core recommendation of the final report—and an archetypal systems insight. The anonymous reviewers certainly understood this; "intervention in person-to-person virus transmission and associated public health policies, whilst falling outside the FSA's remit, could be as important as foodborne vectors." That is a challenging thing to find—and to get across. This, and the other insights produced by this study, were grounded in system dynamics modelling. They also relied on good client engagement, in itself a prerequisite for applied work to have any effect.

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References

Alba S. 2022. Blurring the lines: Journalism and epidemiology in the time of corona. *Significance* **19**: 30–33. https://doi.org/10.1111/1740-9713.01611.

DOI: 10.1002/sdr

Boulding KE. 1956. General systems theory – the skeleton of science. *Management Science* **2**: 197–208.
Farrar J, Ahuja A. 2021. *Spike: The Virus Vs. the People – the Inside Story*. Profile Books: London.
Forrester JW. 1961. *Industrial Dynamics*. MIT Press: Cambridge, MA.
Forrester JW. 1968. *Principles of Systems*. MIT Press: Cambridge, MA.
Forrester JW. 1969. *Urban Dynamics*. MIT Press: Cambridge, MA.
Gilbert S, Green C. 2021. *Vaxxers: The Inside Story of the Oxford AstraZeneca Vaccine and the Race against the Virus*. Hodder & Stoughton: London.
Government Office for Science (2018) *Computational Modelling: Technological Futures* (authors: Walport, M., Calder, M., Craig, C., Culley, D., de Cani, R., Donnelly, C., Douglas, R., Edmonds, B., Gascoigne, J., Gilbert, N. Hargrove, C., Hinds, D., Lane, D. C., Mitchell, D., Robertson, D., Rosewell, B., Sherwin, S., Wilson, A.). Government Office for Science: London. Available from: www.gov.uk/government/publications/computational-modelling-blackett-review.
Horton R. 2022. Offline: COVID-19 as culture war. *The Lancet* **399**: 346.
Kermack WO, McKendrick AG. 1927. A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London A* **115**: 700–721.
Lane DC. 1999. Social theory and system dynamics practice. *European Journal of Operational Research* **113**: 501–527.
Lane DC, Husemann E. 2008. Steering without Circe: Attending to reinforcing loops in social systems (2007 Jay Wright Forrester award address). *System Dynamics Review* **24**: 37–61.
Lane DC, Husemann E, Holland D, Khaled A. 2019. Understanding foodborne transmission mechanisms for norovirus: A study for the UK's Food Standards Agency. *European Journal of Operational Research* **27**: 721–736 Free download: www.sciencedirect.com/science/article/pii/S0377221718310142.
Lawrence L, Kerrod E, Gani R, Leach S. 2004. *Microbiological Risk Assessment for Norovirus Infection – Contribution to the Overall Burden Afforded by Foodborne Infections*. Food Standards Agency: London.
Murray JD. 1989. *Mathematical Biology*. Springer-Verlag: Berlin.
Reicher SD. 2021. COVID-19 has sparked new relationships between academia and policymakers – We must maintain them. *The Conversation* Feb 26, https://theconversation.com/covid-19-has-sparked-new-relationships-between-academia-and-policymakers-we-must-maintain-them-156143.
Spiegelhalter D, Masters A. 2021. *Covid by Numbers: Making Sense of the Pandemic with Data*. Pelican Books: London.
Thompson KM, Duintjer Tebbens RJ. 2007. Eradication versus control for poliomyelitis: An economic analysis. *Lancet* **369**: 1363–1371.
Tooze A. 2021. *Shutdown: How COVID Shook the world's Economy*. Allen Lane: London.
Vennix JAM. 1996. *Group Model-Building: Facilitating Team Learning Using System Dynamics*. Wiley: Chichester.
Vennix JAM. 2000. Group model-building: Tackling messy problems (Jay Wright Forrester prize 2000 lecture). *System Dynamics Review* **15**: 379–401.
von Bertalanffy L. 1950. An outline of general system theory. *Brit. Journal Phil. Soc.* **1**: 134–165.

DOI: 10.1002/sdr