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ORIGINAL ARTICLE

Coupling fishery dynamics, human health and social learning in a model of fish-borne pollution exposure

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Abstract Pollution-induced illnesses are caused by toxicants that result from human activity and are often entirely preventable. However, where industrial priorities have undermined responsible governance, exposed populations must reduce their exposure by resorting to voluntary protective measures and demanding emissions abatement. This paper presents a coupled human–environment system model that represents the effects of water pollution on the health and livelihood of a fishing community. The model is motivated by an incident from 1949 to 1968 in Minamata, Japan, where methylmercury effluent from a local factory poisoned fish populations and humans who ate them. We model the critical role of risk perception in driving both social learning and the protective feedbacks against pollution exposure. These feedbacks are undermined in the presence of social misperceptions such as stigmatization of the injured. Through numerical simulation and scenario analysis, we compare our model results with historical datasets from Minamata, and find that the conditions for an ongoing pollution epidemic are highly unlikely without social misperception. We also find trade-offs between human health outcomes, the viability of the polluting industry and the survival of the fishery. We conclude that an understanding of human–environment interactions and misperception effects is highly relevant to the resolution of contemporary pollution problems, and merits further study.

Keywords Human–environment interactions · Social learning · Pollution abatement · Misperception effects · Trade-offs

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Introduction

A human–environment system (HES) is characterized by the interaction of human activities and natural processes (Liu et al. 2007; Levin and Clark 2010), and may also be called a social-ecological system (SES) when biota are involved (Anderies 2015). Pollution effects on populations and natural resources and the human responses to those effects can exemplify human–environment interactions: human activities can cause an increase in pollution, which in turn can feed back to change human perception and behaviour, and possibly stimulate efforts to control the pollution.

For some applications, mathematical modellers assume that a policymaker wishes to ameliorate environmental impacts, and has the power to make a decision under constraints. Under these conditions, it is possible to use optimal control theory to understand how the policymaker can optimize the amelioration efforts. This approach is used in the context of pollution HESs and similar systems involving humans and their environment (Withagen 1995; Eichner and Pethig 2006; Dubey 2010; Heijnen and

Wagener 2013; Anderies 2015). However, for other applications, it may be desirable to incorporate forces into the model that cause policymakers to act in the first place, such as public pressure.

For example, legal theorists who study the regulatory compliance of polluting industries observe that government policy may not be enforced effectively without pressure from citizens (Van Rooij 2010). Social misperception can be the single largest obstacle to resolving pollution problems. Sometimes this is fuelled by an inconclusive scientific understanding, given that the symptoms of pollution exposure can be slow to appear, and difficult to separate from other causes or confounding variables (Van Rooij 2010; Harada 1972; Grandjean et al. 2010). Misperception may also be fuelled by economic expediency and deliberate foul play. Therefore, citizen action and changing social perception play a large role in helping to resolve pollution problems.

Recognizing that policy decisions depend on social influences and behaviours that emerge from group interactions, social learning models are increasingly used in mathematical epidemiology to study vaccine scares (Bauch and Bhattacharyya 2012), and in ecology to study resource management and conservation (Satake et al. 2007; Innes et al. 2013; Barlow et al. 2014). The idea is that individuals adopt strategies based partly on their own self-interest, and partly from exchanging information with others in a society to imitate the normative behaviour of the group (Satake et al. 2007).

We are interested in incorporating social learning into an HES model of pollution impacts. Our motivating example comes from Minamata, Japan, where a chemical factory operated by the Shin Nitchitsu (Chisso) corporation emitted methylmercury directly into Minamata Bay (Jesty 2012; George 2001; Ui 1992; Yorifuji et al. 2013). This pollutant posed a hazard to humans and organisms due to its high protein-binding affinity and its potential to bioaccumulate rapidly in food chains (Goel 2006; Jackson 1998). As fish was the main source of dietary protein for people in Minamata and was ingested in high quantities (George 2001; Laws 2000; D'Itri 1991), it was not long before people started showing severe neurological symptoms linked with eating fish from the bay. To avoid financial responsibility and pursue record high production, Chisso and its allies in Japan's Ministry of International Trade and Industry concealed test results and stalled independent research that linked its emissions to the illness (Harada 1972; Ui 1992; Smith and Smith 1975).

Faced with ambiguous information and a lack of leadership from the government and industry, the people in Minamata had to resort to their own decision-making and voluntary measures to protect their health. These voluntary actions included fish-eating boycotts and protests to demand pollution abatement. By 1960, fish-eating was reduced, and protests had compelled the government to intervene (Yorifuji et al. 2013; Littlefield 1996; Harada 1995; George 2001; Smith and Smith 1975).

Although citizens had acted and secured government attention, the emissions were not completely abated. The pushback from those who supported the company was significant. Many citizens were reticent to blame pollution on a company that they depended upon for their income (Van Rooij 2010), and many viewed the existing pollution victims and the appearance of new victims as a threat to their livelihood. Workers feared that the factory could be shut down (George 2001), and fishermen feared that their fish would not sell (Smith and Smith 1975). This fed a social stigmatization that dissuaded the injured from coming forward with their symptoms, so that the true extent of the damages remained hidden for many years. Believing that the danger was over after 1960, the community regained confidence in fish-eating even though the pollution continued (George 2001; Harada 1972). After 1960, nearly 200 people developing the full set of mercury-poisoning symptoms were overlooked, while thousands more were exposed (Harada 1995; George 2001; Harada 1975). This latent pollution epidemic continued until 1968, when a second methylmercury-poisoning incident was discovered in another region of Japan, prompting the government to recognize the pollution problem in Minamata.

Although the Minamata incident occurred 50 years ago, it is very relevant to contemporary pollution problems. Minamata represents highly localized human–environment interactions, and gives us an opportunity to understand both the pollution feedback effects on human health and fishing, and the social feedbacks in response to the pollution.

To model the human injuries that result from fish-borne pollution exposure, we observe that few differential equation models study human pollution illnesses. We draw insights from existing ecological and epidemiological approaches to create something new. Models of infectious diseases typically use SIR population compartments or agent-based social contact networks to represent the transmission of communicable illness (Keeling and Eames 2005). In ecology, pollution effects are customarily modelled as an accumulation process that induces mortality or morbidity into the exposed population (Dubey 2010; Hallam and Clark 1983; Hallam et al. 1983). In our model, we develop an accumulation equation to represent the growth of an explicit compartment for pollution-induced injuries.

While medical researchers recognize the role of stigma in prolonging epidemics, especially when it shames people from seeking medical attention (Des Jarlais et al. 2006; Van Brakel 2007), it is not clear that these insights have been readily taken up in epidemiological models. By

comparison, risk perception and behavioural strategies to avoid infectious disease are more widely modelled (Perisic and Bauch 2009), and some agent-based models explicitly distinguish between injured and perceived injured (Poletti 2012). However, these methods have not yet been applied to understanding pollution illnesses, in spite of the fact that pollution is inherently social both in its cause and its cure. We formulate feedbacks involving stigma and fish-eating boycott that represent a break from existing approaches.

The HES model that we present in this paper couples phenomena that up to now have been modelled only separately, and introduces new links that allow us to ask new questions. Using differential equations, we incorporate social learning into the HES by representing pollution abatement as a dynamic function of the aggregate public demand for it, rather than as a static optimal control parameter. This allows us to examine the role of changing social attitudes, pressures, and misperceptions in the effort to control pollution damages. The demand for abatement changes in response to the changing perception of health risk (Bauch and Bhattacharyya 2012; Innes et al. 2013).

Our objectives are to examine the ecological and social conditions that cause the outbreak of a pollution-induced epidemic, and to study the role of social feedbacks and misperception effects that allow the epidemic to persist.

Methods

This model couples three subsystems: the consumer-resource dynamics of fish harvesting; the growth of pollution injuries from fish-eating, and the rate of perception of these injuries; and the social learning dynamics underlying the public pressure to abate pollution. Since parts of this model are developed here for the first time, we provide a detailed model derivation in Sect. S1 of the Supplementary Material.

To maintain our focus on numerical simplicity and qualitative behaviour, we build a system of normalized equations whose state variables are proportions rather than absolute quantities. The system is driven by an exogenous input $n(t)$, which is the baseline emissions loading rate from an industry into a water source. Whereas $n(t)$ is a prescribed function, the net emissions loading $E(t)$ is affected by feedback from the social demand for abatement, as seen in Fig. 1. The fishery consists of fish $F(t)$ and boats $B(t)$, whose interactions are governed by Lotka–Volterra type equations and emissions mortality. We remark that these equations represent an "open-access fishery", in the sense that the number of boats grows as large as catch revenues and costs will allow. Although the fishing rights in Minamata belonged to the local fishermen's cooperative, the cooperative itself was largely self-regulated (George 2001; McIlwain and Smith 2013). Following World War II, food shortages coupled with the arrival of new boat and net technologies compelled fishermen to increase their fishing effort as much as they could (Takahashi 2013; Keibo and Masato 2001).

Human injuries are driven by the fish-borne pollution exposure. This exposure depends on the net emissions level $E(t)$, the fish catch $HF(t)B(t)$ with harvest rate H, and a factor of pollution availability to humans, ϵ_I.

To model human perception, we introduce $P(t)$ for the perceived injured and $I(t)$ for the actual injured. The parameter s is the stigma rate, or the rate at which the community denies new victims as the perceived injured $P(t)$ increases. This stigma effect causes a disparity between the actual and perceived number of injured. If there is no stigma, $s = 0$, then $I(t)$ and $P(t)$ grow at the same rate. However, when $s > 0$, then a distinct negative feedback is introduced in P': the rate at which the community is willing to recognize new victims decreases as the number of perceived victims increases. Then $P(t)$ is an underestimate of the actual health damages $I(t)$.

As Fig. 1 shows, all social feedbacks and decisions are driven by $P(t)$, but they are blind to $I(t)$. $P(t)$ affects the level of fish-eating, $1 - bP(t)$, where b is the boycott rate. $P(t)$ also influences the public demand for abatement $X(t)$, which grows through social learning in the form of an imitation game (Hofbauer and Sigmund 1998). We assume that citizens sample the preferences of others and aim to minimize a cost function, $hP(t) - \beta X(t)$, by choosing either to favour abatement or non-abatement. κ is a sampling and imitation rate, h measures the health costs from pollution, and β reflects costs to productivity and job security that is claimed by the industry and its supporters in response to growing X. In turn, the population preference $X(t)$ feeds back to influence the net emissions $E(t)$, thus closing the loop.

Altogether, the equations for the HES are:

$$\left.\begin{aligned} F'(t) &= rF(t)(1 - F(t)) - HF(t)B(t) - \epsilon_F E(t)F(t) \\ B'(t) &= (1 - bP(t))HF(t)B(t) - cB(t) \\ I'(t) &= \epsilon_I E(t)HF(t)B(t)[1 - bP(t) - I(t)] \\ P'(t) &= \epsilon_I E(t)HF(t)B(t)[1 - bP(t) - sP(t) - P(t)] \\ X'(t) &= \kappa X(t)(1 - X(t))[hP(t) - \beta X(t)] \end{aligned}\right\} \quad (1)$$

where the net emissions loading $E(t)$ is given by:

$$E(t) = n(t)(1 - f(X(t))) \quad (2)$$

Note that the form of the abatement function $f(X)$ is important. It should be monotone increasing in X, and have a social concern threshold $\breve{x}$ above which there is complete abatement: i.e. whenever $X \geq \breve{x}$, then $f(X) = 1$ and $E(t) = 0$. Table 1 gives a summary of the model variables and parameters. See the Supplementary Material for more details.

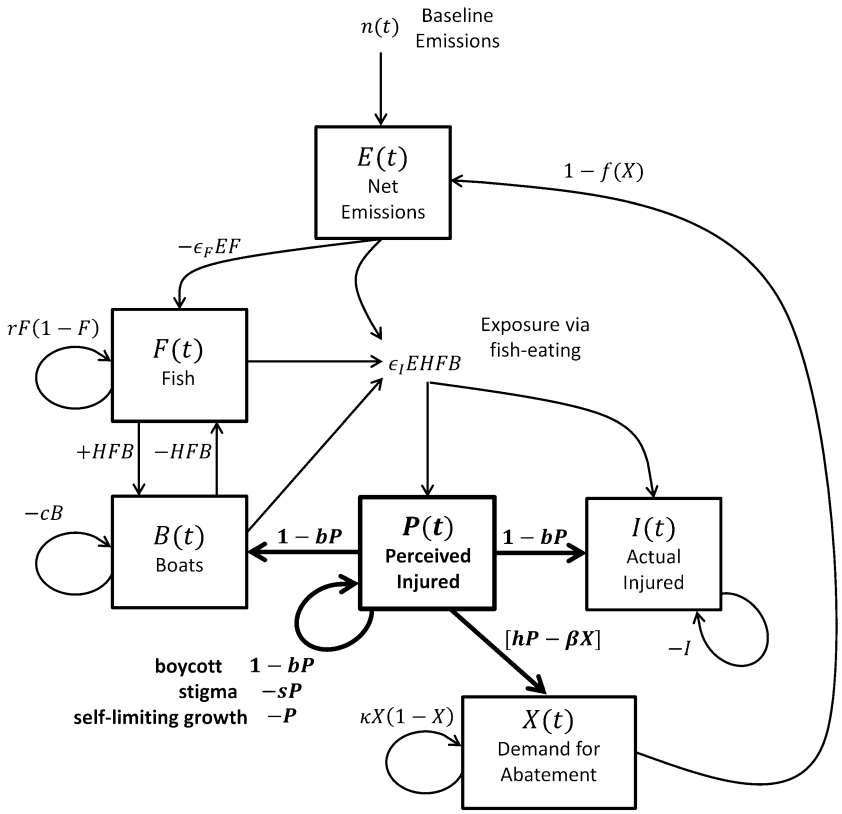


Fig. 1 Causal links and feedback loops among the state variables in the human–environment system. Observe the critical role that human perception plays (shown in *bold*) as a hub for the relay of information. The stigma term $-sP$ acts to dampen the growth of the perceived injured, creating an underestimation of the actual injured. This distorts the signals needed for the fish-eating boycott $-bP$ and the demand for pollution abatement X to fulfil their protective functions

Table 1 HES model variables and parameters

Variable	Meaning	Units	Range
E	Net emissions loading	Proportion	[0, 1]
F	Fish	Population proportion	[0, 1]
B	Boats	Proportion of $B_{\text{threshold}}$	≥ 0
I	Cumulative injured	Population proportion	[0, 1]
P	Cumulative perceived injured	Population proportion	[0, 1]
X	Demand for pollution abatement	Population proportion	[0, 1]
$f(X)$	Abatement level	Proportion	[0, 1]
Parameter	**Meaning**	**Units**	**Range**
H	Harvesting rate	1/year	[0, 1]
r	Fecundity	1/year	(0, 1]
c	Normalized boat costs	1/year	(0, 1]
ϵ_F	Fish pollution mortality	1/year	[0, 1]
ϵ_I	Pollution availability to humans	1/(unit emissions)	[0, 1]
b	Rate of fish boycott per unit injury	1/(fraction increase in P)	≥ 0
s	Rate of stigmatization per unit injury	1/(fraction increase in P)	≥ 0
κ	Sampling and imitation rate	1/year	[0, 1]
h	Rate of health concern per unit injury	1/(fraction increase in P)	≥ 0
β	Pushback to demand for abatement	1/(fraction increase in X)	≥ 0
$\hat{x}$	Social concern threshold	Population proportion	[0, 1]

As mentioned in the historical background of Minamata, the emissions may have continued indefinitely (albeit at a reduced, but still dangerous level) and the pollution victims forgotten, had it not been for a singular event that our model does not capture. That event is the 1965 outbreak of methylmercury poisoning in Niigata, Japan, at an

acetaldehyde factory run by a different company far from Minamata.

Then, in our simulations and analysis, we are interested in the conditions for a nontrivial equilibrium where the emissions and the fish catch coexist (i.e. the route of exposure remains open), and where the true number of injured I continues to grow in the background long after the perceived injured P has settled down to its steady state. Our model does not have a mechanism to resolve this equilibrium: strictly speaking, an equilibrium where injuries grow indefinitely without causing a problem is unrealistic. However, the lax regulatory conditions that permitted the Minamata epidemic to occur seem unrealistic as well, or at least surreal, in hindsight. Rather, designing the model in this way allows us to discover how such a condition could occur in the first place. See the "Discussion" for further remarks.

Our system has 9 equilibria, with a single nontrivial equilibrium

$$\begin{pmatrix} F^* \\ B^* \\ I^* \\ P^* \\ X^* \end{pmatrix} = \begin{pmatrix} \dfrac{c}{H(1-bP^*)} \\ \dfrac{1}{H}\left(r(1-F^*) - \epsilon_F(1-f(X^*))\right) \\ \dfrac{1+s}{1+b+s} \\ \dfrac{1}{1+b+s} \\ \dfrac{h}{\beta(1+b+s)} \end{pmatrix} \tag{3}$$

In general, this equilibrium exists and is locally stable if the following three inequalities hold. These occur when the social concern is below the threshold needed to abate the emissions,

$$0 \leq \frac{h}{\beta(1+b+s)} < \breve{x} \tag{4}$$

when the fish reproduction rate exceeds the pollution mortality,

$$r > \epsilon_F(1-f(X^*)) \tag{5}$$

and when the returns from the harvest exceed the cost of fishing,

$$c < H(1-bP^*)\left(1 - \frac{\epsilon_F(1-f(X^*))}{r}\right) \tag{6}$$

We remark that the HES is sensitive to its initial conditions. Even if the inequalities (4)–(6) are satisfied, the nontrivial equilibrium may not occur if $F(t_0), B(t_0)$ and $X(t_0)$ are sufficiently small, for a given set of parameters, such that the fish catch collapses before the emissions are reduced. See Supplementary Material Sects. S2, S3 and S4 for more details.

Historical datasets and parameter selection

Most of the data available from Minamata are incomplete or sampled at irregular time intervals, making it ill-suited for a rigorous parameter recovery. Rather than estimate the parameters from the data, we make simulations that agree qualitatively with the information we have from Minamata.

Table 2.1 shows the available datasets, and Table 2.2 summarizes our choice of baseline parameter values and initial conditions for the simulation. Table 2.3 contains the simulation plots. Observe that the datasets are reported in absolute numbers, while the simulated variables are plotted as proportions.

To model the baseline emissions $n(t)$, we consider the emissions dataset closely. Prior to 1959, emissions levels increased as part of technical improvements at the factory, the details of which are exogenous to our model. After 1959, emissions were reduced due to public demand for abatement. In our model, we define the following baseline emissions function:

$$n(t): = \begin{cases} 0:100\,\% & \text{interpolated data} & t \leq 1959 \\ 100\,\% & \text{(continuing 1959 level)} & t > 1959 \end{cases} \tag{7}$$

where we take interpolated emissions data up to 1959, and assume that peak levels occur in 1959. The emissions continue at 100 % of 1959 levels if there is no abatement.

To define the relationship between social concern and abatement, we specify a function $f(X)$. Although emissions were abated after 1959, Tsuru reports that many Minamatans supported the company into the 1970s (Tsuru 1999), so it was a vocal minority of citizens that demanded abatement. We take f to be a nonlinear function

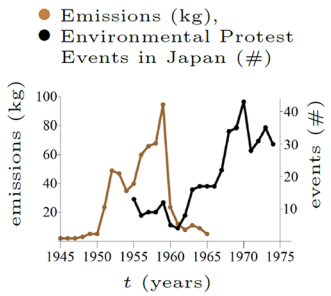
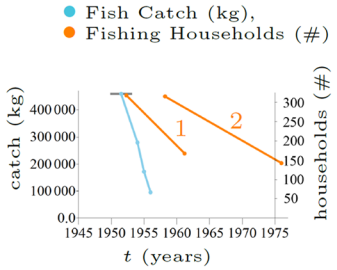
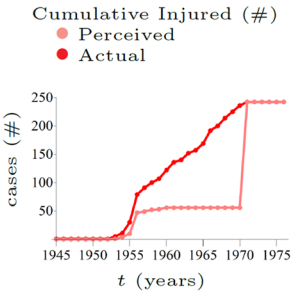
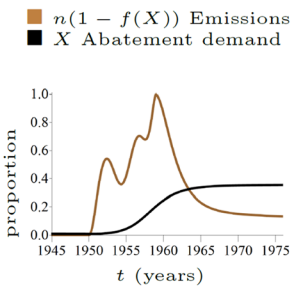
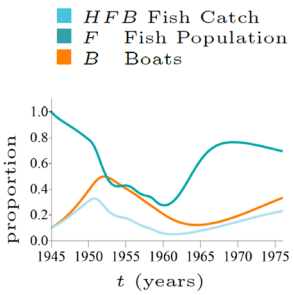
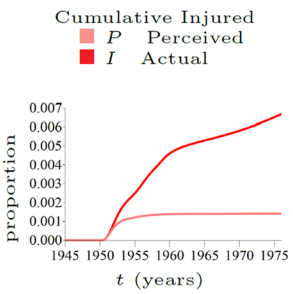
$$f(X) := \begin{cases} 0 & 0 \leq X \leq 0.2 \\ -250(X-0.2)^2(X-0.5) & 0.2 \leq X \leq 0.4 \\ 1 & 0.4 \leq X \leq 1 \end{cases} \tag{8}$$

where $\breve{x} = 0.4$ is the threshold of social concern above which emissions are fully abated.

There is data for the fish catch in Minamata Bay but not for the actual fish population, so the fish population that we generate in the simulation should be regarded as theoretical. The data for the number of fishing households in Minamata (Michiko 2003) and in the villages Tsukinoura, Detsuki, Yudo, and Modo (Tsurumi 2007) are used to make an indirect estimate for the number of boats in Minamata Bay.

As the fish catch data are incomplete, we also make use of qualitative historical information. In 1945, the fish stocks around Minamata were high, as they had not been

Table 2 We use historical datasets (Table 2.1) to select parameters for the HES model (Table 2.2) and generate simulations of Minamata (Table 2.3)

Emissions and Abatement Demand	Fishery	Injuries
2.1 Minamata Datasets		
	 (1) Minamata (2) Tsukinoura, Detsuki, Yudo, and Modo	
2.2 Selection of Baseline Parameter Values		
$X(1945) = 0.01$ $\kappa = 0.5/\text{year}$ $h = 1000$ $\beta = 4.0$	$F(1945) = 1.0$ $B(1945) = 0.1$ $H = 0.7/\text{year}$ $c = 0.35/\text{year}$ $r = 1.0/\text{year}$ $\epsilon_F = 0.7/\text{year}$	$I(1945) = 0$ $P(1945) = 0$ $\epsilon_I = 0.009$ $b = 100$ $s = 600$
2.3 Minamata Simulation		
		

Sources: emissions data (Harada 1975); protest events data (Almeida and Stearns 1998); fish catch data (Harada 1972); fishing households data (Michiko 2003; Tsurumi 2007); cumulative injured data (Harada 1975)

fished since before the Second World War (Keibo and Masato 2001). During the war, fishing people were reticent to fish the bay (Keibo and Masato 2001) because Chisso's factory was a target of intense American bombing (Ui 1992; George 2001). Accordingly, we initialize our model in 1945 with full fish stocks and a low number of boats. After the war, the resumption of fishing and the introduction of methylmercury pollution both put pressure on the fish stocks (Keibo and Masato 2001). While the data are insufficient to separate the effects of these pressures on the

Table 3 Equilibria types (and associated region colours)

	$1 - f(X^*) = 0$	$1 - f(X^*) > 0$
$F^*B^* = 0$	I) No Fish Catch, No Emissions	III) No Fish Catch, Emissions
$F^*B^* > 0$	II) Fish Catch, No Emissions	IV) Fish Catch, and Emissions

mortality of fish, we can assume that ϵ_F and H together outpace the fecundity r. Not shown in the fish catch data, but reported in George (2001) and Harada (1972, 1995) is that after fishing was reduced in 1957 and emissions were reduced in 1959, some species were observed to recover to levels deemed fit to fish openly again by 1964.

The data for the cumulative injured over the years 1945–1976 comes from Harada (1975). We take the "perceived injured" to be all cases discovered up to 1960, while the "actual injured" include additional cases discovered retrospectively in a 1971 epidemiological survey. Adjusting for the population size of Minamata over this period, we scale these datasets to estimate ϵ_I, b and s.

Public opinion poll data about social concern for pollution are not available from Minamata. Instead, we observe that social concern toward pollution at the nationwide-level in Japan underwent an increase during this period, judging from annual time series data on the number of anti-pollution social movement activities reported in Japanese newspapers, journals and magazines from 1955 to 1974 (Almeida and Stearns 1998). These activities include marches, rallies, demonstrations, and demands made by anti-pollution groups.

Results

Simulations and scenario analysis

Given that the social parameters s, b and h have been selected to agree qualitatively with the data, we concede that there is substantial uncertainty in the values selected. To provide context for the choices made in Table 2.2, we depict the steady states of the system in s–b parameter space. We numerically solve F, B and X for large time $t = 1000000$, and colour regions in s-b space according to the equilibrium type (I, II, III or IV) as defined in Table 3. See Supplementary Material Sects. S3 and S4 for more details.

The coloured s–b space is shown in Fig. 2c accompanied by representative time series plots in Fig. 2a, b, d, e. Each figure depicts solutions to the HES system that illustrate the dynamic behaviour of a particular equilibrium type (I, II, III, or IV). The time series in these plots are coloured according to the legend.

Stigma/misperception undermines protective social feedbacks

Figure 2e depicts the Minamata simulation that we generated in the previous section using $s = 600$, $b = 100$. We compare this simulation to the other scenarios in the context of changing stigma s and boycott pressure b.

Figure 2a shows a type-I equilibrium scenario for $s = 0, b = 500$, where both the pollution and the fishing cease. The increased boycott rate b reduces the number of people exposed to the pollution. However, it also lowers the demand for fish, which increases the cost-to-harvest ratio. For b sufficiently large, the fish catch is no longer viable.

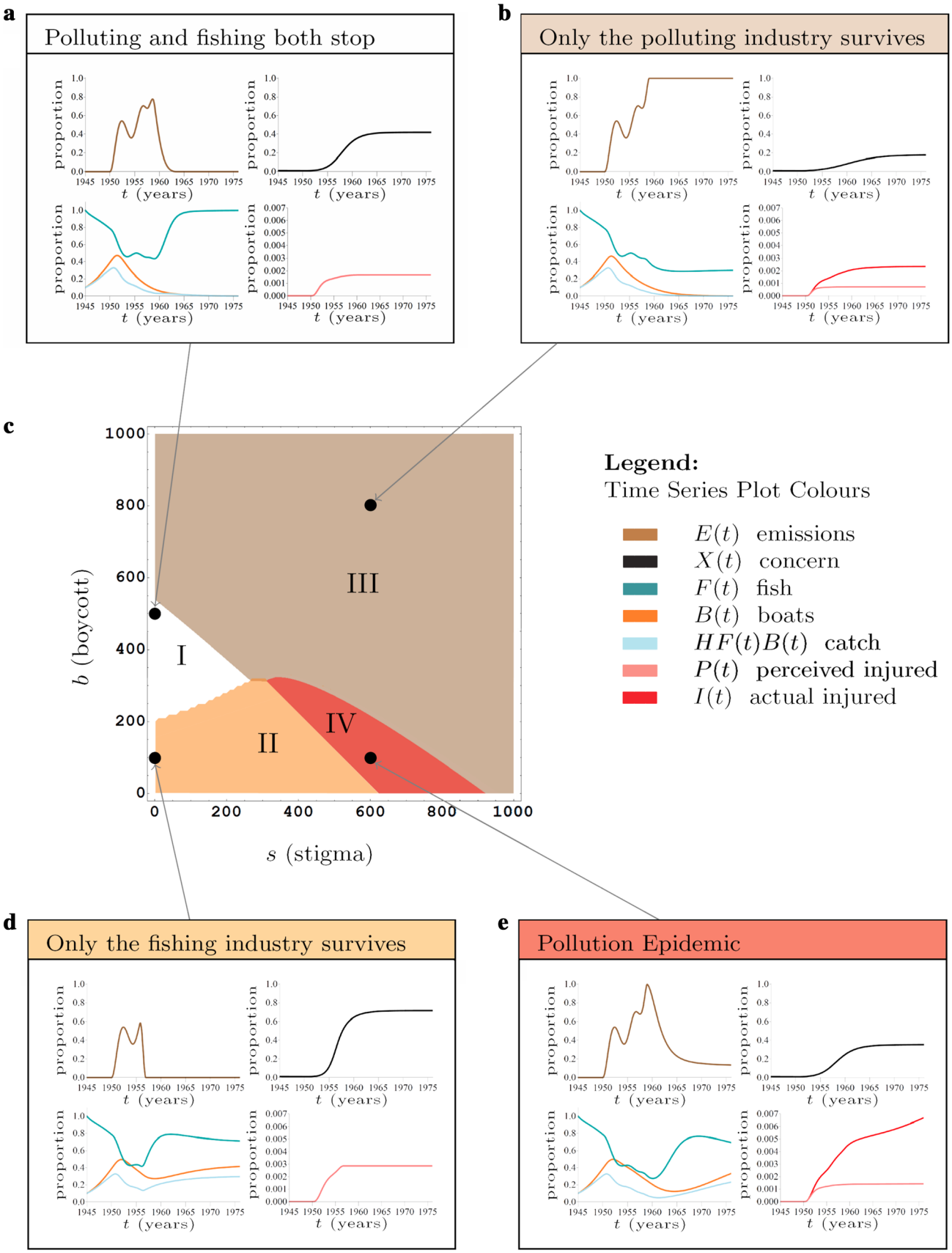
In Fig. 2d, a type-II equilibrium scenario occurs for $s = 0, b = 100$, where fishing survives and the emissions stop. We find that for s and b sufficiently low, enough people are exposed to the pollution that social concern rises and the emissions are eliminated. The boycott rate b is sufficiently low that the fish catch survives.

Figure 2b shows a type-III equilibrium scenario for $s = 600, b = 800$, in which the emissions survive and fishing stops. This scenario occurs when either the stigma or boycott pressure are sufficiently high. If the boycott rate b is increased, then people stop eating fish to protect themselves. If they boycott at such a rate that the fish catch collapses before enough people have been injured to cause full abatement, then the emissions survive. On the other hand, the stigma s has no preventative effect to truly protect people from injury. Increasing stigma causes an underestimation of the true injuries. If the stigma s is increased, or if s and b are both made sufficiently large, then the number of perceived injured is too low to stimulate emissions abatement. In addition, if s is high enough then the abatement is low, and the fish catch loses its viability due to pollution.

The Minamata scenario results when the boycott rate b is low enough to allow the fish catch to survive, and the stigma s is high enough that injuries are underestimated and a pollution epidemic (type-IV) equilibrium occurs.

Social trade-offs

To reinforce what the time series plots in Fig. 2a, b, d, e show us about the effect of varying the boycott and stigma on the cumulative injuries, we numerically solve I for large


a
Polluting and fishing both stop
b
Only the polluting industry survives
c
b (boycott)
s (stigma)
I
II
III
IV
Legend:
Time Series Plot Colours
E(t) emissions
X(t) concern
F(t) fish
B(t) boats
HF(t)B(t) catch
P(t) perceived injured
I(t) actual injured
d
Only the fishing industry survives
e
Pollution Epidemic
proportion
t (years)

◀**Fig. 2** Changing values for the social feedbacks s (stigma) and b (boycott) yield alternative qualitative outcomes. The parameter plane **c** shows the dynamical outcomes defined by the equilibrium types I, II, III, and IV that result for various values of s and b. All other parameters are held at their baseline values from Table 2.2. The accompanying time series plots are simulated at fixed values of s and b, and each is representative of a particular outcome: **a** type I: $s = 0, b = 500$, **b** type III: $s = 600, b = 800$, **d** type II: $s = 0, b = 100$, **e** type IV: $s = 600, b = 100$. The scenario in **e** corresponds to the Minamata simulation defined in Table 2.3

time and plot the distribution of injuries in s–b space. Figure 3 presents this cumulative injuries plot next to a coloured region plot of the various equilibrium types I, II, III and IV in s–b space.

We find that injuries are by far the highest in region type-IV. In general, injuries are higher with increasing stigma, and lower with increasing boycott pressure. With low stigma and high boycott pressure, there is a social trade-off: a high number of injuries are prevented, but this happens at the expense of the fishery, which collapses.

Observe that the simulations which result in the least number of cumulative injured come with collateral disadvantages: steady states I and III result in the collapse of the fishery, which is a collapse of the economic livelihood of a people. Steady state III allows the emissions to continue unabated, and by comparing Fig. 2a, b, d, e, one can see the effect this has on stunting the fish population. This would have major ecological ramifications for the wider food web, which are not modelled here.

Sensitivity to initial conditions

As discussed in "Methods", the qualitative behaviour of the system depends on the initial conditions for the fishery. If the initial catch $F(t_0)B(t_0)$ is sufficiently small, then the catch collapses ($FB \to 0$) faster than the injuries and the abatement grow ($P \to P^*$ and $f(X) \to 1$). Figure 4a shows our baseline-value parameter plane in s–b space, this time depicting the contour lines

$$C1 : h = 0.4\beta(1 + b + s)$$

$$C2 : c = H\left(1 - b\left(\frac{1}{1+b+s}\right)\right) \times \left(1 - \frac{\epsilon_F}{r}\left(1 - f\left(\frac{h}{\beta(1+b+s)}\right)\right)\right)$$

These contours are based on the equilibrium values in (3) and the inequalities (4)–(6). They enclose the region where

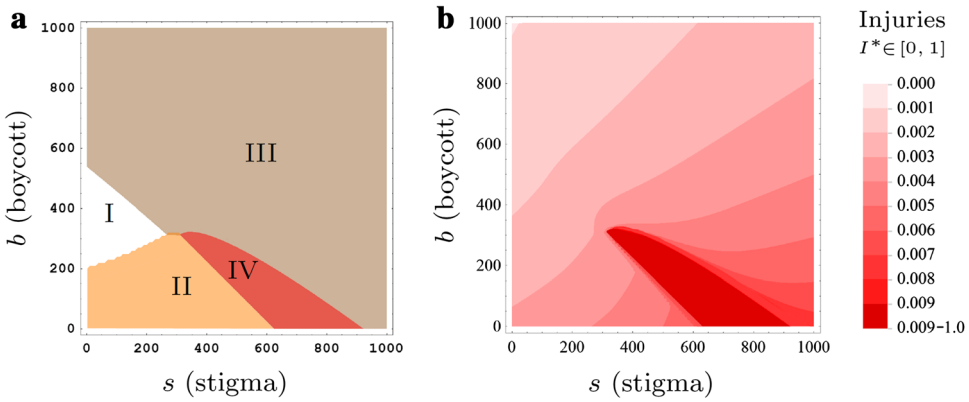


Fig. 3 Parameter planes showing outcomes for various values of s and b. The panels depict **a** the dynamical outcomes defined by the equilibrium types I, II, III, and IV, and **b** the health outcomes defined by gradations in the cumulative injured steady state, $I^* \in [0, 1]$

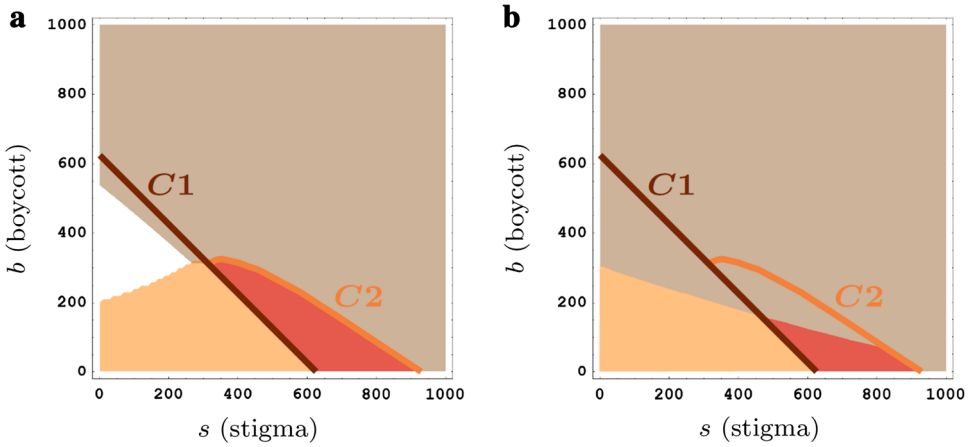


Fig. 4 Parameter planes showing outcomes for various values of s and b with different initial fish population densities: **a** $F(1945) = 1$, **b** $F(1945) = 0.01$. The contour lines $C1$ and $C2$ enclose the region where a pollution epidemic equilibrium is possible, outside of which it is not. We find that the occurrence of a pollution epidemic is sensitive to the size of the initial fish population and the rate of fish catch collapse

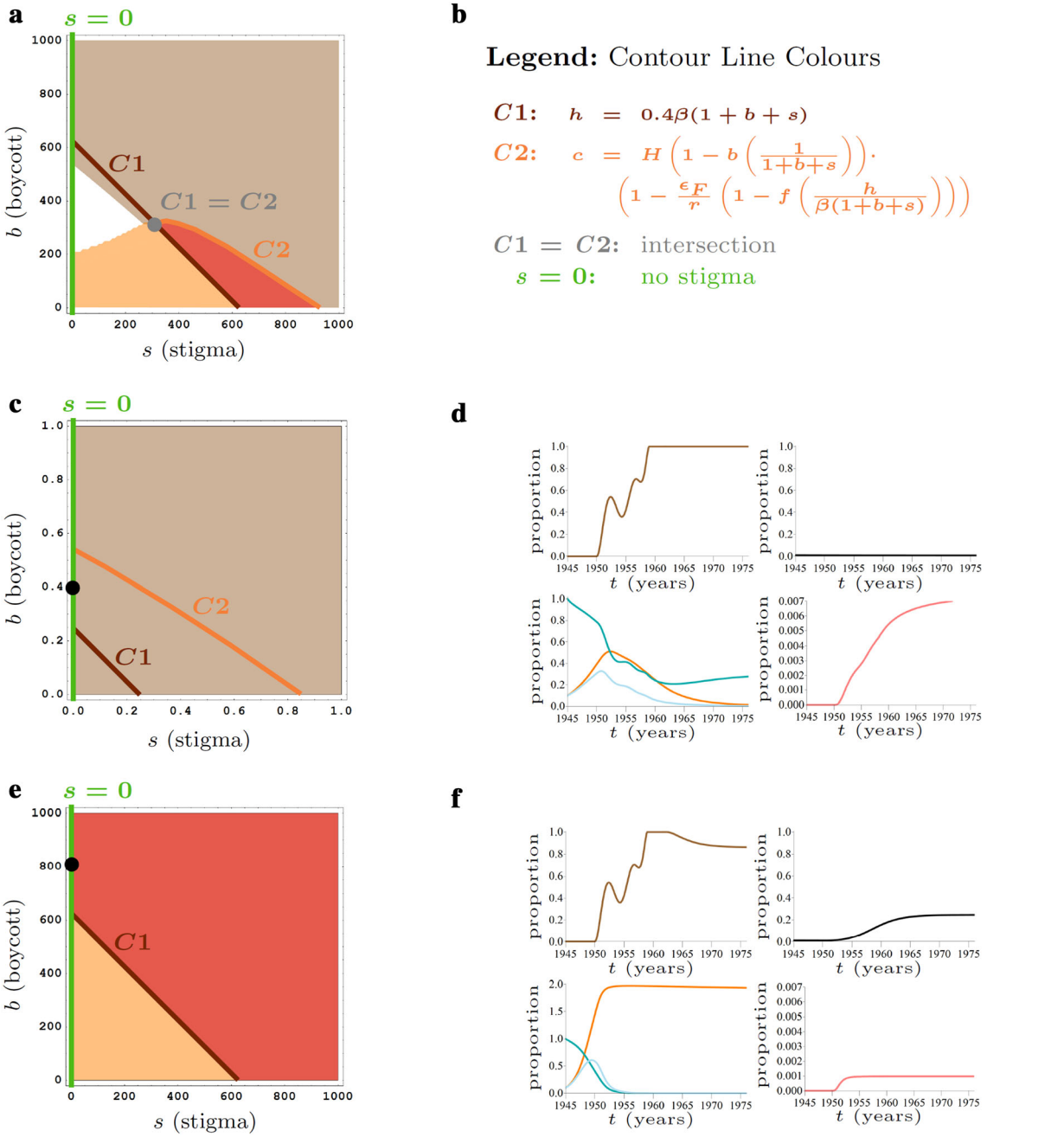


Fig. 5 We seek the conditions for which a pollution epidemic is possible with no stigma. Panel **a** depicts our baseline-value parameter plane in *s*–*b* space, while panel **b** provides a legend. The contour lines $C1$ and $C2$ enclose the region where a pollution epidemic is possible, outside of which it is not. The *green line* is the axis of no stigma, $s = 0$. Panel **c** shows the parameter plane that results when $h = 3$, while panel **d** provides example time series plots for $s = 0$, $b = 0.4$. Panel **e** gives the parameter plane that results when $c = 0.001$, and panel **f** provides example time series plots for $s = 0$, $b = 800$

a pollution epidemic can occur, and outside of which it is not possible. As Fig. 4b shows, whether or not the pollution epidemic occurs in the region between $C1$ and $C2$ depends on the initial conditions. Recognizing this sensitivity is important in further analysis.

A pollution epidemic is unlikely without stigma/misperception

It is very important to recognize how unlikely the conditions for a pollution epidemic (type-IV) equilibrium are

without social stigma/misperception. As the parameter plane in Fig. 2c shows us, an epidemic does not exist unless the stigma s is sufficiently large.

We ask: what parameters are required for us to have a pollution epidemic when $s = 0$, and how physically plausible are they compared to the baseline parameter values that we selected in Table 2.2? Figure 5a shows our baseline-value parameter plane in s–b space, with contours $C1$ and $C2$, and depicts the axis $s = 0$ in green. To find when a pollution epidemic becomes possible without stigma, we must vary our parameters so that the region enclosed by $C1$ and $C2$ crosses the axis $s = 0$.

First let us consider decreasing the health concern h. When $h = 3.2$ the intersection of the contours $C1 = C2$ meets the axis $s = 0$, so a pollution epidemic should be possible for $h < 3.2$. However, as Fig. 5c, d show, with $h = 3$, $s = 0$ and $b = 0.4$ a pollution epidemic does not occur in the region where we expect it to. In effect, the very small h and b make the social response to the pollution negligible. The fish catch collapses before the injuries grow large enough to trigger any abatement. Emissions continue at a high level but exposure stops, so a pollution epidemic is averted.

With low health concern, a pollution epidemic is possible for $s = 0$, provided that the social feedbacks to stop it are negligible and the fish catch survives. However, we find that it is unlikely for the fish catch to coexist with a high level of emissions. In the case of Minamata, the social feedbacks were not negligible; the fish boycott and demand for abatement were significant feedbacks. Unfortunately, the reduction in emissions there made it possible for the fish catch to coexist with the emissions.

Now let us consider changing the boat costs. When $c = 0.0012$, the contours $C1$ and $C2$ meet the axis $s = 0$, so a pollution epidemic should be possible for $c < 0.0012$. Choosing $c = 0.001$, Fig. 5e indicates that we can have a pollution epidemic without stigma! However, is this plausible? The accompanying time series plots in Fig. 5f show that this operating cost for the boats is unrealistically low. The boats grow to an unrealistically high level and push the fish population toward zero. For the short time-scale we are interested in, the potential for a pollution epidemic is averted by the decline of the fish catch to near-zero levels. If we solve numerically as $t \to \infty$, it turns out that the fish and boats exhibit damped oscillations, and the injuries grow.

So with reduced boat costs, we do find that a pollution epidemic is possible when $s = 0$, but that it relies on the unlikely and infeasible condition that a large number of boats survives by fishing a near-zero fish population over a large time scale.

Overall, the occurrence of a pollution epidemic relies highly on the presence of stigma or misperception.

Discussion

The coupled HES model presented in this paper allows us to investigate the dynamics of pollution exposure as intimately linked to the local environment, the economy, and social decisions. The analysis and simulations of this model show us at least three important things: (1) increasing stigma/misperception undermines the social feedbacks that protect people from pollution. (2) A pollution epidemic steady state occurs when emissions and fish ingestion continue indefinitely. The occurrence of this steady state is highly unlikely without stigma/misperception. (3) With multiple socially desirable outcomes, feedbacks can cause one outcome to undermine the other. Here, the scenarios that prevent the most injuries also cause the commercial fishery to collapse.

In the context of the Minamata pollution epidemic, our HES model represents the conditions that might have persisted for decades in Minamata if a second methylmercury-poisoning incident had not occurred in Niigata, Japan. Our model is focused on the localized relationships that are endogenous to Minamata and its people. It represents misperception effects in a novel and critical way. In the model, social decisions to boycott fish-eating and abate pollution are made from information about the variables, F, B, P and X. These variables can reach a false equilibrium that is blind to I, the actual injuries, which grow unperceived in the background. This situation of indefinitely growing injuries may seem unrealistic, but it is useful to consider for two reasons.

Firstly, it indicates that the endogenous and localized system we consider in our model can fail to be self-regulating. If citizen stakeholders have information that is inaccurate or different to that held by industry, then a decentralized, voluntary, and laissez-faire approach to resolving the problem is inadequate.

Secondly, it reflects that the injuries from chronic pollution exposures include subtle, delayed, and misinterpreted cases: the number of actual injuries may grow to vastly outnumber the number initially perceived, only to be discovered decades later. In the 1970s it was discovered that mercury-laden fish had affected people throughout the neighbouring Yatsushiro Sea region. As recently as 2005, medical researchers in this region continued to find people displaying subtle injuries from methylmercury exposure. They estimated that up to 100,000 people have been exposed since 1953—many more than have been officially certified as Minamata disease patients (Futatsuka et al. 2005). Our model is based only on data for the growth of the officially certified victims.

Our model shows that the prevention of injuries involves a trade-off: either the emissions or the fish catch must stop.

Emissions control is a cost to the polluting industry, and a fishing ban is a cost to the fishing industry. Given the community's dependence on both industries, it is easy to see why the temptation for many citizens in Minamata was to downplay the health damages and stigmatize the pollution victims, a situation which the government permitted and the industry took advantage of.

Decisive action to stop damages from pollution is often stalled by debate over the evidence, or obstructed altogether if data are withheld or not collected. Although the extent of the abuse in Minamata may seem like a relic of a different time and place, it is not.

Residents in Mossville, Louisiana, have been exposed to dioxins in emissions from some of the largest petrochemical complexes in the United States that neighbour their community. While the residents suffer from respiratory disorders and high cancer incidence, independent studies show high levels of dioxins in their blood that match the specific congeners emitted by the local petrochemical facilities (Subra 2007). However, state and federal authorities have denied this connection and ignored requests by the people of Mossville to conduct a conclusive epidemiological survey. Evidence is a prerequisite to action here, but the state and federal governments have refused to collect the necessary evidence. In April 2013, the case of the residents was admitted to the International Court of Law to sue the United States government for negligence (Livingston 2013).

The situation is similar in cancer-ridden communities in China (Tianjie 2009; Van Rooij 2010) where local governments have been slow to respond to health damages that have accumulated over decades. These struggles show that the pursuit of economic expediency at the expense of human welfare remains a pivotal obstacle in resolving pollution epidemics today.

When governments or industries misinform citizens or downplay the risks from pollution exposure, the long-term consequences include mounting damages that may become irreversible or more costly to repair later (Guidotti 2011). The livelihoods of people in afflicted communities are damaged, and they become excluded from the prosperity that the polluting industry is intended to bring. Our work highlights the need for citizens to have access to transparent information about the negative by-products of their local industries. To help combat the misunderstandings that may arise about a given pollution risk, and to prevent stigmatization of pollution victims, toxicological data must be gathered and shared.

There is a role for HES modelling approaches to help communicate risks to stakeholders in present-day pollution crises, and possibly to aid them in critical decision-making. A model such as ours may help stakeholders to understand the future or long-term consequences of their decisions, by allowing them to explore the outcomes of alternative scenarios. Simulations may be used in public information campaigns to discourage stigma during a pollution crisis. By generating a model that is tailored to the local pollution problem, simulations can show citizens the long-term extent of the injuries and damages that they could inflict on fellow community-members if they stigmatize the illness.

The insights provided by our model are not a substitute for epidemiological evidence, which is used in legal proceedings to establish the etiology of disputed pollution illnesses, and to prove or disprove the culpability of the polluter (George 2001; Smith and Smith 1975). In general, we believe it would be inappropriate to employ our model to estimate the number of real versus perceived pollution injuries in a polluted community. It may be more effective to try to measure whether members of the community resent the pollution victims, and whether they are aware of the latest scientific opinions about the pollution. Surprisingly, after the end of the first Minamata lawsuit in 1973, a poll by Japan's national broadcaster found that most Minamatans "were either quite indifferent or more sympathetic to Chisso than to the victims" (Tsuru 1999). Such information helps to indicate the level of misperception and stigma in the community, so that medical teams can plan how to reach out to stigmatized individuals who are afraid of seeking medical attention.

Although our model was developed to represent interactions that are highly localized and particular to Minamata, it has the potential to be modified in a variety of ways, and to be applied to a far wider range of pollution problems. In addition, our representation of the Minamata incident can itself be improved, by addressing certain simplifying assumptions that arise from the ordinary differential equations we use. We note that it is the task of the mathematical modeller to find a balance between detailed realism on one hand, and a parsimonious representation that is mathematically tractable, on the other.

We observe that our model aggregates individual variability within populations, which obscures the diversity of the fishery ecosystem, the variations in human fish-eating habits, and the varying degrees of severity of the human injuries. This may be improved, for example, by introducing compartmental equations for the different fish species, human fish-eating classes, and human symptom categories, and possibly also by hybridizing the system with a discrete agent-based model.

To represent more steps in the accumulation pathway of the pollutant, mass-balance equations can be introduced, or time delay terms can be added to the existing equations. Although methylmercury does rapidly accumulate and cause damage to organisms, a time lag becomes relevant if it is encountered chronically or at lower concentrations. A useful avenue of research would be to examine how a time delay might affect stigma towards the victims. This is

especially useful given that many pollution exposures are chronic, and that the time delay before symptoms appear hampers the ability of doctors to assign a causality to the illness.

Our model is spatially localized and homogeneous. It could be improved by considering the role of diffusion and fish migration in spreading the pollution beyond Minamata Bay. Social concern is spatial-dependent, as the demand for abatement is stronger among population groups that live farther from the polluting industry. For example, citizens in Niigata lived far enough down river from the polluting factory that they were more inclined to mobilize against it, and less inclined to stigmatize the victims (Almeida and Stearns 1998; Smith and Smith 1975).

Our model represents social learning as an endogenous process, isolated from external events and opinions. The Niigata pollution epidemic was an event that changed the social attitudes of people across Japan to oppose the polluting industry in Minamata. Concerned citizens in Minamata depended on external allies and protests to galvanize public attention for their cause. By contrast, our model lacks a mechanism for the system to move away from a pollution epidemic equilibrium. To correct this, we can introduce an impulse term $+\eta(t)(I - P)$ in the equation for the perceived injuries, $P'(t)$, which will make P move closer to I given an impulse $\eta(t) > 0$.

We close by acknowledging the potential for HES modelling approaches to further elucidate the complex interactions among environmental, economic, and social processes. We also believe that the recent pollution problems in both Mossville, Louisiana, and China's "cancer villages" demonstrate the relevance of stigma and misperception to the study of contemporary pollution problems. Coupled HES models that include misperception effects as part of their human–environment interactions are highly topical and should be further studied.

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