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# Epidemics in markets with trade friction and imperfect transactions

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## AUTHOR-HIGHLIGHTS

- Trade can support the transmission of infectious diseases, but it is not clear how.
- Market dynamics are constrained by trade friction because exchanges have constraints and are costly.
- We develop a model of market dynamics with trade friction and spread of disease.
- Friction can be a stronger determinant of epidemics than other forms of behaviour.
- Lower trade friction requires greater immediacy in implementing epidemic control.

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## ABSTRACT

Market trade-routes can support infectious-disease transmission, impacting biological populations and even disrupting trade that conduces the disease. Epidemiological models increasingly account for reductions in infectious contact, such as risk-aversion behaviour in response to pathogen outbreaks. However, responses in market dynamics clearly differ from simple risk aversion, as are driven by other motivation and conditioned by "friction" constraints (a term we borrow from labour economics). Consequently, the propagation of epidemics in markets of, for example livestock, is frictional due to time and cost limitations in the production and exchange of potentially infectious goods. Here we develop a coupled economic-epidemiological model where transient and long-term market dynamics are determined by trade friction and agent adaptation, and can influence disease transmission. The market model is parameterised from datasets on French cattle and pig exchange networks. We show that, when trade is the dominant route of transmission, market friction can be a significantly stronger determinant of epidemics than risk-aversion behaviour. In particular, there is a critical level of friction above which epidemics do not occur, which suggests some epidemics may not be sustained in highly frictional markets. In addition, friction may allow for greater delay in removal of infected agents that still mitigates the epidemic and its impacts. We suggest that policy for minimising contagion in markets could be adjusted to the level of market friction, by adjusting the urgency of intervention or by increasing friction through incentivisation of larger-volume less-frequent transactions that would have limited effect on overall trade flow. Our results are robust to model specificities and can hold in the presence of non-trade disease-transmission routes.



## 1. Introduction

While it is widely accepted that trade can drive disease epidemics and other biological invasions, the interaction of these processes with the inherent dynamics of markets remains unclear. Economic markets can propagate diseases among market agents (e.g. farms) through the exchange of contaminated products (e.g. animals). Conversely, market dynamics are influenced by complex adaptive behaviour of trade agents in response to regulation and individual awareness of epidemics. Markets that contribute to infectious disease epidemics include livestock trade of cattle (Rautureau et al., 2011), swine (Lentz et al., 2011), and sheep (Kiss et al., 2006); prostitution (Rocha et al., 2011); and airline

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transportation (Colizza et al., 2006). Other types of epidemics occur through exchange of information on the Internet (Lloyd and May, 2001) and exchange of debt in financial markets (May and Arinaminpathy, 2010; Haldane and May, 2011).

The likely possibility that there may be an interaction or feedback loop between epidemic dynamics and host behaviour has generally not been considered in studies for identifying effective strategies for the control of infectious diseases. Recent modelling studies, however, have explored the epidemiological impact of one particular response that can cause such an interaction, namely, adaptive risk-aversion (RA) behaviour (Funk et al., 2009, 2010; Durham and Casman, 2012; Nicolaides et al., 2013). RA behaviour is a form of disease prevention where asymptomatic hosts reduce exposure to infection by reducing their contact rate (e.g. by staying home) and/or their probability of infection per contact (e.g. by wearing protective masks); it implies that hosts have some information about a given disease outbreak and act on their own initiative rather than relying on community-wide measures by regulatory bodies. If this behaviour is determined by the ongoing perception of a variable risk, then it is said to be 'adaptive' RA. In the literature, RA has been expressed as a simple function of disease prevalence or outbreak awareness (Funk et al., 2010), or evaluated via complex optimisation of a host's economic weighing between the benefits of interacting with other hosts and the cost of infection that may be acquired through such contacts (Fenichel et al., 2011; Morin et al., 2013). Naturally, epidemiological models that neglect RA behaviour tend to overestimate the probability of occurrence and severity (e.g. infectious peak and cumulative cases) of epidemics (Funk et al., 2009, 2010; Fenichel et al., 2011; Morin et al., 2013). To the best of our knowledge, epidemiological modelling studies have focused on adaptive human behaviour that is altered solely in response to awareness of outbreaks.

In this paper, we investigate the epidemiological effects and implications for disease control of more general human adaptive behaviour, which may be difficult to anticipate. We focus on markets of goods, where the dynamics of potentially infectious contacts are driven, primarily, by economic decisions very different from those underlying disease RA. Specifically, we aim at modelling the influence of market dynamics on the dynamics of infectious-disease epidemics, and in turn, the influence of epidemics on market dynamics. Indeed, when an epidemic shock occurs in a market, the subsequent actions and behaviour may either help us to restore or further disturb the balance between supply and demand. Sanatory regulation and RA aimed at reducing infectious contacts can diminish supply and demand. Conversely, the responses exhibited by market agents can have either positive or negative impacts on disease dynamics. For example, agents that try to establish alternative but potentially infectious trade relationships could outweigh the effect of regulation and RA efforts, i.e. the effort of individual agents to adjust their own supply and demand to changing price could worsen disease outbreaks. Furthermore, the establishment of trade relationships, which underpin the epidemiological contacts, is conditioned by physical impediments such as the minimum time and effort involved. These resources are limited by (i) producing profitable goods (e.g. reproduction and growth of livestock), (ii) searching business partners and cutting deals (e.g. a buyer needs to find a seller with whom to trade a given number of goods at a given market price), and (iii) delivering goods (e.g. organising transport from a buying to a selling holding). In labour economics, such interaction constraints shaping relationships between work sellers and work buyers are known as 'friction' (see the model of Diamond, Mortensen and Pissarides (Pissarides, 1985; Mortensen and Pissarides, 1994; Pissarides, 2011)). We transpose this concept to exchange-markets that can conduce infectious diseases. Therefore, by limiting the development of potentially infectious trade contacts, friction may have a suppressive effect on epidemics. Phenomena such as friction and adjustment in supply and demand illustrate that human behaviour in response to disease epidemics that are supported by trade does not simplify to regulation and RA.

To the best of our knowledge, existing mathematical models of market dynamics do not seem to represent explicitly the variety of transient non-equilibrium dynamics that occur when a market is disturbed and until it eventually reaches a steady-state equilibrium (see ESM Section A); therefore, they may not fully incorporate processes and parameters that establish a time scale for market steady-state equilibration, which is expected to vary widely among markets for rapidly changing external conditions such as those induced by epidemic outbreaks. In order to represent the joint-dynamics of markets and epidemics at appropriate and mutually consistent time scales (see Section 2.1), we have developed a novel economic-market model, the *frictional-trade market (FTM) model* (see Section 2.2), where transient and long-term dynamics are determined by the level of trade friction and agents' decisions to supply or demand goods. Subsequently, we integrate market and epidemiological processes in a *market-epidemiological (ME)* modelling framework where trade influences disease transmission and disease control actions affect trade (see Section 2.3).

We first study the behaviour of the FTM model in the absence of epidemics (see Section 3.1). Then, we investigate how market dynamics affect epidemic development, and, conversely, how epidemics disrupt short- and long-term market dynamics (see Section 3.2). We consider two forms of response to disease outbreaks taken from the literature: the removal (inactivation) of market agents found to be infected by regulators and their later re-introduction or replacement, and an adaptive RA behaviour of market agents. Therefore, we highlight differences in concept and impact on epidemic development between market dynamics and RA; market dynamics are influenced by centrally regulated actions and by collective behaviour that drives changes to supply and demand in response to changing conditions, while RA behaviour is determined solely by individual decision-making. Finally, we extend our study beyond an isolated (e.g. national) market, by contrasting scenarios where infectious diseases are propagated through trade pathways with differing degree of openness to international trade and non-trade disease-transmission pathways. We expect our central results to apply to a range of different types of markets, and illustrate applications to cattle and swine livestock markets in France.

## 2. Market-epidemiological modelling framework

### 2.1. Overview

We develop a novel theoretical framework for the propagation of infectious diseases in economic markets where the exchanged goods can transmit an infectious organism between market agents (Fig. 1A). In order to represent this process, we link a model of an economic market system and a model of an epidemiological system. Each model dynamics can exist per se, i.e. epidemics can occur in host populations unaffected by markets, and markets often operate without disease outbreaks through trade routes. However, by building a system that links the dynamics of these subsystems we can study their interdependencies. As the epidemiological model we use is a simple adaptation of a standard compartmental epidemiological model, it is introduced later with brief explanation. The dynamic economic-market model, however, is novel, and is derived in detail. A key property of this model is its coefficient of friction, which characterises a market's inherent

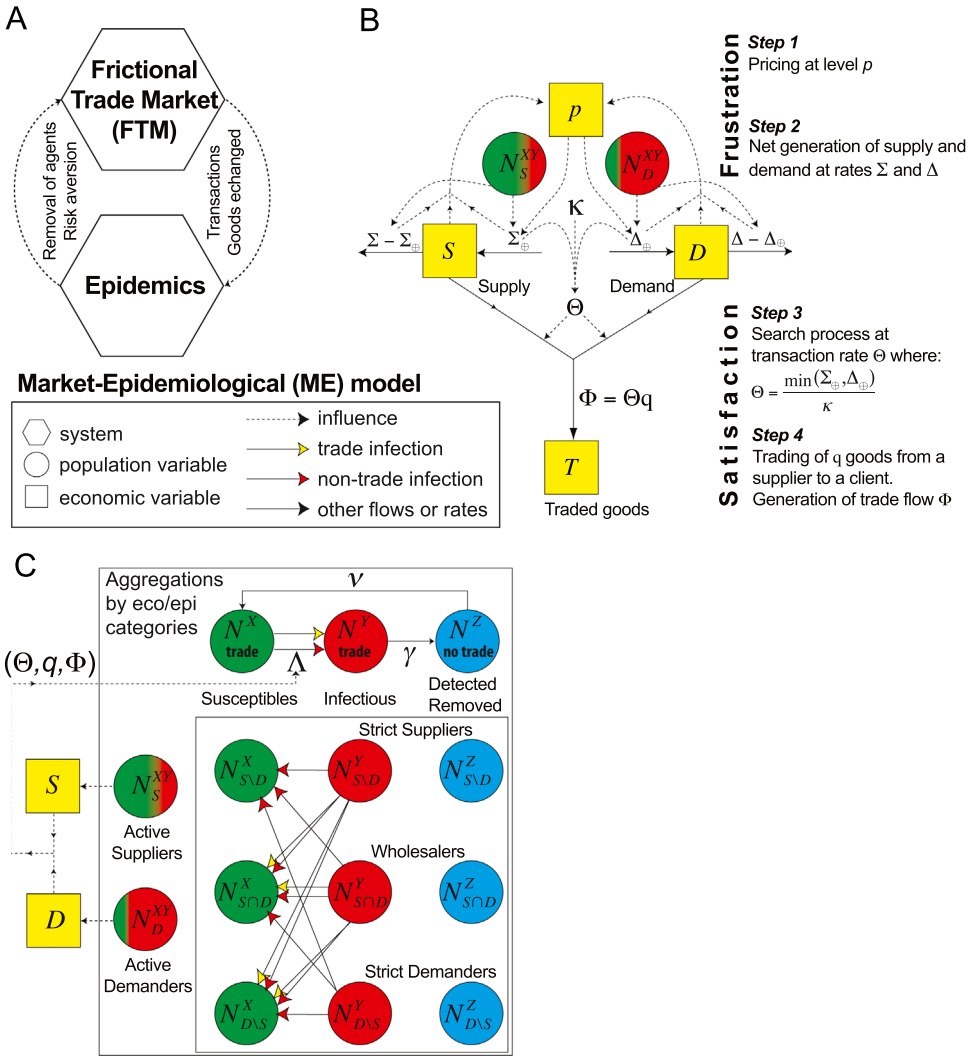


**Fig. 1.** Joint market-epidemiological modelling framework. (A) General structure and key links between the market and epidemiological subsystems. (B) Components of the frictional-trade market (FTM) model. (C) Components of the market-epidemiological (ME) model. Yellow (red) arrows represent direction of disease transmission through trade (non-trade) routes. The market agents host the disease-causing pathogen. $N_A$ represents the number of agents of arbitrary type $A$ that can vary over time. $N_{S\setminus D}$, $N_{S\cap D}$ and $N_{D\setminus S}$ denote the numbers of *strict suppliers*, *wholesalers* (supplier and demander) and *strict demanders* respectively. From an epidemiological point-of-view, each agent can be in the *Susceptible* ($X$), *Infectious* ($Y$), or *Removed* ($Z$) state. Susceptible agents become infectious at rate $\Lambda(t)$; infectious agents are removed at rate $\gamma$; and removed agents re-enter the market (recover) at rate $\nu$. Here, 'removal' means that an infectious agent is detected and removed from the market by a regulator and becomes *inactive*. Each agent, whether a strict supplier, wholesaler, or strict demander, can be in each of the epidemiological states. For example, $N_S^X$ and $N_S^Y$ denote the number of susceptible and infectious suppliers, respectively. Therefore, there are $N_S^{XY} = N_{S\cap D}^{XY} + N_{S\setminus D}^{XY}$ active suppliers. Accumulated supply and demanded stocks $S$ and $D$ quantify the *willingness to trade* or *frustration* of suppliers and demanders respectively. Supply stock consists of goods available for sale, while demanded stock represents potential goods that buyers want to purchase and are virtual. The *trade flow* $\Phi$ out of the supply and demanded stocks aggregates all exchanges between supply and demand agents that agree to trade some of their stock at price $p$. Transactions occur at *transaction rate* $\Theta$ and in each transaction an *average transaction stock* $q$ is exchanged. At *the reference market* steady-state equilibrium, $q$ is equal to $\kappa$, *the coefficient of friction*. If trade flow were kept at the corresponding equilibrium level $\Phi^*$, then the transaction of goods would occur at a lesser frequency $\Theta^* = \Phi^*/\kappa$ if the number of goods exchanged per transaction $q = \kappa$ would increase (see 'Results'). (For interpretation of the references to colour in this figure caption, the reader is referred to the web version of this paper.)

dynamics and response to disturbance caused, for example, by disease outbreaks (see the 'Results' section).

### 2.2. A frictional-trade market (FTM) model

Here we develop a FTM model for the dynamics of markets (without epidemics) where goods of a single type are exchanged for money in *transactions* between suppliers (sellers) and demanders (buyers), and all transactions at given time are based on a single price per good. The model tracks the dynamics of *extensive* state variables (i.e. with a 'size' or a 'scale') such as stocks, number of agents, and trade flow, and *intensive* state variables (i.e. global indices) such as price of goods. Usually, price, trade flow, and number of agents are observable market quantities, while overall stocks are not observable.

Our model market is defined, at each time $t$, by an overall *price* per good $p(t)$, and overall accumulated *supply stock* $S(t)$ and *demanded stock* $D(t)$ (see Table 1 for an explanation of relationship with common economic terminology and concepts). Accumulated supply stock consists of goods available for sale but not yet sold, while demanded stock represents potential goods that buyers want to purchase but have not yet been purchased. These accumulated stocks quantify the *willingness to trade* of suppliers and demanders. These accumulated stocks quantify the suppliers' and demanders' *willingness to trade*. The supply stock is part of the *inventory stock* (e.g. total animals within farms), which also includes goods not for sale owned by sellers. The demanded stock cannot exceed the *physical capacity* of demanders (e.g. maximum number of animals), which is similar to a carrying capacity in ecology. Our FTM model integrates many complex mechanisms

**Table 1**
Relationship between terminology in the frictional-trade market (FTM) model and in economics.

| Terminology adopted (notation/equation) | Meaning | Related economic concept | References |
|---|---|---|---|
| (Accumulated) supply stock ($S$) ((1) and (8)) | Amount of goods available for sale (e.g. in a farm, may be a fraction of the total number of animals) | A part of the inventory, capital, or stock of natural resources (e.g. in a farm, may be the total number of animals) | Clark (1976), Barro and Sala-i Martin (2003), Khan and Thomas (2003), Caputo (2005) |
| Supply flow, or rate of generation of supply stock ($\Sigma_\oplus$, (3)) | Increase in amount of goods available for sale per unit time | (in excess or equal to) The amount of goods sold per unit time, known as 'supply' | Fair and Jaffee (1972), Hahn (1982), Mas-Colell et al. (1995), McCauley (2009) |
| (Accumulated) demanded stock ($D$, (1) and (8)) | Number of goods wanted for purchase but not yet purchased, including accumulated demand that has so far not been satisfied (e.g. farmers who have been losing livestock during an undetected disease outbreak have accumulated the will to purchase $D$ replacement animals; depending on current conditions of supply of stock, there may be multiple subsequent transactions that gradually satisfy this demand, but which can spread over an economically significant time period | · | · |
| Demand flow, or rate of generation of demand for purchase of stock ($\Delta_\oplus$, (3)) | Increase in potential amount of goods to be purchased per unit time | In excess or equal to the amount of goods purchased per unit time (rate of depletion of stocks not including losses), known as 'demand', where 'demand' is often assumed to equal 'supply' | Fair and Jaffee (1972), Hahn (1982), Mas-Colell et al. (1995), McCauley (2009) |
| Reference transaction rate ($\min\{\Sigma_\oplus;\Delta_\oplus\}$, (6)) | Reference number of transactions per unit time when as few as one good can be exchanged per transaction | · | · |
| Trade friction ($\kappa \geq 0$, (6) and (9)) | Physical impediments to the trade of goods: constraints in production, effort and delay in the search for business partners and negotiation of deals, and logistical constraints in the delivery of goods. In steady-state equilibrium $\kappa$ is the average number of goods exchanged per transaction, implying that $\kappa \geq 1$ for most systems where it is not possible to trade less than one unit of goods (6) | Transaction costs; friction in labour markets, e.g. intensity of search and matching | Klaes (2008), Pissarides (1985, 2011), Mortensen and Pissarides (1994), Williamson and Masten (1999), Klos and Nooteboom (2001), Tesfatsion (2001) |
| Transaction rate ($\Theta = \frac{\min\{\Sigma_\oplus;\Delta_\oplus\}}{\kappa}$, (6)) | Number of stock exchanges in the market (deliveries from sellers to buyers) per unit time | 'Matching function' in labour market economics: number of successful matches per unit time between job vacancies and unemployed workers | Pissarides (1985, 2011), Mortensen and Pissarides (1994) |
| Average transaction stock ($q$, (5)) | Average number of goods exchanged per transaction which we currently set to the maximal match between current supply stock and demanded stock in each transaction | · | · |
| Trade flow ($\Phi = \Theta\, q$, (4)) | Market-level amount of goods actually exchanged per unit time | Related to actual quantity exchanged at a given point in time, but not identical when the period during which exchanges occurred was not specified (see also ESM Section A.3.3) | Fair and Jaffee (1972) |
| Occurrences of 'non-equilibrium' in the FTM model | *Overall rates of change are not zero*, i.e. non-negligible rate of change of market state variables: supply stock $\left(\frac{dS}{dt}\right)$, demanded stock $\left(\frac{dD}{dt}\right)$, or price $\left(\frac{dp}{dt}\right)$ in (8) | The absence of steady-state or inter-temporal equilibrium | Clark (1976), Pindyck (1982), Barro and Sala-i Martin (2003), Khan and Thomas (2003), Caputo (2005), Nowak (2006), McCauley (2009) |
| | The two possibilities below may associate with the occurrence of a non-steady-state: | | |
| | *Imperfect transactions*, i.e. the supply and demanded stocks of trading partners do not necessarily match, causing residual stocks that subsequently accumulate ($q$ (5) is the smallest of $S/N_S$ and $D/N_D$) | Disequilibrium | Fair and Jaffee (1972), Quandt (1988) |
| | *Imbalance between net generation rates of supply and demanded stocks*, i.e. net rates of generation of supply stock and demanded stock differ ($\Sigma \neq \Delta$, (2)). The level of imbalance depends on how much the market is competitive, as determined by parameter $\mu$ in our model which sets a timescale for price dynamics ((7) and (8)), and may or not preclude an eventual steady state | Imperfect competition, potentially leading to unbalanced long-run profits between selling and buying firms. Potential causes of such market failures include epidemic outbreak, monopolistic behaviour, externalities, free-riding, information asymmetries, transaction costs, and price rigidities | Mas-Colell et al. (1995), Williamson and Masten (1999), Klos and Nooteboom (2001), Klein et al. (2007), Klaes (2008), Silvestre (2008) |

We focus on notions that may help readers with economics background. A dot ( · ) means the corresponding concept does not appear to be defined in economics (to the best of our knowledge).

and dynamical feedbacks that have seemingly not been explored concurrently in the literature (see ESM Section A). To keep our model parsimonious, we hence model market dynamics at a whole-market level, where agent-level stocks and transactions are approached by average values per agent. Inspired by well-known population dynamics models (e.g. May, 1977; Durrett and Levin, 1994), our whole-market-scale model is a population-level description of agents and stocks with mass-action interactions (transactions). At this simplified level of description, we define the market model through temporal change in overall stocks. Each *stock* ($S$ and $D$) is created at a specific *net generation rate* and depleted through a *trade flow*, represented by the rate equation:

$$\frac{d\,[\text{stock}]}{dt} = [\text{net generation rate}] - [\text{trade flow}]. \tag{1}$$

The *net generation rate* of supply stock $\Sigma$ or demanded stock $\Delta$ is composed of (1) a *generation rate*, $\Sigma_{\oplus}(p, N_S)$ or $\Delta_{\oplus}(p, N_D)$, respectively, that depends on current price $p$ and numbers of supply agents $N_S$ and demand agents $N_D$, (2) a net *loss rate* $L$, e.g. spoilage of supply goods and loss of demanders interest (positive loss) or multiplication of goods such as reproduction of livestock (negative loss), and (3) an *external flow* of stock $E$, e.g., import or export of raw materials or goods:

$$[\text{net generation rate}] = [\text{generation rate}] - [\text{loss rate}] + [\text{external flow}],$$

$$\Sigma = \Sigma_{\oplus}(p, N_S) - L_S + E_S \quad \text{(net supply flow)},$$
$$\Delta = \Delta_{\oplus}(p, N_D) - L_D + E_D \quad \text{(net demanded flow)}. \tag{2}$$

Following the economic literature (see ESM Section A for details), generation rates are defined as

$$\Sigma_{\oplus}(p, N_S) = N_S \sigma_0 p^{\varepsilon_S},$$
$$\Delta_{\oplus}(p, N_D) = N_D \delta_0 p^{-\varepsilon_D}, \tag{3}$$

where $\sigma_0$ and $\delta_0$ are respectively the reference *per-agent* generation rates in supply and demanded stocks at the reference price $p = p_0 = 1$, and $\varepsilon_S \geq 0$ and $\varepsilon_D \geq 0$ are the price elasticities of supply and demand respectively. $\varepsilon_S$ and $\varepsilon_D$ are assumed constant and are defined, respectively, as the relative changes in the generation rates of supply and demanded stocks in response to the relative change in price. Notice that $\Sigma_{\oplus}(p, N_S)$ increases while $\Delta_{\oplus}(p, N_D)$ decreases with increasing price. Furthermore, we assume that the loss rates are directly proportional to stocks, i.e. $L_S = r_S S(t)$ and $L_D = r_D D(t)$ with $r_S$ and $r_D$ being constants, and that external flows $(E_S, E_D)$ are constant.

The *trade flow* $\Phi(t)$ out of the supply and demanded stocks aggregates all exchanges between supply and demand agents that agree to trade some of their stock (i.e. exchange of supply stock for demander's money). Transactions occur at *transaction rate* $\Theta(t)$ and in each transaction an *average transaction stock* $q(t)$ is exchanged; therefore, the trade flow is

$$[\text{trade flow}] = [\text{transaction rate}]\ [\text{average transaction stock}],$$
$$\Phi(t) = \Theta(t) q(t). \tag{4}$$

First we define $q$. The average per-agent supply and demanded stocks are $S(t)/N_S$ and $D(t)/N_D$ respectively. Here we take the number of market agents to be constant in time, but later, when considering epidemics in markets, we allow for removal of infectious agents and their subsequent re-introduction after sanitation measures. In this model we assume that, once a pair of supply and demand agents has been identified and agreed to transact, they exchange the maximum possible per-agent stock (a best-possible match): $S(t)/N_S$ if there is excess accumulated demand ($D(t)/N_D \geq S(t)/N_S$), and $D(t)/N_D$ if there is excess accumulated supply ($S(t)/N_S \geq D(t)/N_D$). Hence, the average transaction stock, conditional on best-possible matching, is

$$q(t) = \min\{S(t)/N_S; D(t)/N_D\}. \tag{5}$$

The transaction rate $\Theta(t)$ in (4) is determined by a driving factor, the *reference transaction rate*, and a *limiting factor* that compounds multiple constraints, such as the time-consuming *production* of goods, the *search* for trading partners, and the logistics of stock *delivery*. Hence

$$[\text{transaction rate}] = \frac{[\text{reference transaction rate}]}{[\text{limiting factor}]},$$
$$\Theta(t) = \frac{\min\{\Sigma_{\oplus}(p, N_S); \Delta_{\oplus}(p, N_D)\}}{\kappa}. \tag{6}$$

We assume that the *reference transaction rate* is determined predominantly by present decisions to increase stocks and, thus, by the generation rates in (2) and (3); more specifically, it is determined by the current maximum possible rate of exchange of indivisible goods between the two sides of the market, $\min\{\Sigma_{\oplus}(p, N_S), \Delta_{\oplus}(p, N_D)\}$. Here, the reference transaction rate is not determined by the net generation rates in (2) because these include loss rates $L$ and external flows $E$ that are likely to depend on the overall stocks $S$ and $D$, which usually are imperfectly known or unquantifiable; however, the current price $p(t)$, which determines the production rates, is known to market agents. In addition, we represent the limiting factor of the transaction rate by a dimensionless coefficient $\kappa$ that, through physical analogy, we call coefficient of *friction* or *inverse fluidity* of the market (see more below).

To finalise the specification of our market model, we need to specify price dynamics. The *net willingness to trade* or *frustration* of agents at a given time is the excess in accumulated demanded stock, $D(t) - S(t)$. While different relationships between price and other state variables can be specified, we assume for definiteness, and in agreement with literature (see ESM Section A), that changes in log price are directly related, via a dimensionless coefficient $\mu$, to changes in net willingness to trade:

$$\frac{d[\text{price}]}{dt} = \mu \frac{d[\text{net willingness to trade}]}{dt}\,[\text{price}],$$
$$\frac{dp}{dt} = \mu \frac{d(D-S)}{dt}\, p, \tag{7}$$

which can be solved explicitly as an exponential relationship (see (8)).

With these specific assumptions, and in the absence of disturbances such as epidemics, our FTM dynamics are defined by the equations (represented diagrammatically in Fig. 1B)

$$\frac{dS}{dt} = \overbrace{\underbrace{N_S \sigma_0 p^{\varepsilon_S}}_{\Sigma_{\oplus}} - r_S S + E_S}^{\Sigma} - \overbrace{\underbrace{\frac{\min\{\Sigma_{\oplus}; \Delta_{\oplus}\}}{\kappa}}_{\Theta} \underbrace{\min\left\{\frac{S}{N_S}; \frac{D}{N_D}\right\}}_{q}}^{\Phi},$$
$$\frac{dD}{dt} = \overbrace{\underbrace{N_D \delta_0 p^{-\varepsilon_D}}_{\Delta_{\oplus}} - r_D D + E_D}^{\Delta} - \overbrace{\Theta q}^{\Phi},$$
$$p(t) = p(0) \exp\{\mu[D(t) - S(t) - (D(0) - S(0))]\}. \tag{8}$$

The special case where $r_S = r_D = 0$ and $E_S = E_D = 0$ is referred to as the *reference market*. Hereafter, we use a star in superscript to denote market variables at steady-state equilibrium in the reference market in the absence of disturbances (e.g. $\Phi^*$ is the reference trade flow at steady-state equilibrium).

At a market (macroscopic) level, our model has two determinants of trade flow (see (4)–(6)): the coefficient of friction $\kappa$, and the average transaction stock $q$.

We explain first the interpretation and significance of friction. An increase in the coefficient of friction $\kappa$ reduces the transaction rate (see (6)) as in physical systems, friction is a macroscopic manifestation of microscopic resistance to movement. The microscopic-level constraints that underlie friction in trade (transactions) are a result of 'viscosity' in *production* of tradable good,

*search* of partners, and *delivery* of stock; therefore, we may consider that $\kappa$ has at least three components:

$$\kappa = \kappa_{\text{production}} + \kappa_{\text{search}} + \kappa_{\text{delivery}}. \quad (9)$$

As an example, let us consider livestock markets and suppose $\kappa_{\text{delivery}}$ is the dominant component and 1000 animals are produced, consumed and traded per farmer per year. If a minimum of $\kappa = 10$ animals are delivered in a single shipment, the transaction rate is at most $\Theta = 1000/10 = 100$ per farmer per year. If, however, the nature of the animals (for instance pigs are smaller than cattle) and transportation mean it is more viable to ship a minimum of 100 animals, then the transaction rate would be at most $\Theta = 1000/100 = 10$ per farmer per year. This illustration involves simplifications; in practice the macroscopic coefficient $\kappa$ is unlikely to associate so directly with a microscopic quantity like minimum shipment size. In our reference market, $\kappa$ turns out to be the average stock exchanged per transaction when the market is at steady-state equilibrium $q^*$ (Results section *Trade without stock loss: the reference market*); this suggests $\kappa$ must exceed, but can still be arbitrarily larger than the minimum shipment size. In markets of money and financial products, for example, goods can be subdivided almost indefinitely, so we expect $\kappa$ to be potentially close to 0, which translates into an almost frictionless market, in line with the high liquidity of monetary and financial markets. In contrast, for indivisible goods such as livestock, the minimum shipment size is at least one, so we expect $\kappa \geq 1$. Due to a combination of production constraints and costly transportation of single animals, farmers often exchange batches of animals rather than individual animals, which further implies that $\kappa > 1$. For simplicity, $\kappa$ is estimated assuming the French cattle and swine markets are in steady-state equilibrium in years 2009 and 2010 respectively. Temporal analyses at both monthly and yearly scales indeed suggest that these markets seem to be in steady-state equilibrium in years 2005–2009 for cattle and year 2010 for swine (unpublished data). While we estimate $\kappa$ under the assumption of market steady-state equilibrium, we expect friction to exist both at equilibrium and outside of equilibrium (see Table 1 for explanation of non-equilibria accounted for by our FTM model). We also expect the coefficient of friction to be independent of whether the system is at steady-state equilibrium or not at least in the model as formulated; although we expect the effect of friction to be more apparent outside of equilibrium. Moreover, we expect estimation of friction to rely only weakly on the accuracy of the equilibrium assumption because the model is only weakly sensitive to linear variation in $\kappa$ (Fig. 2A). We find $\kappa = q^* \approx 3$ for cattle and $\kappa = q^* \approx 72$ for pigs, where $q^*$ is mean number of animals exchanged per transaction at steady-state equilibrium (calculated at the reference market steady-state equilibrium, see Material and Methods and ESM Section B). In fact, in line with this interpretation of the coefficient of friction, we find from direct inspection of the datasets that the empirical average $\langle q \rangle$, calculated based on pairs of trading agents, is $\langle q \rangle \approx 3$ (SD $\approx 6$) for cattle in 2009 and $\langle q \rangle \approx 63$ (SD $\approx 102$) for swine in 2010, where SD is the empirical standard deviation.

The second determinant of trade flow is the average transaction stock $q$ (see (4) and (5)). A characteristic of this market model is that the match between supply and demand is generally imperfect, in the sense that there can be *residual* supply or demanded stock after each transaction (imposed by the min function in (5) when there is excess accumulated stock per agent). These residuals lead to a transient 'excess frustration' in market agents whose duration depends on the 'fluidity' (or conversely, the 'friction') of the market. As trade flow depletes both stocks $S$ and $D$ and is an observable quantity, the cumulative trade flow over a period, $T([t_0, t_f]) = \int_{t_0}^{t_f} \Phi(t)\, dt$, is a measurable indicator of the evolution of satisfaction (or frustration) of market agents. Detailed analyses of our market model (ESM Section C.5 and Fig S.3) show that supply stock and demanded stock in the model's steady-state equilibrium are typically lower than the inventory stock of cattle in holdings recorded in France. These results support our definitions of supply and demanded stocks and suggest that the inventory stock of a farm is close to its maximal capacity. Our explicit representation of market transactions driven by imperfect and frictional individual-level supply and demand, from which potentially long-lasting out-of-steady-state market dynamics can emerge depending on the coefficient of friction (Results section *Market dynamics without shocks – effect of trade friction*), seems fundamentally different from some economic models (see Table 1 for explanations of potential differences relative to terminology or concepts in economics literature, ESM Section A for a comparative review of existing market models, and ESM Section C.1 for further details on the microeconomic foundations of our FTM model).

### 2.3. Market-epidemiological (ME) model with risk aversion

To investigate how disease epidemics and economic markets can influence each other, we model the spread and control of infectious diseases in markets by incorporating a standard epidemiological (E) model into our FTM model. We call this aggregate

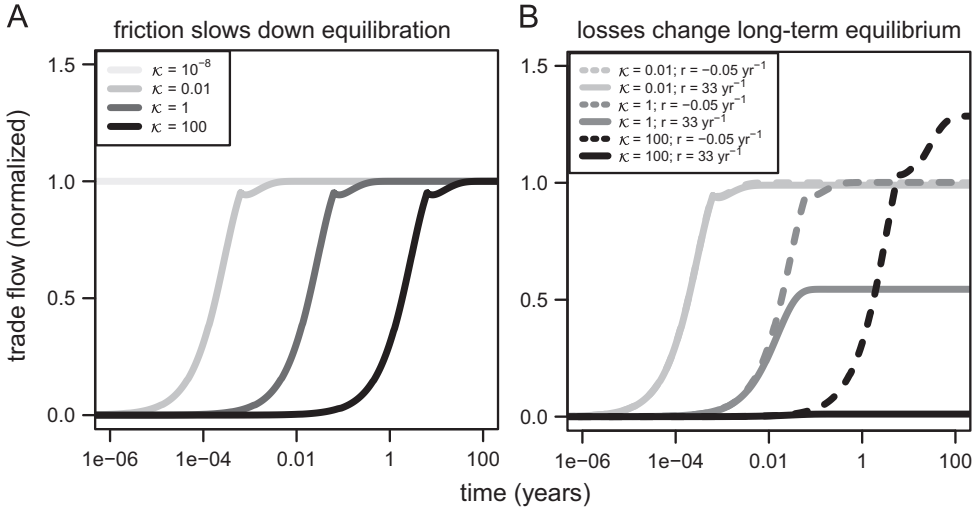


**Fig. 2.** The influence of frictional-trade on transient and long-term market dynamics. Evolution of normalised trade flow ($\Phi(t)/\Phi^*$) for variable levels of friction (from $\kappa = 10^{-8}$ in light grey to $\kappa = 100$ in dark) when losses are negligible ($r = 0\ \text{yr}^{-1}$ (A)) or non-negligible ($r \in \{-0.05, 33\}\ \text{yr}^{-1}$ in dashed and plain lines respectively (B)). Initial conditions are set to $[S(0) = 0, D(0) = 0, p(0) = 1.2p^*]$. We take $\mu = 10^{-6}$, $\varepsilon_S = 0.4$ and $\varepsilon_D = 0.8$. Other parameters are set from the French cattle estimates (see Table 3).

*market-epidemiological* (ME) model. In order to compare and integrate our framework with the literature, we include in the model adaptive risk aversion (RA) behaviour by the market agents.

The market agents are the population hosting the disease-causing pathogen. We use notation $\mathcal{N}_A$ to represent a set of agents of arbitrary type $A$, and $N_A$ to represent their number, which can vary over time. An agent can be a *strict supplier*, a *wholesaler* (supplier and demander), or a *strict demander* (Pautasso et al., 2010). The corresponding sets of agents are $\mathcal{N}_{S\setminus D}$, $\mathcal{N}_{S\cap D}$, and $\mathcal{N}_{D\setminus S}$; and, the total number of agents is $N=N_{S\setminus D}+N_{S\cap D}+N_{D\setminus S}$. The markets are hence composed of $N_S=N_{S\setminus D}+N_{S\cap D}$ suppliers and $N_D=N_{S\cap D}+N_{D\setminus S}$ demanders. We use a standard 'SIRS' epidemiological model (Anderson and May, 1991) where each agent (host) can be in the *Susceptible* ($X$), *Infectious* ($Y$), or Removed ($Z$) state (the notation $XYZ$ is preferred to $SIR$ to avoid confusion with the market model notation). Susceptible agents become infectious at rate $\Lambda(t)$ (the force of infection); infectious agents are removed at rate $\gamma$; and removed agents re-enter the market (recover) at rate $\nu$ (Fig. 1C). Here, 'removal' means that an infectious agent is detected and removed from the market by a regulator and becomes *inactive*. The infectious period ($1/\gamma$) is, therefore, the average time during which an infectious agent remains *active*, which is determined by the swiftness of the regulators (we assume that removal occurs immediately after detection and before the end of the biological infectious period). The recovery period ($1/\nu$) is the quarantine and sanitation time during which an infectious agent remains *inactive*, and is generally determined by regulators and agents. Each agent, whether a strict supplier, wholesaler, or strict demander, can be in each of the epidemiological states (Fig. 1C). For example, $N^X_{S\cap D}$ and $N^Y_{S\cap D}$ denote the number of susceptible and infectious wholesalers, respectively. Therefore, there are $N^{XY}_{S\cap D}=N^X_{S\cap D}+N^Y_{S\cap D}$ *active* wholesalers and $N^{XY}_S=N^{XY}_{S\cap D}+N^{XY}_{S\setminus D}$ active suppliers.

In specifying how disease spreads in the market model, we consider the general case where the pathogen can be transmitted both through trade routes ($tr$) and non-trade routes ($\overline{tr}$). We assume that transmission through trade occurs in the direction of transactions, i.e. through the shipment of contaminated stock from active infected suppliers ($\mathcal{N}^Y_S$) to active non-infected demanders ($\mathcal{N}^X_D$), while transmission through non-trade routes occurs from active infected agents ($\mathcal{N}^Y$) to active non-infected agents ($\mathcal{N}^X$). We also allow for import of contaminated stock though external flow $E$ (see (2)). In this case, the force of infection on demanders has terms associated with transmission through trade and non-trade routes, and a risk-aversion factor:

$$\Lambda(t)=\left[\Lambda_{tr}(t)+\Lambda_{\overline{tr}}(t)\right]P_{RA}(t). \tag{10}$$

The term for trade routes is $\Lambda_{tr}(t)=\beta_{tr}\cdot N^Y_S/N^{XY}_S$, i.e. a rate of transmission via trade $\beta_{tr}$, times the probability of transacting with an active supplier that is infected, $N^Y_S/N^{XY}_S$. The rate $\beta_{tr}=P_{tr}(q)\cdot\Theta(t)/N^{XY}_D$, i.e. the probability $P_{tr}(q)$ of acquiring infection from the average transaction stock $q(t)$ shipped from an infected supplier during a single transaction, times the transaction rate per active demander $\Theta(t)/N^{XY}_D$. If each of the $q$ units of stock has similar and independent probability of infection $\phi$, the probability of no infection from the stock is $(1-\phi)^q$, and the probability that the demander is infected during a single transaction by at least one unit of stock is $P_{tr}(q)=1-(1-\phi)^q$. Similarly, the force of infection via non-trade routes is $\Lambda_{\overline{tr}}(t)=\beta_{\overline{tr}}\cdot N^Y/N^{XY}$, the rate of transmission $\beta_{\overline{tr}}$ per active agents via yet unspecified transmission routes, times the probability of contacting an infected active agent, $N^Y/N^{XY}$. Finally

$$\Lambda_{tr}=\overbrace{\underbrace{\left[1-(1-\phi)^q\right]}_{P_{tr}(q)}\frac{\Theta}{N^{XY}_D}}^{\beta_{tr}}\frac{N^Y_S}{N^{XY}_S},$$

$$\Lambda_{\overline{tr}}=\beta_{\overline{tr}}\frac{N^Y}{N^{XY}}. \tag{11}$$

Following the literature (Funk et al., 2010), we include RA in our model by allowing agents to reduce their probability of infection per transaction, or per non-trade contact, according to the level of disease detection. We assume that RA reduces the probability of infection per contact by a factor $0\le P_{RA}\le 1$ given by

$$P_{RA}=\left(1-\frac{N^Z}{N}\right)^{\alpha}, \tag{12}$$

where $\alpha\ge 0$, and $N^Z$ is the number of inactive (detected) agents. When $\alpha=0$ there is no RA ($P_{RA}=1$), while in the limit $\alpha\to\infty$ RA is maximal ($P_{RA}=0$).

In addition to how epidemics affect the active agent population, we consider how epidemics affect their stocks. When an infected supplier is removed (at rate $\gamma$) its share of the stock, $S/N^{XY}_S$, is also removed; hence the rate of removal of infected supplier stock is $\rho_S=\gamma N^Y_S(S/N^{XY}_S)$. As the generic stock-loss rates in the FTM model without epidemics (Eqs. (2) and (8)) are analogous to $\rho_S$, in this paper we neglect losses other than $\rho_S$ by setting $r_S=r_D=0$.

Finally, the ME model is defined by the dynamics of market stocks (which generalises (8)) and agents under an epidemic (we show wholesaler equations as regard to epidemics, see the ESM Section D for other agents):

$$\frac{dS}{dt}=\overbrace{\underbrace{N^{XY}_S\sigma_0 p^{\varepsilon_S}}_{\Sigma_\oplus}-\gamma\frac{N^Y_S}{N^{XY}_S}S+E_S}^{\Sigma}-\overbrace{\underbrace{\frac{\min\{\Sigma_\oplus;\Delta_\oplus\}}{\kappa}}_{\Theta}\min\left\{\frac{S}{N^{XY}_S};\frac{D}{N^{XY}_D}\right\}}^{\Phi},$$

$$\frac{dD}{dt}=\overbrace{\underbrace{N^{XY}_D\delta_0 p^{-\varepsilon_D}}_{\Delta_\oplus}-\gamma\frac{N^Y_D}{N^{XY}_D}D+E_D}^{\Delta}-\overbrace{\Theta q}^{\Phi},$$

$$\frac{dN^X_{S\cap D}}{dt}=\nu N^Z_{S\cap D}-\Lambda N^X_{S\cap D},$$

$$\frac{dN^Y_{S\cap D}}{dt}=\Lambda N^X_{S\cap D}-\gamma N^Y_{S\cap D},$$

$$\frac{dN^Z_{S\cap D}}{dt}=\gamma N^Y_{S\cap D}-\nu N^Z_{S\cap D}, \tag{13}$$

where the market price $p(t)$ is given by (7) and the forces of infection by (10)–(12).

We will compare epidemics in the ME model with those in simpler models. In particular, we will consider the disease-free market steady-state equilibrium ($q^*=\kappa$ and $\Theta^*=\Phi^*/\kappa$)) as a system without explicit market dynamics, whose trade transmission rate (in (11)) is

$$\beta^*_{tr}=P_{tr}(q^*)\frac{\Theta^*}{N^{XY}_D}=\left[1-(1-\phi)^{\kappa}\right]\frac{\Phi^*}{\kappa N^{XY}_D}. \tag{14}$$

In addition, when comparing with the literature on epidemiological models with RA, we will take the limit of (14) in a frictionless (immediate steady-state equilibration) market ($\kappa\to 0$):

$$\beta^0_{tr}=\ln\left(\frac{1}{1-\phi}\right)\frac{\Phi^*}{N^{XY}_D}, \tag{15}$$

which is identical to known functional forms of the transmission rate (see e.g. Keeling and Rohani, 2008). As in previous epidemiological models incorporating host adaptive behaviour driven by health economics and other factors (Funk et al., 2009; Fenichel et al., 2011; Morin et al., 2013), we allow for RA-driven reduction in transmission rate ((15) or, more generally, that in (11)) through the RA factor (12), as in (10). However, by incorporating frictional-market dynamics, our model differs from those in the epidemiological literature, which, to our knowledge are comparable to frictionless markets.

## 3. Insights on market dynamics out-of-steady-state equilibrium and their interaction with epidemics

To help understanding the implications of our new theoretical framework, we study the FTM and ME models using a bottom-up approach (Table 2). We first analyse our FTM model in the absence of epidemics (Fig. 1B), and then explore the integrated ME model where epidemics and trade influence each other (Fig. 1A–C). The FTM and ME models are described mathematically by systems of non-linear ordinary differential equations ((8) and (13) respectively). We derive key analytical insights on the FTM and the ME models (see ESM Sections C and D respectively) using standard tools in the study of dynamical systems: existence and uniqueness theorem; steady-state equilibria, stability and bifurcation analyses.

Our FTM and ME models incorporate a variety of parameters, the values of which may not be well known. To assess the robustness of our findings to uncertainty or variation in these parameters, as well as uncertainty about initial conditions, we carried out two global sensitivity analyses (GSA) on key economic and/or epidemiological outputs. Specifically, we did one GSA for the FTM model, and another one for the ME model. We ranked the relative importance of parameters and initial conditions of interest using an improved version of the Morris method, a suitable GSA technique for high-dimension models (Campolongo et al., 2007). In a nutshell, the improved Morris method can discriminate the sign and overall influence of factors at a low computational cost and minor risk of error (see ESM Section B).

To parameterise the FTM model and estimate the range of values of the coefficient of friction that encompasses real markets (Table 3), we analysed trade records in two livestock markets: cattle (BDNI dataset) and swine (BDPorc dataset). The BDNI and BDPorc datasets are respectively managed by the French ministry in charge of Agriculture and the French professional union BDPorc. Each dataset details the movement of livestock within France among all economic agents involved in the supply chain, from strictly breeding farms to slaughterhouses, with various categories of wholesalers in between (e.g. breeding-fattening farms, strictly fattening farms, markets, dealers). To estimate model parameters, we extracted and reconstructed a table of individual transactions from a subset of each dataset. This table details, for each transaction: the supplier–demander pair, the date and associated volume of goods exchanged (see ESM Section B for details on the specific subset of the dataset that we have used). Traceability is imposed by the regulator at the scale of individual animals for cattle and batches of animals for swine. As a result, transactions could be directly extracted from the BDNI dataset but not from the BDPorc dataset. We explain in the ESM Section B how detailed transactions can be inferred from the BDPorc dataset.

### 3.1. Market dynamics without shocks - effect of trade friction

The FTM model introduces the notion of imperfect transactions with friction, bringing together differing economic models (see ESM Section A). To assess the impacts of friction on transient and long-run trade dynamics per se, we analyse the FTM model in the absence of epidemic shock (system (8)). We start by exploring the reference market where stock losses and external flows are neglected. We then analyse the cases of non-negligible stock losses ($r_S \neq 0$ or $r_D \neq 0$) and symmetric imports ($E_S = E_D = E \geq 0$). In all cases, transients and steady-states are investigated analytically and numerically.

#### 3.1.1. Trade without stock loss: the reference market

In the *reference market* we neglect stock loss ($L_S = L_D = 0$, i.e. $r_S = r_D = 0$) and external flows ($E_S = E_D = 0$), and denote the steady-state equilibrium value of state variables with a *star* in superscript. This market has an infinite number of steady-state equilibria (see ESM Section C for proof):

$$(S^* = S^*_{min}; D^* \geq D^*_{min}; p = p^*),$$

**Table 2**
Overview of the models investigated in our study.

| Model | Frictional trade | Price dynamics | Stock loss | External flows | Epidemics | Risk aversion |
|---|---|---|---|---|---|---|
| **Market dynamics without shocks (FTM)** | | | | | | |
| Reference market | ✓ | ✓ | · | · | · | · |
| Trade with stock loss (special case) | ✓ | · | ✓ | · | · | · |
| Trade with stock loss (general case) | ✓ | ✓ | ✓ | · | · | · |
| Trade with external flows | ✓ | ✓ | · | ✓ | · | · |
| Numerical illustrations | ✓ | ✓ | ✓ | · | · | · |
| **Market dynamics with epidemics** | | | | | | |
| Frictionless epidemiological model | · | · | · | · | ✓ | ✓ |
| Market-epidemiological (ME) model | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |

Ticks (dots respectively) represent the mechanisms included (not included respectively) in each model.

**Table 3**
Parameter values calculated for French cattle and swine markets.

| Notation | Meaning | Cattle | Swine | Unit |
|---|---|---|---|---|
| $\kappa$ | Coefficient of friction | 3.4 | 71.7 | None |
| $\Phi^*$ | Trade flow | 7,578,476 | 8,075,973 | Animals per year |
| $\Theta^*$ | Transaction rate | 2,224,182 | 112,683 | Deliveries per year |
| $\sigma_0$ | Per-agent generation rate of supply stock | 39 | 1474 | Animals per year |
| $\delta_0$ | Per-agent generation rate of demanded stock | 64 | 761 | Animals per year |
| $N_S$ | Number of suppliers | 193,354 | 5480 | None |
| $N_D$ | Number of demanders | 118,503 | 10,619 | None |
| $N_{S\setminus D}$ | Number of strict suppliers | 88,761 | 1314 | None |
| $N_{S\cap D}$ | Number of wholesalers | 104,593 | 4166 | None |
| $N_{D\setminus S}$ | Number of strict demanders | 13,910 | 6453 | None |

$$(S^* \geq S^*_{min}; D^* = D^*_{min}; p = p^*), \tag{16}$$

where $S^*_{min} = \kappa N_S$ and $D^*_{min} = \kappa N_D$ are respectively the minimal supply and demanded stocks for which the market is in steady-state equilibrium, and $p^* = (N_D\delta_0/N_S\sigma_0)^{1/(\varepsilon_S+\varepsilon_D)}$ is the unique equilibrium price, obtained by solving $\Sigma_\oplus(p^*) = \Delta_\oplus(p^*)$. The steady-state equilibria in supply and demand depend on the initial conditions (see ESM Section C); there is hence an infinite number of unstable equilibria $(S^*, D^*)$ with a switched fixed point: either $S^* = S^*_{min}$ or $D^* = D^*_{min}$. Since $p^*$ is unique, trade flow at steady-state equilibrium is unique and given by

$$\Phi^* = \Sigma_\oplus(p^*) = \Delta_\oplus(p^*) = [N_S\sigma_0]^{\varepsilon_D/(\varepsilon_S+\varepsilon_D)}[N_D\delta_0]^{\varepsilon_S/(\varepsilon_S+\varepsilon_D)}. \tag{17}$$

The market converges asymptotically to reference flow ($\Phi(t) \to \Phi^*$) and price ($p(t) \to p^*$) for any initial conditions or external perturbations (see ESM Section C for proof). The famous law of supply and demand (LSD) is a particular case (see ESM Section C for proof). The LSD implies that supply should equal demand (both in terms of accumulated stocks and flows of stock) when price is equilibrated; which is a very special case of our model with unique steady-state equilibrium $S^* = D^* = \max\{S^*_{min}, D^*_{min}\}$ (Fig. S2D). We study this case analytically, and then return to the general model formulation (system (8)) as there is little empirical support for the LSD (McCauley, 2009).

### 3.1.2. Trade with stock loss: detailed analysis of a special case

To study market transient behaviour, we consider initial conditions and parameter values that enable us to solve system (8) analytically. We set $[S(t_0) = D(t_0) \geq 0; p(t_0) = p^*]$ at initial time $t_0$ and track trade flow $\Phi$ until steady-state equilibration. This set of initial conditions is compatible with the LSD since $S(t_0) = D(t_0)$ and $p(t_0) = p^*$. Once accumulated over time at rates $\Sigma_\oplus$ and $\Delta_\oplus$, supply and demanded stocks are converted through trade ($\Phi$) and losses (at rates $r_S S$ and $r_D D$). For simplicity, we consider symmetrical losses ($r_S = r_D = r$). In this case, Eqs. (8) are symmetrical, which, with the above initial conditions ensuring that stocks remain symmetrical ($S(t) = D(t)$ for $t \geq 0$) and price remains constant, $p(t) = p^*$ (see ESM Section C). Therefore, system (8) reduces to

$$\dot{S} = \Phi^* - \left(r + \frac{a\Phi^*}{\kappa}\right)S, \tag{18}$$

where $a = \min\{1/N_S, 1/N_D\}$ is a dimensionless constant, and can be solved analytically to give (see ESM Section C):

$$\Phi(t) = \Phi^* \frac{\Phi^*}{\Phi^* + \frac{r\kappa}{a}}[1 - e^{-t(r + a\Phi^*/\kappa)}]. \tag{19}$$

In the long term ($t \to \infty$), flow diverges ($\Phi \to \infty$) if $r \leq -a\Phi^*/\kappa$, while, if $r > -a\Phi^*/\kappa$ flow converges to

$$\Phi_{eq} = \Phi^* \frac{\Phi^*}{\Phi^* + \frac{r\kappa}{a}}, \tag{20}$$

where $eq$ in subscript denotes steady-state equilibrium values in the general case (in contrast with the special case of the reference market where steady-state equilibrium is denoted by a star in superscript). In the more realistic case where losses are strictly positive ($r > 0$), trade flow is sub-optimal ($\Phi_{eq} < \Phi^*$), i.e., friction $\kappa$ and $r$ have an overall negative impact on steady-state equilibrium flow. If, however, the losses were negative with $r \in ]-a\Phi^*/\kappa, 0[$, trade flow would be over-optimal ($\Phi_{eq} > \Phi^*$). When losses are negligible ($r = 0$), we recover the reference market flow ($\Phi_{eq} = \Phi^*$).

### 3.1.3. Trade with stock loss: the general case

We now consider the more general case of a market with asymmetric positive losses ($r_S \geq 0$ and $r_D \geq 0$), and no external flows ($E_S = E_D = 0$), and consider arbitrary initial conditions $[S(0); D(0); p(0)]$ (see ESM Section C for derivations). From (8), we deduce that the steady-state equilibrium flow is always suboptimal:

$$\Phi_{eq} \leq \min\{\Sigma^{eq}_\oplus, \Delta^{eq}_\oplus\} \leq \Phi^* = \min\{\Sigma^*_\oplus, \Delta^*_\oplus\}. \tag{21}$$

Then, two cases arise. When the steady-state equilibrium limit of the average transaction stock $q_{eq} \equiv \min\{S_{eq}/N_S, D_{eq}/N_S\}$ is bounded by the per-agent supply (i.e. $q_{eq} = S_{eq}/N_S$), we find

$$\Phi_{eq} = \Sigma^{eq}_\oplus \frac{\min\{\Sigma^{eq}_\oplus, \Delta^{eq}_\oplus\}}{\min\{\Sigma^{eq}_\oplus, \Delta^{eq}_\oplus\} + r_S\kappa N_S}. \tag{22}$$

Conversely, when $q_{eq}$ is bounded by the per agent demand ($q_{eq} = D_{eq}/N_D$), we have, by symmetry, that $q_{eq}$ is given by (22) but with $N_S$ and $r_S$ replaced by $N_D$ and $r_D$ respectively. Importantly, Eq. (22) generalises the special case of Eq. (20) and, likewise, imply that $r_S$, $r_D$ and $\kappa$ have a negative impact on steady-state equilibrium flow. Since the dynamics with stock loss are not fully analytically tractable in the general case, we resort to extensive numerical simulations to confirm the key influence of $r_S$, $r_D$ and $\kappa$ on trade dynamics (see Global Sensitivity Analysis (GSA) of the FTM model in the ESM Section C).

### 3.1.4. Trade with external flows

To examine the impact of external flows on market dynamics, we consider, for simplicity, positive and symmetric external inflows ($E_S = E_D = E > 0$) and neglect losses ($r_S = r_D = 0$). Symmetry ensures that the steady-state equilibrium price and trade rate are the same as in the reference market, $p_{eq} = p^*$ and $\Theta_{eq} = \Theta^*$; while trade flow and average stock exchanged per transaction increase to $\Phi_{eq} = \Phi^* + E$, the solution to $dS/dt = dD/dt = 0$, and $q_{eq} = \kappa(\Phi^* + E)/\Phi^* > \kappa$, as $q = \Phi/\Theta$. Similar to (16), steady-state equilibrium supply and demanded stocks have two infinite sets of possible values: either $S_{eq} = \kappa N_S(\Phi^* + E)/\Phi^*$ and $D_{eq} \geq \kappa N_D(\Phi^* + E)/\Phi^*$, or $S_{eq} \geq \kappa N_S(\Phi^* + E)/\Phi^*$ and $D_{eq} = \kappa N_D(\Phi^* + E)/\Phi^*$. Hence, external flow increases trade flow through the average stock exchanged, but not the transaction rate, which is determined by number of agents, trade friction, and price.

### 3.1.5. Numerical illustrations

To confirm and extend the analytical insights on the impact of trade friction and losses on trade dynamics, we now explore the market model numerically. We use $[S(0) = 0; D(0) = 0; p(0) = 1.2p^*]$ as initial condition, and consider symmetric losses ($r_S = r_D = r$) and no external flows ($E_S = E_D = 0$). As the initial price is not equilibrated, trade flow equilibrates over time in a way that depends on market characteristics such as trade friction $\kappa$ and stock loss rate $r$ (Fig. 2). Increasing $\kappa$ drastically slows down market steady-state equilibration (Fig. 2A, B). In addition, the steady-state equilibrium flow depends on $r$. Without stock losses ($r = 0$), flow converges to the reference market level $\Phi^*$ (Fig. 2A). However, equilibrium flow is sub-optimal, $\Phi_{eq} \leq \Phi^*$, when there are positive losses ($r > 0$) and friction is large enough; conversely, flow is over-optimal when losses are negative $r < 0$ and friction is large (Fig. 2B). Scenarios with a wide range of loss rates $r_S$ and $r_D$, external flows $E_S$ and $E_D$, and initial conditions, explored via GSA, confirm these findings (see ESM Section C), which also agree with our previous analytical findings ((20) and (22)). Note that a negative loss rate corresponds to exponential inflows of supply and demanded stocks, a scenario that may appear to contrast real markets; we include it to show the general scope of the model. Overall, we find that, in the model, friction can increase market steady-state equilibration time by several orders of magnitude, while stock loss and external stock flow can alter the long-term steady-state equilibrium of the market. We expect these parameters to play a central role in understanding trade dynamics in markets and, therefore, in the epidemiology of trade-driven diseases.

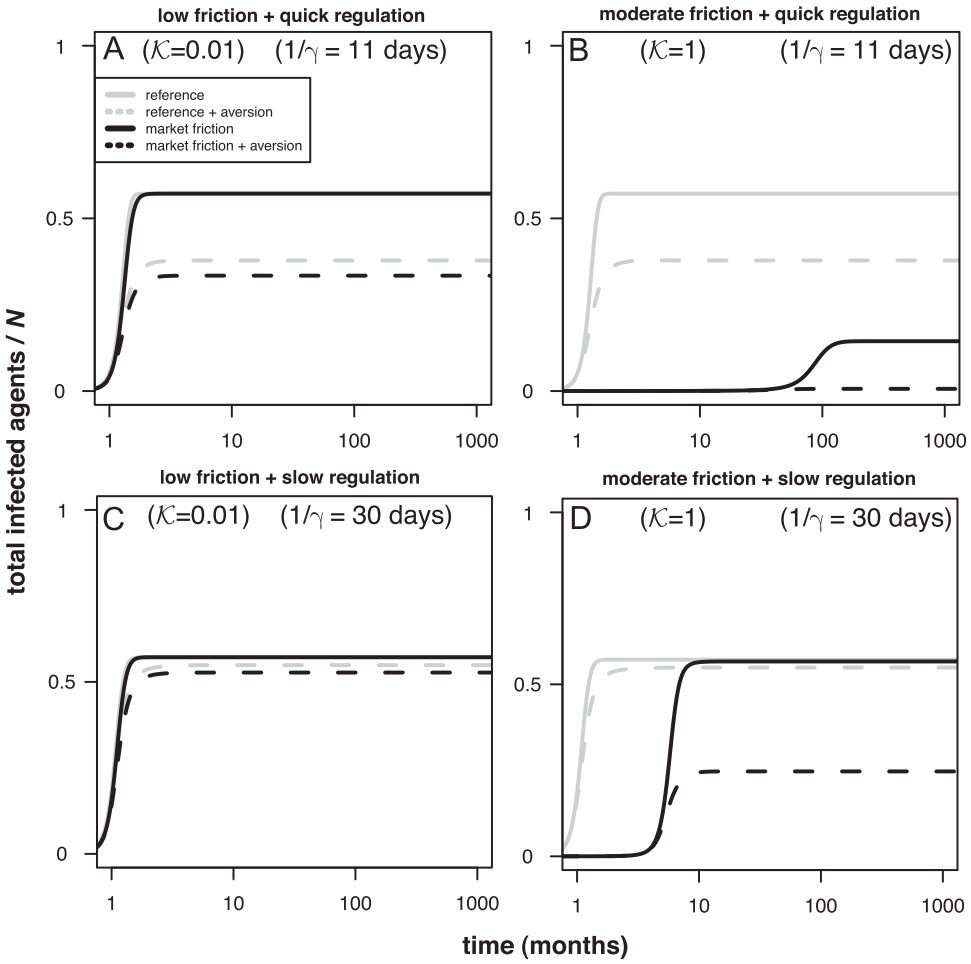


**Fig. 3.** Impacts of frictional-trade dynamics with risk aversion for contrasted delays in enforcement of regulation. Evolution of total infected agents normalised by the number of agents as a function of time for various types of frictional-trade dynamics with risk aversion and contrasted delays in enforcement of regulation. We assume trade is the only path of transmission ($R_0^{tr} = R_0$) and parameterise our model to replicate an *XYZ* outbreak ($\phi = 0.9780$, $\gamma > 0$, $\nu = 0$). Friction is either low ($\kappa = 0.01$, A–C) or moderate ($\kappa = 1$, B–D). The trade transmission rate is either set to its frictionless reference value $\beta_{tr}^0$ (in grey; see also (15)) or to its frictional-market value $\beta_{tr}$ (in black; see also (11)). RA is either negligible ($\alpha = 0$, plain lines) or non-negligible ($\alpha = 8$, dashed line). Enforcement of disease regulation is either quick ($1/\gamma = 11$ days (A, B); corresponding to a reference value of $R_0 = 4$ without friction) or slow ($1/\gamma = 30$ days, C–D; corresponding to a reference value of $R_0 = 11$ without friction). Initial conditions are set to start from a steady-state equilibrated market $[S(0) = \kappa N_S, D(0) = \kappa N_S, p(0) = p^*]$ and one agent per market category is initially infected (one strict supplier, one wholesalers, one strict demander). Other parameters are as in Fig. 2.

### 3.2. *Market dynamics with epidemic shocks*

We now explore the dynamics of the ME model, where epidemics and trade dynamics can influence each other (Fig. 1A–C and system (13)).

#### 3.2.1. *Relative impacts of trade friction and adaptive risk aversion for contrasted delays in enforcement of regulation*

We parameterise our model to investigate epidemics with contrasted delays in enforcement of regulation, and explore the impacts of frictional-trade and RA behaviour on epidemic dynamics (Fig. 3). We assume that the market has reached steady-state equilibrium before epidemic onset and trade is the only path of pathogen transmission. The trade-transmission rate has either a frictionless-market value $\beta_{tr}^0$ (Eq. (15); grey in Fig. 3) or its corresponding frictional-market value $\beta_{tr}$ (Eq. (11); black in Fig. 3). When market friction is very low (Fig. 3A–C), infection reaches the same endemic level with or without friction. The inclusion of RA (dashed lines) reduces the number of infected agents, and does so similarly with or without friction (Fig. 3A–C). This reduction in infections in the frictionless market is in agreement with the literature (e.g. Funk et al., 2010), and is expected as RA decreases the force of infection in response to an outbreak (10). As expected intuitively, increasing the delay in enforcement of regulation results in a lower effect of RA (Fig. 3A–C). When market friction has a significant level and provided the speed of regulation is quick enough (Fig. 3B), the endemic level is considerably lower in the frictional than in the frictionless market. Compared with the frictionless market, delaying regulation enforcement does not change the endemic level but slows down the epidemic (Fig. 3D). Again, the inclusion of RA has a similar effect on the endemic level with and without friction, but this is comparatively less important than the effect of a significant increase in friction ($\kappa$ from 0.01 to 1, Fig. 3A–C and Fig. 3B–D). Overall, our results suggest that trade friction can mitigate trade-driven disease transmission significantly, possibly more than in Fig. 3B–D, and to a significantly greater extent than RA behaviour, as our analyses of cattle market data suggest $\kappa = 3.4 > 1$. In addition, the combined effects of market friction and RA can lead to epidemic elimination (Fig. 3B) when trade is the only pathway of transmission and the delay to enforce regulation is small enough.

#### 3.2.2. *Impact of market friction level on epidemics*

Increasing trade friction reduces the severity (Fig. 3 and GSA of the ME model in the ESM Section D) and magnitude of the peak

(Fig. S4 and GSA in the ESM Section D) of epidemics when trade routes are the only pathway of transmission. Trade friction is also a key determinant of the epidemic threshold as assessed by the basic reproduction number $R_0$, a fundamental epidemiological summary. $R_0$ is the average number of susceptible agents infected by a single infectious agent propagated in an initially disease-free agent population (Anderson and May, 1991). In a deterministic framework, the pathogen eventually dies out if $R_0 \leq 1$; while if $R_0 > 1$, the pathogen eventually invades the population. In the general case with both trade and non-trade pathogen transmission (10), $R_0$ is given by (see ESM Section D)

$$R_0 = \frac{R_0^{tr} + R_0^{\overline{tr}} + \sqrt{(R_0^{tr} - R_0^{\overline{tr}})^2 + 4R_0^{tr}R_0^{\overline{tr}}\frac{N_S N_D}{N N_{S\cap D}}}}{2},$$
$$R_0^{tr} = \frac{\beta_{tr}}{\gamma}\frac{N_{S\cap D}}{N_S},$$
$$R_0^{\overline{tr}} = \frac{\beta_{\overline{tr}}}{\gamma}, \tag{23}$$

where $R_0^{tr}$ ($R_0^{\overline{tr}}$ respectively) is the value of $R_0$ when trade (non-trade) provides the only pathway of pathogen transmission. When trade is the only transmission route ($R_0^{\overline{tr}} = 0$), and inserting expression (11) for $\beta_{tr}$, yields (noting that $N_D^{XY} = N_D$ in this context)

$$R_0 = \frac{P_{tr}(q_{eq})}{\gamma}\frac{N_{S\cap D}\Theta_{eq}}{N_D N_S} \leq \frac{1}{\gamma}\frac{N_{S\cap D}\Phi^*}{\kappa N_D N_S}, \tag{24}$$

since $P_{tr}(q_{eq}) \leq 1$ and $\Theta_{eq} = \min\{\Sigma_{\oplus}^{eq}, \Delta_{\oplus}^{eq}\}/\kappa \leq \Phi^*/\kappa$. Therefore, $R_0$ vanishes in the limit when the market friction is large:

$$\lim_{\kappa\to\infty} R_0 = 0 \tag{25}$$

Result (25) stands for *any* modelling choice for $P_{tr}(q)$, including our current $P_{tr}(q) = [1-(1-\phi)^q]$. In addition to its mechanistic interpretation, this choice has the advantage of yielding a finite value for $R_0$ (Eq. (24)) in the limit of negligible friction ($\kappa \to 0$, when $\beta_{tr} \to \beta_{tr}^0$ given by (15)):

$$R_0 = \frac{\ln\left(\frac{1}{1-\phi}\right)}{\gamma}\frac{N_{S\cap D}\Phi^*}{N_D N_S}, \tag{26}$$

which allows comparison with existing epidemiological models that implicitly assume $\kappa = 0$. Increasing trade friction can cause decrease in $R_0$ up to the critical point where $R_0 < 1$ (Fig. 4A). Provided that the delay in enforcing regulations is small enough, this result also stands when trade is not the main transmission pathway (Fig. 4B). Therefore, accounting for trade friction is central to the estimation of epidemic thresholds in markets. This finding is confirmed by a GSA of $R_0$ in response to variation of its composing parameters (see GSA in ESM Section D). We can also use our expression of $R_0$ to rank the relative risk of sustaining an epidemic for various markets. As an example, French swine markets are characterised by a larger coefficient of friction ($\kappa = 71.7$; see Table 3) than the French cattle market ($\kappa = 3.4$). Since $R_0^{tr}(\text{swine})/R_0^{tr}(\text{cattle}) \approx 0.8$ for $\phi$ and $\gamma$ kept constant, trade of swine is less likely to sustain epidemics than trade of cattle. This result would appear counter-intuitive for typical epidemiological models, because trade flow is larger in swine than cattle (Table 3).

### 3.2.3. Open international market versus closed national market

When we consider an open market by including imports in the model, we find that international trade can boost epidemics moderately in comparison with closed national markets (Fig. S5). Imports increase stocks, and thus the average stock exchanged $q$ (5) and probability of infection $P_{tr}(q) = 1-(1-\phi)^q$ per transaction. However, imports do not affect the transaction rate $\Theta$. Therefore, as the force of infection involves the product $P_{tr}(q)\Theta$ and $P_{tr} \leq 1$, the effect of imports on the force of infection, and thus on epidemics, is limited. Another limiting factor is the current level of control measures, i.e. removal of infected agents after a given period of infectiousness.

### 3.2.4. Markets with different types and intensities of disease regulation

In our model, regulation to control or prevent disease spread in markets can be implemented in two ways: by removing infected agents at rate $\gamma$ or by limiting their re-introduction, after sanitation, at rate $\nu$. We show that epidemics can be mitigated by increasing the exit rate of infected agents and/or decreasing their reentry rate (Figs. 3 and S5B). The GSA suggests that increasing the removal rate $\gamma$ may be more efficient at mitigating disease than decreasing the rate of agent re-introduction $\nu$ (see ESM Section D). In particular, epidemics can be mitigated (Fig. 3B–D) and even eradicated by increasing $\gamma$ (Fig. 4A–B). Our analytical summaries of $R_0$ (Eqs. (23)) provide estimates of the maximal delay in the enforcement of regulation $1/\gamma_{max}$ that still allows prevention of epidemics for various types of markets and combinations of transmission pathways.

## 4. Discussion

Market trade routes propagate epidemics differently from other transmission pathways due to the unique contact structure that emerges from the willingness of agents to sell and buy goods (Fig. 1). This willingness arises from the business motivation of economic agents and inherent features of trade dynamics that differ from other epidemic-conducive human behaviour. In the model the we propose, trade markets involve recurrent interactions (transactions) between suppliers and demanders (consumers are demanders), and in each transaction there is a variable volume of goods exchanged ($q$) that may be contaminated and lead to the spread of infection. This notion of 'transaction' is somewhat different from that in the economics literature, particularly from the notion of 'match' in labour economics (see ESM Section A). Epidemiologically, it essential to precise this concept beyond an economic perspective because the transaction of goods, in addition to a market function, also provides a dynamic contact structure that can support disease transmission among agents and consumers. In our model, the frequency of transactions is limited by multiple constraints that we summarise in a single coefficient of friction, $\kappa$. Causes of such friction include production of exchangeable goods, the search for a trading partner, and the logistics of stock delivery. While there is much scope for discussing interpretation, this coefficient can be easily measured as a characteristic feature of markets; we have estimated $\kappa$ relatively easily from data on trade flow of livestock (see ESM Section B for application to exchanges of swine and cattle in France). In relation to the concepts in economics of "matching friction", and other types of friction induced for example by transaction costs (see Table 1), the concept of trade friction used by us accounts additionally for production constraints, e.g., when the minimum tradable unit is one living animal. This tangible mechanism has significant epidemiological consequences as well as economic implications, and is absent in models that implicitly assume exchange of continuous amounts of goods, including 'infinitesimal pieces of animals'. We believe that the notion of frictional dynamical transactions is an improvement on existing market models (see ESM Section A for details) as it may help to better understand the interactions between trade and the epidemiology of infectious diseases.

Taken together, our findings show that frictional markets are associated with a specific response to infectious diseases that contrasts with the response of other complex systems that sustain epidemics

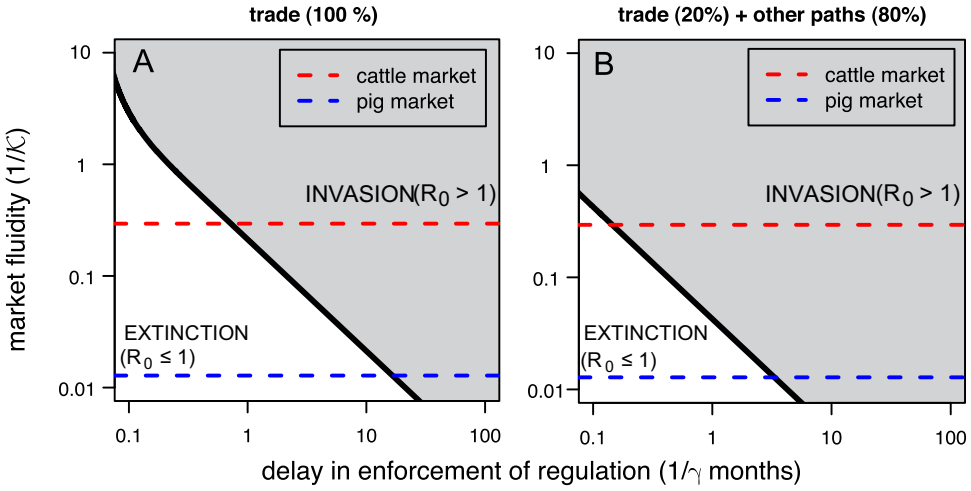


**Fig. 4.** Maximal delay in enforcement of regulation that still allows prevention of epidemics depending on market fluidity and other paths of transmission. Probability of invasion $P_I$ for various levels of market fluidity $1/\kappa$, delays in enforcement of regulation $1/\gamma$ and intensity of other paths of transmission (A, B). For each panel, the black curve represents the equation $R_0 = 1$ that separates the $(1/\kappa, 1/\gamma)$ space into two subspaces: the area under (above) the curve leads to an extinction (invasion) of the disease, i.e. $P_I = 0$ ($P_I = 1$). The epidemic is either only caused by trade (the black curve is given by $R_0^{tr}(\kappa_c, \gamma_c) = 1 R_0 = 1$; A) or by trade and other paths of transmission (the black curve is given by $R_0^{tr}(\kappa_c, \gamma_c) = 0.2 R_0 = 0.2$; **B**), where $1/\gamma_c$ is the maximal delay to enforce regulation corresponding to the critical amount of friction $1/\kappa_c$, the only value of $1/\kappa$ for which $R_0 = 1$. The dashed red (blue) line represent the estimated level of market fluidity of the French cattle market (French swine market). Other parameters are as in Fig. 3. (For interpretation of the references to colour in this figure caption, the reader is referred to the web version of this paper.)

and are often assumed to be frictionless. In particular, the coefficient of friction $\kappa$ is a central parameter governing trade and disease dynamics: $\kappa$ can increase market steady-state equilibration time by several orders of magnitude (Fig. 2) and suppress trade-driven disease transmission to a significantly greater extent than RA behaviour (Fig. 3). Our findings hence suggest that while frictional markets can still be responsible of disease introductions, trade itself cannot sustain epidemics in such markets. This counter-intuitive implication should be taken with care until confirmed by subsequent studies grounded on agent-based models.

The outcomes of our model suggest that to minimise contagion in markets, $\kappa$ could be increased to allow for larger-volume, less-frequent transactions, without necessarily affecting overall trade flow, and therefore, business activity. However, increasing friction may be difficult to achieve in practice due to practical constraints underlying $\kappa$ (e.g. trucks have a limited size). The model also suggests that in markets with a given level of friction $\kappa$, international trade (Fig. 3C) and regulatory measures (Figs. 3D and 4A, B) have strong, but contrasting, influence on trade-driven epidemics. In today's globalised world, a key question is how to mitigate epidemics efficiently in open international markets. The GSA of the ME model suggests that increasing the intervention rate $\gamma$ is more efficient at mitigating disease than decreasing the business re-establishment rate $\nu$, irrespective of imports (see ESM Section D). In other words, an alternative policy for minimising contagion in markets could adjust the delay of intervention $1/\gamma$ depending on $\kappa$.

From a cross-disciplinary perspective, the FTM model can be used to approximate market dynamics when there is lack of empirical data. The model can also be used to predict the impact and potential non-equilibrium in trade dynamics caused by disturbances such as unexpected disease outbreaks, for example in livestock markets (Fig. S7). Three forms of non-equilibrium are accounted for by the FTM model (see Table 1 for details). One form is when the rates of change in supply stock, demanded stock, and price are not zero, i.e. when the system is not in steady-state or inter-temporal equilibrium. A second form is when there is disequilibrium in state variables at a given time such as a mismatch between supply and demanded stocks of trading agents; this may be caused by past disturbances such as epidemic outbreaks and abrupt loss or increase in supply or demanded stocks. We allow for this possibility by limiting the stock exchanged in each transaction to the match between supply and demanded stocks ($q$). A third form is when there is a difference between the rates of generation of supply stock and demanded stock at given market price, which is a form of imperfect competition, which could be caused by epidemics or market failures. The level of competitiveness in our model is influenced by parameter $\mu$, which sets a timescale for price dynamics but does not necessarily preclude an eventual steady state (see Table 1 for further details). We indeed expect that disease epidemics where transmission is predominantly driven by trade are likely to cause asymmetric economic shocks, in the form of persistent excess of either supply or demanded stock. For example, quarantine measures could cause limited accumulated supply and thus excess accumulated demanded stock, while disease-control measures or consumer fright could cause excess accumulated supply of goods. Moreover, with the exception of wholesalers, disease transmission will occur from suppliers to demanders rather than in the opposite direction (Section D.1.3 of the ESM). It follows that suppliers and demanders would generally be differently impacted by disease, and would also be differently targeted by disease-control measures. The specific outcomes would depend on the specific market and disease. Therefore, accounting for non-equilibrium in trade dynamics is of central importance for understanding market-epidemic feedbacks. However, some approaches have often assumed that all transactions occur without mismatch between supply and demand (e.g. Mas-Colell et al., 1995), i.e. without focusing on differences between stocks and flows that are crucial in a market-epidemiological context. In other words, these studies consider that there is no accumulation of stock of goods or of demand for goods over time by implicitly assuming that both suppliers and demanders are fully satisfied through transactions where there is perfect match between what is available for sale and what is wanted for purchase. In contexts of market disturbance caused by disease outbreak it is essential to allow for such unbalance over the time scale of the outbreak or longer. Hence, following disequilibrium approaches from economics (Fair and Jaffee, 1972), we have allowed for imperfect match between supply and demanded stocks; this required defining accumulated stocks in addition to what we called flows (or 'rates of generation') of supply and demanded stock, which, formally, are the default supply and demand in economics (Table 1). We note that while

'demanded stock' seems not to be considered in economics, there are good reasons for considering that demand can accumulate over time when we do not assume the current needs of buyers are fully satisfied in one time step but remain to be satisfied over an economically significant period of time. For example, farm owners who experience accumulating losses of livestock due to an ongoing undetected infectious disease would increase demand for livestock for sometime to replace ongoing animals deaths; alternatively, their would interrupt business; either route would have a lasting unbalancing influence on the wider market.

Further on the usefulness of a market-epidemiological model, predictive assessments of market resiliency to disturbance such as disease outbreak are important for health economics. However, economic models applied to the study of epidemics often focus on descriptive assessments that neglect temporal dynamics (e.g. Soliman et al., 2010) or predictive equilibrium-based discrete-time approaches (e.g. Zhao et al., 2006) that can be at odds with the multiple and contrasting time scales encompassed by economic and epidemiological processes. It is important to model in continuous time to adequately resolve the multiple processes of infection and the market transactions. Even when economic model of trade operate in continuous time, they apparently do not consider explicit contact structures among economic agents (e.g. Caputo, 2005), which render them inapplicable to describe epidemics. Epidemiologists, ecologists and network-scientists have a long-standing expertise in formalising such contact structures, which suggest the tools developed by those communities could also benefit to the study economic systems, especially when submitted to systemic shocks (see also May et al., 2008). The use of $R_0$ can also be of importance in economics; for example, in assessing whether trade can drive epidemics that have the potential to damage trade. Specifically, the ME model shows cases where, depending on whether $R_0$ is less or greater than 1, contagion in markets can lead to either full recovery or long-lasting recession of the economic sector. However, interestingly, when considering the dynamics of a perturbed market per se, i.e. the FTM model without coupling to an epidemiological model, we typically find that the market is robust to disturbance as it returns rapidly to the original steady-state equilibrium. A fundamental aspect, therefore, lies in the feedback between dynamical processes that are often left separated because they associate with different disciplines or research communities. We note, however, that $R_0$ has limitations in its usefulness, and in particular is not a good predictor of the severity of an outbreak. For example, our model suggests that in face of an epidemic scenario ($R_0 > 1$) (24) swift regulation can eliminate the epidemic and mitigate the impact on the market when there is risk aversion and moderate friction, but not without risk aversion (Figs. 2B and S6, black-dashed versus plain-black curves). However, risk aversion is a behavioural adaptation that occurs over the course of the epidemic, and as such is not incorporated in the calculation of $R_0$, which focuses on the early stages of the epidemic. Another example is the case of an outbreak occurring in markets outside of equilibrium (i.e. prior to supply stock, demanded stock, and/or price having reached steady-state equilibrium. We find that the market's initial supply and demanded stocks generally have minor impact on trade and epidemics. However, an initially non-equilibrated price can strongly influence the size of the outbreak until the market reaches a steady-state (GSA of the ME model; Section D.2.4. of the ESM). Therefore, the same epidemiological system, characterised by a single $R_0$ value, can lead to contrasted epidemic and market outcomes, depending on adaptive risk-aversion behaviour and the market conditions at the time of epidemic onset. Both of these examples highlight the importance of accounting for adaptive mechanisms of interacting but apparently disparate dynamics, which bares resemblance to ecological dynamics.

From an economic perspective, our study only stands for emerging or re-emerging epidemics since agents adapt their behaviour in response to outbreaks. Further work of interest could transpose the current ME model to endemic diseases that are often anticipated by market agents. Agent anticipation could be implemented using explicit inter-temporal optimisation functions that are widely used in economics (e.g. by Zhao et al., 2006; see ESM Section C.1.5 for details and presentation of other potential extensions inspired by the economic literature). In addition, we could explicitly account for inventories and capacities of economic agents (Khan and Thomas, 2003), for example, by dissociating animals within a farm (an inventory) from animals for sell within a farm (a supply stock); or by dissociating the maximal number of animals that can be present within a farm (a capacity) from the number of animals expected to be bought (demanded stock). Our model can also be extended to account for heterogeneous contact structure such as individual-based networks of trading agents (e.g. Atalay et al., 2011). An explicit trade-agent model would account for transactions involving identified pairs of agents who jointly decide to exchange goods against money. For simplicity, this pairing process is assumed here to be governed by a homogeneous mixing process. However, where trade flow and the number of business partners are positively correlated at agent level, which is likely in many cases, we expect heterogeneous contact structure to boost epidemic development in comparison with homogeneous settings (Kamp et al., 2013). A key open question we are currently investigating concerns the conditions under which realistic levels of friction can also mitigate epidemics propagated on heterogeneous markets.

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## Appendix A. Supplementary data

Supplementary data associated with this paper can be found in the online version at http://dx.doi.org/10.1016/j.jtbi.2015.02.025.

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