Financial Contagion and Economic Development: an Epidemiological Approach*

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Abstract

We develop an epidemiological approach to analyze how financial contagion may affect and be affected by economic activity. We show that, according to specific parameter values, the economy may converge either to a non-speculative or to a speculative equilibrium: in the former situation the level of per capita income is maximal, while in the latter it is reduced by financial contagion. The presence of economic and financial feedback effects may also give rise to macroeconomic fluctuations during the transitional path, clearly showing that such economic and financial links are an important driver of the short run macroeconomic performance. By extending the analysis to a spatial dimension, we also show that financial contagion in some specific region may propagate quickly also in regions far away from those in which the contagion initially occurs, highlighting the role of regional policy coordination to avoid interregional contagion.

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JEL Classification: C60, E32, O40

1 Introduction

Following the pioneering work by King and Levine (1993), a large literature aiming to assess the implications of financial activities on economic development has rapidly grown (see, among others, a survey by Levine, 2005). From a theoretical perspective, only a limited number of works analyze the mechanisms through which financial intermediation impacts on economic growth through physical capital accumulation (Trew, 2014; Bucci et al., 2018), human capital formation (De Gregorio, 1996; De Gregorio and Kim, 2000; Bucci and Marsiglio, 2018) and technological progress (Pagano, 1993; Morales, 2003; Trew, 2008). From an empirical point of view, instead, an extensive body of studies discusses whether financial intermediation is beneficial or detrimental for growth, and the most recent view on this issue concludes that answering such a question is not simple at all, since the relation between economic growth and finance is non-monotonic, and most likely bell-shaped (Cecchetti and Kharroubi, 2012; Law and Singh, 2014; Arcand et al., 2015; Bucci et al., 2018).

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This clearly suggests that predicting how financial and economic activities interact is all but trivial. This type of conclusion has been reinforced after the recent global financial crisis, which has shown us that the financial and real sides of an economy are interconnected in a quite complex fashion which we still do not fully understand. Indeed, very little is known about the mechanisms through which a financial crisis can give rise to an economic crisis and how this in turn may feed the financial crisis again. Our paper wishes to shed some light on this issue by analyzing the way in which financial contagion and economic development affect each other and how specific policies can be used to improve the overall economic and financial outcomes.

Several papers, especially following the great recession in 2008, have tried to analyze how a financial crisis can give birth to financial contagion. By relying on either an empirical (Mistrulli, 2011; Baur, 2012; Mondria and Quintana-Domeque, 2013) or a theoretical (Allen and Gale, 2000; Martinez–Jaramillo et al., 2010, Ait–Sahalia et al, 2015) approach, extant literature focuses mainly on risk transmission between financial intermediaries and within the financial system, eventually accounting for transmission across national borders (Reinhart and Rogoff, 2009; Mishkin, 2011; Campello et al., 2010).[1] To the best of our knowledge, however, none of these works analyzes what contagion within the financial system implies for the real side of an economy and how economic activities at macroeconomic level in turn determine financial contagion. This is exactly the goal of our paper which wishes to conceptualize the mutual links between an economy's real and financial sides with particular emphasis on financial contagion. Specifically, we rely on an epidemiological approach to characterize how the exchange of assets between banks can determine the overall health status of the financial system, which determines the level of productivity in the whole economy. Since macroeconomic activity affects the number of asset exchanged between banks, the financial and economic sides are mutually related. Our model allows for two different equilibria: in the non-speculative equilibrium the level of per capita income is maximal, while in the speculative equilibrium it is reduced by financial contagion (which depends on both economic and financial factors). We also show that the convergence to the speculative equilibrium may give rise to economic fluctuations even in absence of random shocks, and such fluctuations are simply driven by the interactions between the economic and financial systems. By allowing for a spatial dimension we also analyze how the presence of financial contagion in some specific region may generate dramatic effects even in regions far away from those in which the contagion initially occurs. This extension allows us to provide a simple and intuitive explanation of why the recent financial crisis has rapidly become a global phenomenon, but also to provide a straightforward explanation of the widely-spread argument claiming that policy coordination across regions is effectively needed.

The paper proceeds as follows. Section 2 discusses the possible channels of financial contagion according to extant literature. All the reviewed studies focus on the implications of contagion on the financial industry itself and none accounts for its broader macroeconomic implications, which is the main goal of our paper. By taking an epidemiological approach, section 3 presents our a-spatial model of financial contagion and economic development which is entirely summarized by a system of differential equations. We analyze first the case in which the financial system affects the real economy, and then the case in which financial and economic systems are mutually related. We show that according to which parametric configuration holds true, and specifically according to how the effective risk-transmission rate and the effective risk-decay rate compare, the economy may converge either to a non-speculative or a speculative equilibrium. Section 4 presents our spatial model in which financial activities and capital may diffuse across the entire spatial economy; the model is thus described by a system of partial differential equations. We show that the presence of financial contagion in some specific region may generate dramatic effects even in regions far away from those in which the contagion initially occurs. Section 5 as usual presents concluding remarks and highlights directions for future research. Appendix A presents a generalization of the explicit results discussed in the body text, while appendix B extends our analysis to the case in which the exchange of asset

[1] The extent and speed of contagion is indeed the most striking novelty of the recent global financial crisis, in the sense that a rapid transmission occurred not only across different segments of the financial market but also across countries (see Beck et al., 2010).

between banks is proportional to the level of per capita income rather than per capita capital showing that also in this setting our main qualitative results still apply.

2 Financial Contagion in Interbank Markets

The financial and banking industry[2] is a complex system linking households, firms, and governments wishing to make payments, invest savings, issue or adopt credit, trade ownership stakes and assets, reduce financial risk, or disseminate financial information. Therefore, the financial sector is far more important than its direct share in the whole economy would suggest, and any turmoil affecting this industry can have dramatic effects also on the real economy as we will later clarify. The most important functions of the financial system consist mainly of: easing the exchange of goods and services by facilitating payments; pooling savings and allocating the available resources from lenders to borrowers; managing different liquidity and maturity needs by intermediating between short maturity lenders preferring and long maturity borrowers; and providing risk management. While the first one is the typical and most characteristic function of any financial system, allocating capital deals primarily with investing a financial institution's outside liabilities (i.e., obligations towards nonfinancial entities) into outside assets (i.e., claims on nonfinancial institutions). This process normally entails converting illiquid assets into money-like claims. The resulting maturity and liquidity transformation leaves banks vulnerable to a loss of funding, and interbank borrowing and lending serves precisely the scope of helping banks to manage the risk of fluctuations in their outside funding. Banks also help corporations manage their exposures to different categories of economic risk (stemming, for example, from exchange rates or interest rates, or else commodity-prices oscillations) through derivatives and other contracts. Again, banks hedge this risk by trading with other banks. The need for a place where all these exchanges between banks can occur represents therefore the most important motivation behind the existence of an interbank market in modern and globalized financial systems. As a result of the variegated functions that banks nowadays perform, interbank linkages are thus the key to the well-functioning of a financial system, since they allow banks with liquidity-shortages to borrow from banks with liquidity-surpluses. In a word, by redistributing funds among banks, the interbank market stabilizes the whole banking system. At the same time, however, the dense linkages existing between banks may also become a source of financial contagion through which solvency or liquidity problems of a single bank can spread and spill over to other banks as well, thus destabilizing the entire system.

Although the growing interconnectedness of the financial institutions is now considered one of the most important factors contributing to the recent global financial crisis of 2007–2008, it is also among the least understood, as the following questions are still at the center of the academic and policymaking debate. What are the most relevant channels through which financial contagion can take place in the interbank market? Does more interbank connectivity promote stability (via risk-sharing), or does it instead lead to greater fragility (by increasing the opportunity for contagion)? In other words, what is the role that the interaction between banks in a globalized financial system concretely plays in the propagation of a systemic shock and, following this shock, how is the interbank market's stability eventually affected by its structure? Is it possible to quantitatively assess the economic consequences of the widespread losses in the financial system due to shocks that arise from particular parts of it? In order to find a possible answer to these fundamental questions, in the last decade there has been an upsurge in the empirical as well as theoretical works on these issues. These works can be classified into two broad categories according to the approach used to describe interbank dynamics. On the one hand, network-based studies explicitly model the links between banks trying to micro-found their behavior and their interactions; due to the complexity of the banking industry's structure most of the results are derived from numerical simulations and such results largely depend on the specific assumptions made on the nature of these links. On the other hand, epidemiological studies take

[2] For the sake of simplicity in the remainder of the paper we will use the terms financial institution and bank interchangeably.

an aggregative approach to model the interactions between specific bank subgroups without modeling the behavior of any individual bank; this simplification in the structure of the banking industry allows to derive analytical results that provide a neat characterization of the possible outcomes. We will now briefly review such alternative approaches and their latest developments.

Because the structure of modern financial systems resembles, due to its dense connections between financial as well as nonfinancial entities, that of network-based industries (such as transport, power generation, or communications), most of the recent literature in the field adapts the existing network theory (see Newman, 2010, for an introduction to this theory) to the analysis of interbank interactions in order to emphasize the existence of four different sources of contagion occurring through the links of banks' balance sheets (see Glasserman and Young, 2016, for an extensive survey). The first, and probably the most intuitive, is represented by the occurrence of a negative shock to the value of a bank's assets: a drop, say, in the value of a bank's outside assets (for example, a downturn in an industrial sector to which the bank has made sizeable loans) is initially absorbed by the same bank's net worth. However, if the negative shock is big enough, the net worth of the bank is sooner or later wiped out, the bank becomes unable to fully repay all of its liabilities, and it ultimately defaults. Moreover, if the bank's payment shortfall is sufficiently large, it can push another bank to default as well. In the end, an initial shock to one bank's assets can spill over to other banks, creating a cascade of defaults (Wagner, 2010). Similarly, a second type of contagion arises from the fact that banks may have direct exposures to each other through borrowing and lending (Muller, 2006). To be more specific, suppose that a bank is forced to pull funding from another bank. Such a behavior can be triggered by an unexpected liquidity shock stemming, for example, from the reduction in the bank's deposits of nonfinancial entities. With its funding reduced, the bank needs to reduce its assets, as well. If it withdraws its lending to a second bank, this one will in turn need to reduce its own lending, so creating a funding run. Thus, while on the one hand interbank lending helps banks manage their liquidity risks, on the other hand it also creates ways through which a shock can be propagated from one bank to another one (Diamond and Dybvig, 1983; Allen and Gale, 2000; Freixas et al. 2000; Brusco and Castiglionesi, 2007; Gai et al., 2011). Another pathway through which banks' balance sheets may interact with each other in generating financial contagion is via information disclosure. Banks may have common exposures through their overlapping outside asset holdings (for example, real estate): a disclosure by one bank about its own assets may lead creditors to make inferences about the assets held by other similar banks, so producing a sort of "information contagion" (Acharya and Yorulmazer, 2008). A related branch of this literature highlights also the importance of within-network uncertainty as an amplifier of financial contagion in interbank markets (Pritsker, 2013; Caballero and Krishnamurthy, 2008). Finally, financial contagion can be originated by a "fire sale": if a bank is pushed to sell an illiquid asset (because of a shock that hits such an asset and creates a cash-shortfall for the bank), then the price of the same asset can be driven down, with the consequence that other banks (holding similar assets) may be forced to raise cash, as well. It is worth noticing that this negative externality operates solely through the price drop of the initially sold illiquid asset, and is independent on any payment obligation across banks. This source of financial contagion is therefore illustrative of the possible trade-off that originates from the search for diversification of a bank's investments portfolio: investment diversification reduces risks for each bank, on the one side, but if all banks hold similar diversified portfolios the system as a whole may be more vulnerable, on the other side (Caccioli et al., 2014; Chen et al., 2014; Duarte and Eisenbach, 2013).

Assessing how the stability of a financial system is ultimately affected by the structure of the interbank links within a network is not easy because connectivity is at the same time a channel for diversifying risks (which makes the financial system more stable) and a channel of contagion (which makes it less stable). Several studies have attempted to analyze this issue (see Glasserman and Young, 2016). By measuring the degree of connectivity through the probability of random connections, Nier et al. (2007) are among the first to find a nonlinear, possibly non-monotonic (M-shaped) impact of this variable on the total number of defaults in a network: increasing connectivity increases not only shock-transmission but also shock-

absorption, with the first effect dominating at a low level of connectivity and the second effect dominating instead at a higher level. More recently, by relying on an agent-based setup Acemoglu et al. (2015) confirm that interconnectedness not only improves the ability of a banking system to absorb shocks but it also increases the possibilities of contagion. In particular, they conclude that a ring network always produces the greatest number of defaults. By holding the network structure fixed, and instead varying the size of an initial shock, they also infer that a completely connected network produces the least number of defaults if the shock is small and the greatest number of defaults if the shock is large. Muller (2006) assesses the potential for contagion in the Swiss interbank market using data on bilateral bank exposures and credit lines within a simulation approach. Her conclusions suggest that the structure of the interbank linkages has a considerable impact on the interbank market's systemic stability. For one thing, the structure is decisive for the optimal redistribution of the available liquidity within the system (if banks with excess liquidity cannot pass it over to banks with a lack of liquidity, the banking system is definitely more prone to contagion). For another, the structure is crucial for the banking system's ability to absorb spillover effects (in case of a default situation, homogeneous and dense interbank networks turn out to be more stable than centralized and sparse networks). These findings are consistent with Allen and Gale's (2000) showing that "completeness" makes an interbank strucutre more robust to financial contagion.

Apart from such a network-based approach, and because of its similarity with an infectious disease, financial contagion has also been modeled by borrowing from the mathematical epidemiology literature (see Hethcote, 2000, for a review of epidemiological studies), which has also been successfully used in other disciplines to explain the diffusion of computer viruses, change in behaviors, advertising and rumors. Such an epidemiological approach has only recently been brought into the discussion of financial contagion, and the most representative works are represented by Toivanen (2013) and Kostylenko et al. (2017). Different from the network-based models which allow to distinguish across different sources of contagion, due to the richness with which they can describe individual bank's behavior and its interaction with other banks, epidemiological models do not allow to clearly identify the possible sources of contagion but allow to explicitly describe the contagion dynamics with the ultimate purpose to fully understand its underlying mechanisms. Both Toivanen (2013) and Kostylenko et al. (2017) rely on a susceptible-infected-recovered (SIR) epidemiological model to describe interbank dynamics: banks can be susceptible to speculation, infected by speculation or immunized from speculation after recovery from the infected phase. By interacting with infected ones, a susceptible bank may become infected as well such that financial contagion may spread within the financial sector exactly as a disease would diffuse across the human population. The ability of an epidemiological characterization of financial contagion to clearly describe how contagion propagates at aggregate level is the main advantage of this specific approach with respect to the more widely used network-based one. Using data on large cross-border banks in Europe, Toivanen (2013) shows that the crisis has, indeed, divided banks into two different categories: contagious and healthy (i.e., infected and susceptible, respectively). Moreover, as some banks can in principle be more dangerous (infectious) to the whole banking system than others, Toivanen (2013) also analyses whether the propagation of contagion is exacerbated by bank-specific factors, and ultimately which ones. He shows that the larger the first failing bank is, the more widespread the contagion will be; however, he also shows that other factors, including high clustering of the banking system, a high bank's connectivity, and large interbank loans, pose a greater systemic risk for the banking system as a whole than a bank's size.

Both the network and epidemiological approaches developed thus far to describe financial contagion focus only on the interbank dynamics and the consequences of contagion on the banking industry. Our goal in this paper is instead to analyze the macroeconomic implications of financial contagion, an issue which extant literature has to a large extent completely neglected. In order to do so we rely on an epidemiological approach which, because of its aggregative nature consistent with the typical macroeconomic framework, is the most natural candidate to develop an analytically tractable economic-financial model. Different from extant epidemiological literature, we assume that banks can be only susceptible to or infected by

speculation, meaning that upon recovery from speculation banks do not ever become immune. We believe that this assumption, allowing to characterize financial contagion as in a susceptible-infected-susceptible (SIS) model, represents interbank dynamics more realistically than in a SIR setup (where, instead, full immunization from speculation can effectively be achieved, a scenario difficult to imagine in the real world financial system). Moreover, different from existing epidemiological works which simply reinterpret the basic model of infectious diseases in financial terms, we first modify it in order to account for the most important features that characterize bank interactions and then integrate it in a traditional macroeconomic framework to account for the mutual links between economic development and financial contagion. A more detailed description of our epidemiological model of financial contagion follows.

3 The Baseline Model

We develop a simple model that analyzes how financial contagion arising from the financial system can affect and be affected by the real economy. We focus on a setup with heterogeneous banks which, through their normal business activities exchange assets, leading to transmission of risk from speculative to non-speculative banks. Specifically, the assets of both groups of banks are in the form of loans and non-loan assets, and non-loan assets may be either risky or non-risky . We assume that the two groups differ in the share of their assets held in the form of loans and in the type of non-loan assets held: on the one-hand, speculative banks hold only a small share of their assets in the form of loans and hold high-risk non-loan assets, while on the other hand, non-speculative banks hold a substantial share of their assets in the form of loans while also holding low-risk non-loan assets. We embed such a setting within an aggregative Solow-type (1956) neoclassical growth model in which, through the loan channel (that allows financial intermediaries to effectively funnel resources to the most productive uses in the economy), non-speculative banks positively influence the total factor productivity, so determining the level of income per capita. The transmission of risk from speculative to non-speculative banks affects capital accumulation through its impact on productivity. Our goal is to analyze how the degree of financial contagion can affect the real economy with a special emphasis on its level of economic development, proxied by the stock of per capita capital (we shall show in appendix B that the results do not change even if we use per capita income as a proxy of economic development).

In order to show more clearly our results and the different mechanisms that we wish to emphasize, we proceed in two steps. In the first we assume that the rate at which risk is transmitted across banks is exogenous. In this case, the direction of the relation between the financial and the real sides of the economy goes from the former to the latter: the share of speculative banks in the economy affects negatively the dynamics of physical capital accumulation, whereas capital is unable to affect the evolution of the share of speculative banks. As a second step, we extend such a basic setup to a more realistic framework where the risk-transmission rate is endogenous and dependent on the stock of per capita capital the economy is endowed with. In this more interesting case, the direction of the relation between the economy's financial and real sides is twofold: the share of speculative banks affects the dynamics of capital investment, with the capital stock being itself able, in turn, to affect the evolution of the share of speculative banks. Irrespective of the fact that the risk-transmission rate is exogenous or endogenous, in both cases we find the possibility of multiple equilibria, namely equilibria where the degree of financial contagion is either null or strictly positive. Our setting allows us to characterize the determinants of such equilibria and their dynamic properties, ultimately contributing to a better understanding not only of the complexity of the mutual relations between the economic and financial systems, but also of how such relations might be the source of endogenous fluctuations in per capita income, even in the absence of random shocks.

3.1 The Financial Side Affecting the Real Side

We consider a setting in which the interactions across banks in the financial sector are modeled as in a basic susceptible-infected-susceptible epidemiological framework[3] (Kermack and McKendrick, 1927; Hethcote, 2000). The whole population of banks, B_t, is composed of two groups, non-speculative, N_t, and speculative, S_t, such that at any time $B_t = N_t + S_t$. Both these two groups of banks hold assets ether in the form of loans or in the form of non-loan assets, and their strategies differ according to the choice of which share of assets to hold in the form of loans and to their chosen risk-profile in non-loan assets. While speculative banks hold only a small share of their assets in the form of loans, non-speculative banks hold a substantial share of loans. Moreover, while speculative banks opt for holding short-term very risky non-loan assets (e.g., asset-backed securities, or junk bonds, among others), non-speculative banks hold in their portfolio long-term, non-risky non-loan assets (e.g., perpetual bonds, for example). The size and the composition of the financial sector changes over time according to business dynamics (some new banks are created and others close down) and to the interactions between the two groups of banks. At any moment in time new banks are created at a given rate $0 < b < 1$, and we assume that newly created banks are all non-speculative at birth but may become speculative themselves by trading assets with speculative banks. Indeed, as the brief review of the previous section has made clear, due to everyday business interactions, speculative and non-speculative banks normally do sell and buy assets from each other in the interbank market. As an example, both the exchange of overnight loans in the reserve market and the exchange of personal/corporate loans in the loan market lead to an exchange of assets between speculative and non-speculative banks. Through these interbank exchanges, the overall high degree of risk characterizing the business profile of speculative banks may be transmitted also to non-speculative banks (via solvency or liquidity problems of a single bank), spreading across the entire financial system, such that financial contagion may take place. We postulate that risk is transmitted from speculative to non-speculative banks at a rate $\theta > 0$, measuring the average number of asset transactions required for the risk-exposure of the portfolio of a non-speculative bank to increase enough to be considered, in turn, speculative itself. High-risk assets held by speculative banks have a finite duration $\eta > 0$, which determines the decay-rate of risk-exposure. We also assume that the life span of banks is potentially infinite, however holding high-risk assets might lead banks to bankruptcy which occurs with a certain probability $0 < d < 1$, meaning that due to default reasons speculative banks may cease to exist. Thus, while speculative banks leave the financial market with a positive probability, such a probability is null for non-speculative banks. In light of the above characterization of the financial sector, given the initial conditions, $N_0 \geq 0$, $S_0 \geq 0$, and $B_0 = N_0 + S_0 \geq 0$, the interactions between speculative and non-speculative banks along with asset and vital business characteristics determine the flow of banks between the two subgroups, meaning that the number of speculative, non-speculative and total banks evolve according to the following differential equations:

$$\dot{N}_t = bB_t + \eta S_t - \theta \frac{N_t S_t}{B_t} \tag{1}$$

$$\dot{S}_t = \theta \frac{N_t S_t}{B_t} - \eta S_t - dS_t \tag{2}$$

$$\dot{B}_t = bB_t - dS_t \tag{3}$$

The above equations state that the risk-transmission, that is the extent to which risk may propagate across speculative and non-speculative banks is determined by $\theta \frac{N_t S_t}{B_t}$, where θ is the risk-transmission rate (measuring the average number of transactions required for risk-transmission to occur), and $N_t \frac{S_t}{B_t}$ represents the

[3]For the sake of simplicity, we rely on one of the simplest epidemiological frameworks, namely the susceptible–infected–susceptible model. More sophisticated setups which have been developed in the mathematical epidemiology literature (see, among others, Imran et al., 2014; Sharomi and Malik, 2015; Thompson et al., 2016) could be considered as well, but this would complicate our analysis without providing additional insights on the mutual relation between financial contagion and economic development.

number of random matchings per unit of time between the two groups of banks. Hence, for any $\theta > 0$ and $N_t > 0$, the risk-transmission ultimately depends on the share $\frac{S_t}{B_t}$ (rather than the absolute number, S_t) of speculative banks in the banks population, suggesting that the level of transactions between banks does not change with risk .[4] Also note that the rate at which speculative banks leave the market (d) is given by the probability of default induced by risk exposure: since non-speculative banks exchange assets with speculative banks, they might become speculative banks and forced to default as well. Moreover, the above model's formulation is completely deterministic, since in line with the mathematical epidemiology literature, we focus on a deterministic approximation of the stochastic model in which matching, and thus the exchange of assets, between speculative and non-speculative banks occurs randomly. Focusing on such a deterministic approximation of a stochastic framework allows us to analyze the implications of contagion on average, obtaining thus a setup consistent with traditional macroeconomic models.[5] As we shall see, this allows us to develop an intuitive framework to characterize the mutual links between macroeconomic outcomes and financial contagion.

By defining the share of non-speculative and speculative banks as $n_t = \frac{N_t}{B_t}$ and $s_t = \frac{S_t}{B_t}$, respectively, the above system can be recast as follows:

$$\dot{n}_t = b(1 - n_t) + \eta s_t - (\theta - d) n_t s_t \tag{4}$$

$$\dot{s}_t = \theta n_t s_t - (\eta + b) s_t - d s_t (1 - s_t) \tag{5}$$

The above equations describe how the relative composition of the banking sector changes over time. In particular, the spread of risk trough the exchange of assets implies that some non-speculative (N_t) banks may become speculative (S_t) and thus the share of speculative banks (s_t) may increase over time. Therefore, the prevalence of speculative banks in the financial system entirely captures the contagion effects in the financial system and thus in the following we shall refer to s_t as the *"degree of financial contagion"*. In particular, we wish to understand what are the determinants of financial contagion and what are its potential implications for economic development.

From the economic side we consider a Solow-type setting in which agents save a constant share $0 < v < 1$ of their income. Output can be either consumed or invested to accumulate more capital or else to replace depreciated capital. Specifically, output, Y_t, is produced according to a Cobb-Douglas production function by employing capital K_t and labor L_t as follows: $Y_t = A_t K_t^{\alpha} L_t^{1-\alpha}$, where $0 < \alpha < 1$ measures the capital share of income and A_t the total factor productivity (TFP). One of the most important roles of banks in modern economies consists of funneling savings to those firms that are able to employ (scarce) resources more efficiently, which in turn (at an aggregate level) results in a higher TFP (see, among others, Pagano, 1993; Morales, 2003; Trew, 2008; Bucci et al., 2018). In order to capture this effect, we assume that banks determine the level of TFP and in particular we postulate that this level increases with the share of non-speculative banks in the economy as follows: $A_t = a n_t^{\beta}$, where $a > 0$ is a scale parameter and $\beta > 0$ measures the efficiency of intermediation activity in affecting productivity.[6]. By denoting with $n > 0$ the growth rate of the labor force (coinciding with the population size, since we abstract from unemployment), the evolution

[4] By relying on epidemiology terminology, we would say that risk-transmission is *frequency dependent*, rather than *density dependent* (see Hethcote, 2000).

[5] It may be possible to analyze also the stochastic version of our banking model which, in line with other studies on financial contagion (see Glasserman and Young, 2016, for a recent survey), would take a network structure. In this framework banks represent nodes within a specific fixed network of financial institutions and at random times a given bank exchanges assets with some of its closest neighbors leading eventually to risk transmission. Apart from precluding analytical results, this alternative formulation has the disadvantage of not being easily reconciled with the typical macroeconomic setup that we need to rely upon in order to link the financial and the real sides of the economy. For this reason, we do believe that our approach based on the deterministic approximation of such a stochastic model represents the most effective way to address our research questions.

[6] Note that in our framework when all banks are non-speculative ($n_t = 1$) the level of the TFP is maximal ($A_t = a$), while the presence of speculative banks ($n_t < 1$) reduces the TFP ($A_t < a$). This is due to the fact that, unlike non-speculative banks, the speculative ones hold only a small share of their assets in the form of loans, which are the means to channel funds towards the most productive uses in an economy. Since such a share is small, speculative banks have negligible effects on the TFP. We

of per capita capital, $k_t = \frac{K_t}{L_t}$, is given by:

$$\dot{k}_t = van_t^{\beta} k_t^{\alpha} - (\delta + n)k_t, \tag{6}$$

As usual we denote with lowercase letters per capita variables and with uppercase letters aggregate variables, thus $y_t = \frac{Y_t}{L_t}$ denotes per capita output which is given by $y_t = an_t^{\beta} k_t^{\alpha}$. Since our focus is on per capita variables, in what follows we shall refer to per capita capital as capital and to per capita income as income as a matter of expositional simplicity.

Equations (4), (5) and (6) describe how the interactions between non-speculative and speculative banks determine the composition of the banking sector which, in turn, by affecting capital accumulation, ultimately determines the level of economic development. Since the shares of speculative and non-speculative banks sum to one at any moment in time, the system can be analyzed by focusing on only one of the two shares. In other words, we can simply concentrate our attention on how the evolution of the share of speculative banks, s_t (and, therefore, of the degree of financial contagion), affects capital accumulation. The dynamics of these two variables are given by the following equations:

$$\dot{s}_t = s_t[(\theta - b)(1 - s_t) - \eta - d] \tag{7}$$

$$\dot{k}_t = va(1 - s_t)^{\beta} k_t^{\alpha} - (\delta + n)k_t. \tag{8}$$

Note that in the case we are now focusing on (exogenous θ), the degree of financial contagion (s_t) affects the dynamics of capital, while the opposite is not true. This means that in this setting it is the financial side to influence univocally the real side of the economy. The above system (7) and (8) admits two equilibria, $E_1 = (\overline{k}_1, \overline{s}_1)$ and $E_2 = (\overline{k}_2, \overline{s}_2)$, where:

$$\overline{k}_1 = \left(\frac{va}{\delta + n}\right)^{\frac{1}{1-\alpha}} \qquad \overline{s}_1 = 0 \tag{9}$$

$$\overline{k}_2 = \left(\frac{va}{\delta + n}\right)^{\frac{1}{1-\alpha}} \left(\frac{\eta + b}{\theta - d}\right)^{\frac{\beta}{1-\alpha}} \qquad \overline{s}_2 = \frac{\theta - d - \eta - b}{\theta - d} \tag{10}$$

It is possible to observe that while E_1 always exists, E_2 does exist only whenever $\theta > d + \eta + b$ and such a parameter condition determines the stability properties of the two equilibria. Indeed, whenever a unique equilibrium exists (i.e., $\theta \leq d + \eta + b$) E_1 is asymptotically stable, while when two equilibria exist (i.e., $\theta > d + \eta + b$) E_1 turns out to be unstable while E_2 asymptotically stable. Thus, which equilibrium the economy converges to crucially depends on the number of equilibria, determined by how the risk-transmission rate (θ) and what we can refer to as the "effective risk-exposure decay rate" ($d + \eta + b$) compare. Note that the effective risk-exposure decay rate depends on the pure risk-exposure decay rate (η) but also on the default probability (d) and the birth rate of new banks (b). This is due to the fact that the probability of default, by forcing some speculative banks out of the financial system, and the birth rate, by diluting the presence of speculative banks in the financial sector (since at birth every bank is non-speculative), both contribute to increase the risk-exposure decay rate.

In particular, E_1 represents what we may refer to as a "non-speculative" equilibrium where the degree of financial contagion is null ($\overline{s}_1 = 0$) and thus capital achieves its maximal level ($\overline{k}_1$) determined only by economic fundamentals (namely, v, a, δ, n and α). E_2 represents what we may label as a "speculative" equilibrium where the degree of financial contagion is positive ($\overline{s}_2 > 0$) and entirely determined by financial factors (namely θ, η, b and d); this implies that capital does not achieve its maximal level ($\overline{k}_2 < \overline{k}_1$) since

could alternatively claim that both speculative and non-speculative banks contribute to the TFP, such that $A_t = an_t^{\beta} s_t^{\omega}$ where $0 \leq \omega \leq \beta$ measures the efficiency of speculative banks' intermediation activity in affecting productivity. Apart form reducing the negative macroeconomic effects associated with financial contagion, this setting would lead to the loss of multiple equilibria which, as we shall see later, are essential to give rise to nontrivial conclusions. By assuming that $\omega = 0$ we therefore focus on the most interesting and realistic case.

it is negatively affected by financial factors. Such factors, indeed, by increasing the share of speculative banks existing in the economy, decrease the TFP level, and therefore the economic incentives to invest in capital accumulation. Our above discussion suggests thus that the economy converges towards a non-speculative equilibrium whenever the effective risk-exposure decay rate is faster than the risk-transmission rate ($\theta \leq d + \eta + b$), while it converges towards a speculative equilibrium whenever the effective decay rate is slower than the transmission rate ($\theta > d + \eta + b$). The dynamics of the degree of financial contagion and capital are shown in Figure 1, in the case in which the non-speculative equilibrium is stable (dashed curves) and in the case in which the speculative equilibrium is stable (solid curves). It is worth observing that the evolution of financial contagion is monotonic, and so is the evolution of capital.

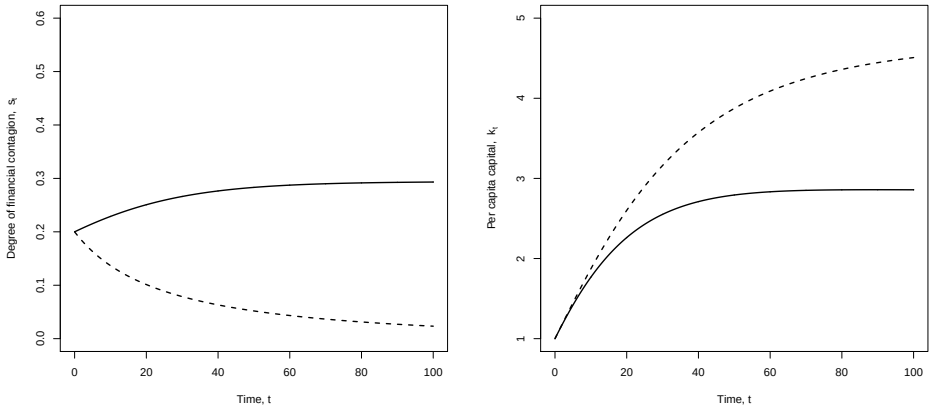

Figure 1: Evolution of the degree of financial contagion (left panel) and per capita capital (right panel) over time whenever $\theta \leq d + \eta + b$ (dashed curves) and $\theta > d + \eta + b$ (solid curves), starting from the same initial conditions, $i_0 = 0.2$ and $k_0 = 1$. Parameter values: $\alpha = 0.33$, $a = 1$, $v = 0.2$, $\delta = 0.05$, $n = 0.02$, $\theta = 0.2$, $\eta = 0.1$, $\beta = 1$, $b = 0.03$, with $d = 0.08$ (dashed curves) or $d = 0.02$ (solid curves).

It is interesting at this stage to emphasize the twofold role of the parameter d in our setting. As a matter of fact, the banks' default probability affects not only the capital level in a speculative equilibrium, but also the effective risk-exposure decay rate (that crucially determines which equilibrium the economy converges to). As long as two equilibria do exist, that is the effective decay rate is sufficiently low, an increase in the probability of banks' default increases the equilibrium capital level, as well; however, as soon as the effective decay rate becomes large enough, such that only one equilibrium does exist, then capital achieves its maximal level, which turns out to be independent of the probability of banks' default (since entirely determined by economic fundamentals). This suggests that, despite the common wisdom that policymakers should do whatever is in their power in order to rescue banks from bankruptcy, in order to favor economic development it might be ultimately most convenient to allow them to default and exit the financial market. In the end, the increased probability of banks' default would result in a higher equilibrium capital and income levels. We can summarize the results as follows.

Proposition 1. *In a Solow-type growth model, characterized by equations (7)-(8), in which the risk-transmission rate θ is exogenous, the economy converges to the non-speculative equilibrium whenever $\theta \leq d+\eta+b$, while it converges to the speculative equilibrium whenever $\theta > d+\eta+b$. Increases in the probability of banks' default (d) may either increase the speculative equilibrium capital level ($\overline{k}_2$) or allow the economy to achieve its maximal capital level ($\overline{k}_1$).*

In words, Proposition 1 states that in our framework an approach to economic policy aimed at rescuing banks no matter what might be detrimental to economic development. Policymakers should, instead, allow

banks eventually to fail, since this would ultimately result in higher levels of capital and income. The intuition behind this result is that a higher probability of default would lead speculative banks out of the financial market sooner; this will reduce the degree of financial contagion favoring a healthier (non-speculative) banking system which is conductive to faster capital accumulation and higher income levels.[7] This type of conclusion is in line with the results of some recent works (Hart and Zingales, 2014; Heitfield et al., 2010).[8]

3.2 Mutual Interdependence between the Financial and the Real Sides

We now extend our baseline model by endogeneizing θ in order to account for the presence of mutual interdependence between the financial and real sides of the economy. Specifically, we now postulate that the average number of asset-transactions across banks required for risk to be transmitted is time-varying and increases with per capita capital as follows: $\theta_t = \theta k_t^{\epsilon}$, where $\epsilon \geq 0$ quantifies the elasticity of the risk-transmission rate to the capital stock. By assuming that the such a rate increases with capital we explicitly take into account the fact that, everything else equal, in more developed economies on average a larger number of transactions between speculative and non-speculative banks is needed before transferring risk. This might be due, for example, to the role of financial regulation which is generally more stringent in advanced economies, where central banks' efforts to promote financial stability are more effective.[9] Under our new assumption on θ_t and its link with capital, the dynamic equations for per capita capital and the degree of financial contagion are mutually related as follows:

$$\dot{s}_t = s_t[(\theta k_t^{\epsilon} - b)(1 - s_t) - \eta - d] \tag{11}$$

$$\dot{k}_t = va(1 - s_t)^{\beta} k_t^{\alpha} - (\delta + n)k_t. \tag{12}$$

Note that in the special case in which $\epsilon = 0$ the model boils down to our baseline setup in which the transmission rate is an exogenous constant. Simple comparison of the systems (7)-(8) and (11)-(12) reveals that when the risk-transmission rate is endogenous and positively dependent on capital, the relation between the economy's financial and the real sides is no longer unidirectional: while the composition of the banking sector continues to affect capital accumulation and therefore economic development (see (8) and (12)), economic development (i.e., capital) now affects the banking sector (see (7) and (11)). As we shall see in a while this bidirectional relation between the financial and economic systems gives rise to important macroeconomic consequences.

Exactly as in our baseline model, the above system (11)-(12) admits two equilibria: $E_1 = (\overline{k}_1, \overline{s}_1)$ and $E_2 = (\overline{k}_2, \overline{s}_2)$. E_1 is the same non-speculative equilibrium as in the previous section with null degree of financial contagion and maximal capital level, as in equation (9). E_2 is again a speculative equilibrium characterized by a positive degree of financial contagion and capital less than its maximal level; clearly, with endogenous θ_t the equilibrium values of capital and share of speculative banks associated with the

[7] This is consistent with the view that any banking regulation needs a resolution scheme that governs bank failures in order to solve the trade–off between imposing market discipline and minimizing the effects of such failures on the rest of the banking system (Beck, 2011).

[8] By considering a simple model that abstracts from issues related to the degree of interconnectedness of financial institutions (which, instead, is the main focus of our paper), and where security markets are complete but consumers cannot pledge future income or wealth, Hart and Zingales (2014) show that a shock to banks disproportionately affects the agents who need liquidity the most, reducing thus aggregate demand and the level of economic activity. The optimal fiscal response to such a shock, however, is to help people, not banks. Therefore, even the special role played by banks *"[...] does not necessarily justify banks' bailouts."* (Hart and Zingales, 2014). Similarly, Heitfield et al. (2010) strikingly conclude: *"[...] pumping funds into the financial system may sustain zombie banks and amplify problems".*

[9] Note that we could alternatively claim that, due to the larger degree of complexity of the financial system, in more developed economies less transactions are needed for risk to be transmitted, that is $\theta_t = \theta k_t^{-\epsilon}$. Apart form reducing the beneficial effects of economic development on the financial system, this setting would lead the speculative equilibrium to be always stable. Our working assumption instead allows us to focus on the most interesting and probably realistic case.

speculative equilibrium are different from those in (10) with an exogenous θ. In fact, in order to derive the equilibrium values of capital and financial contagion in the speculative equilibrium, we need to solve the following equation for s:

$$\left[\theta\left(\frac{va}{\delta+n}\right)^{\frac{\epsilon}{1-\alpha}}(1-s)^{\frac{\beta\epsilon}{1-\alpha}}-b\right](1-s)=\eta+d \tag{13}$$

Such an equation cannot be solved explicitly in general, nonetheless it is possible to show that a unique solution always exists (see appendix A). Moreover, from (13) it is also clear that the equilibrium value of the share of speculative banks now depends (unlike the case of exogenous θ) on economic fundamentals (v, a, δ, n and α), as well. In order to look at the nature of the relation between the economic and financial systems in the simplest possible way, we focus on the case in which an explicit solution for (13) does exist, but our main results extend even to more general setups (see appendix A). This occurs whenever $\epsilon=\frac{1-\alpha}{\beta}$, which we assume to hold true in what follows. Note that such a parametric restriction is very mild since it allows us to consider situations in which ϵ is smaller or larger than or even equal to unity, according to whether β is larger or smaller than or equal to $1-\alpha$, respectively. If such a condition is met then the speculative equilibrium E_2 is characterized by the following equilibrium values of capital and degree of financial contagion:

$$\overline{k}_2=\left(\frac{va}{\delta+n}\right)^{\frac{1}{1-\alpha}}(1-\overline{s}_2)^{\frac{\beta}{1-\alpha}} \qquad 1-\overline{s}_2=\frac{b+\sqrt{b^2+4\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}(\eta+d)}}{2\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}} \tag{14}$$

As already discussed in the previous section E_2 exists only if a certain condition is met, and in this case the condition reads as $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}>d+\eta+b$, requiring what we can refer to as the "effective risk-transmission rate" $(\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}})$ to be larger than the effective risk-exposure decay rate. Exactly as before, whenever a unique equilibrium exists (i.e., $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}\leq d+\eta+b$) E_1 is asymptotically stable, while when two equilibria exist (i.e., $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}>d+\eta+b$) E_1 turns out to be saddle point stable while E_2 asymptotically stable. This suggests that economic fundamentals (v, a, δ, n and α) by determining the effective risk-transmission rate play an essential role in determining also which equilibrium the economy converges to. Specifically, we observe that, ceteris paribus, the effective risk-transmission rate increases with the saving rate but decreases with the depreciation rate and the labor force growth rate.[10] In other words, in more developed economies (i.e., those in which the saving rate is higher, and/or the labor force growth rate and the capital depreciation rate are lower) the condition for a speculative equilibrium to be reached is, all the rest remaining equal, more easily satisfied: in such economies the degree of financial contagion is likely to be higher (see equation (14)).

Regarding the determinants of the equilibrium outcomes in the economic and financial systems, the same results discussed in the previous section apply apart from the fact that now economic fundamentals affect the outcome in the financial system as well. The dynamics of the degree of financial contagion and capital are shown in Figure 2, in the case in which the non-speculative equilibrium is stable (dashed curves) and in the case in which the speculative equilibrium is stable (solid curves). We now observe that, while in the case of convergence to the non-speculative equilibrium dynamics are monotonic as in the previous section, in the case of convergence to the speculative equilibrium the evolution of financial contagion and capital are non-monotonic, suggesting that the presence of a mutual relation between the economic and the financial systems might give rise to macroeconomic fluctuations during which phases of economic expansion and contraction alternate each other. It is worth stressing at this stage that this occurs independently of the fact that our model is purely deterministic (actually, recall that it is a deterministic approximation of a stochastic model), thus the result is not driven by the presence of any exogenous source of random shocks,

[10] This is a direct consequence of the fact that the transmission rate, $\theta_t=\theta k_t^{\epsilon}$, is, for $\epsilon>0$, an increasing function of per capita capital and that at equilibrium (see equation (14)) capital depends positively on the saving rate and negatively both on the labor force growth rate and the depreciation rate.

as in most theories of economic fluctuations (see, among many others, the path-breaking works by King et al., 1988a, 1988b).[11] Instead, in our setting this result is due to a totally different mechanism based on the persistent and reciprocal interaction between the composition of the banking sector and the economic incentives to invest in physical capital[12]. Specifically, such a mechanism can be qualitatively explained as follows. Assume that $\theta_t = \theta k_t^{\varepsilon}$ raises (due, for example to an exogenous increase in θ): this will lead, ceteris paribus, to a rise in the number of speculative banks (S_t) and hence in the degree of financial contagion (s_t), as well; consequently, the share of non-speculative banks ($n_t = 1 - s_t$) and the productivity ($A_t = \alpha n_t^{\beta}$) would both decrease; with a lower TFP, per capita income (y_t) and per capita capital (k_t) would ultimately decrease too and this, in turn, leads to a lower level of θ_t. With a lower θ_t we would assist, respectively, to a decrease in S_t and s_t, and to a rise of n_t, A_t, y_t, k_t, and θ_t. This process will continue (with phases of declining capital that precede/follow phases of growing capital) until when a long run equilibrium is reached in which s_t, n_t, A_t, y_t, k_t, and θ_t remain all constant.

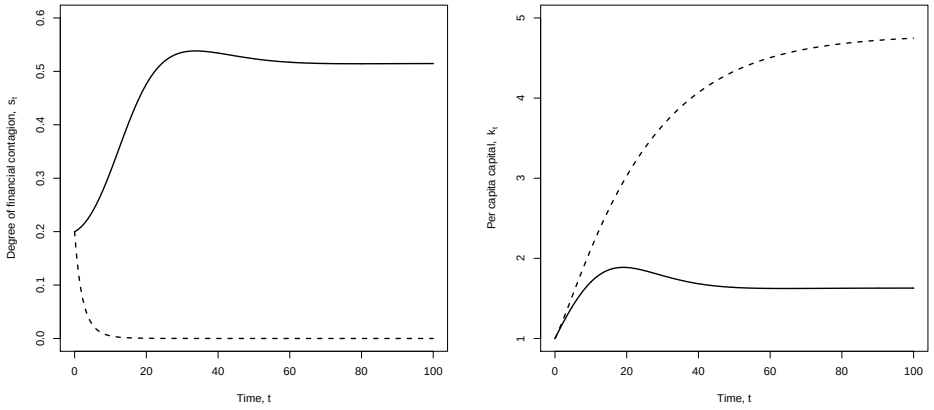

Figure 2: Evolution of the degree of financial contagion (left panel) and per capita capital (right panel) over time whenever $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}} \leq d + \eta + b$ (dashed curves) and $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}} > d + \eta + b$ (solid curves), starting from the same initial conditions, $i_0 = 0.2$ and $k_0 = 1$. Parameter values: $\alpha = 0.33$, $a = 1$, $v = 0.2$, $\delta = 0.05$, $n = 0.02$, $\theta = 0.2$, $\eta = 0.1$, $\beta = 1$, $b = 0.03$, $\epsilon = \frac{1-\alpha}{\beta}$, with $d = 0.5$ (dashed curves) or $d = 0.02$ (solid curves).

As a final remark, exactly as in the previous section banks' default probability d plays a significant role not only on the speculative equilibrium capital level, but also on the level of the effective risk-exposure decay rate, which determines what equilibrium the economy converges to. As long as the effective decay rate is sufficiently low and a speculative equilibrium exists, an increase in the probability of banks' default will increase the capital level, $\overline{k}_2$; however, if, following the same increase in banks' default probability, the effective decay rate becomes too high (compared to the effective risk-transmission rate) then the economy converges to the non-speculative equilibrium and capital ($\overline{k}_1$) achieves its maximal level, which is independent of the probability of default and entirely determined by economic fundamentals. We can summarize the results as follows.

Proposition 2. *In a Solow-type growth model, characterized by equations (11)-(12), in which the risk-transmission rate $\theta_t = \theta k_t^{\varepsilon}$ is endogenous and $\epsilon = \frac{1-\alpha}{\beta}$, the economy converges to the non-speculative equilibrium whenever $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}} \leq d + \eta + b$, while it converges to the speculative equilibrium whenever*

[11] Other theories of economic fluctuations based on deterministic settings are related to the existence of equilibrium indeterminacy (see for example, Benhabib and Farmer, 1994; Benhabib and Perli, 1994; Boldrin and Rustichini, 1994). Note that, differently from this branch of literature, our setup gives rise to fluctuations even if the equilibrium is determinate.

[12] See Marsiglio and Tolotti (2018) for a model where social interactions within the research industry might give rise to income fluctuations.

$\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}} > d + \eta + b$. *Increases in the probability of banks' default (d) may either increase the speculative equilibrium capital level ($\bar{k}_2$) or allow the economy to achieve its maximal capital level ($\bar{k}_1$).*

Proposition 2 is qualitative identical to Proposition 1 suggesting thus that rather than trying to rescue speculative banks at any cost policymakers might support economic development by allowing banks eventually to default. However, with respect to a situation in which the risk-transmission rate is fully exogenous, when it is endogenous the presence of reciprocal interactions between the economy's financial and economic sides makes policy decisions more interesting and less mechanical. Indeed, the effective risk-transmission rate depends positively on the saving rate and negatively on the capital depreciation and population growth rates. As it is well known, these rates (especially the saving and population growth rates) may greatly differ not only across industrialized and developing economies, but also within the group of advanced economies. In terms of our model, all this implies two different conclusions. The first is that, compared to developing countries (where, ceteris paribus, the effective risk-transmission rate is smaller), in advanced countries the probability of a bank's default needs to be significantly high in order to achieve a non-speculative equilibrium characterized by no financial contagion and maximal level of per capita capital and income. The second conclusion is that, looking instead solely at the group of industrialized economies, ceteris paribus, the probability of bank's default needed to achieve a non-speculative equilibrium may differ according to the individual country's specific economic fundamentals such as the saving rate, the population growth rate, the rate at which physical capital depreciates and, last but not least, the efficiency with which financial institutions are able to affect the TFP.

4 The Spatial Model

Financial contagion can be the result of two different mechanisms: the financial system affects the real side of the economy (which might feed back on the financial system as well), or one economy affects another giving rise to geographical patterns of contagion. Thus far, we have focused on the first mechanism showing the extent to which the financial and real sides of the same economy may be mutually related. We now focus on the second mechanisms trying to understand how financial contagion occurring in a single economy may spread and affect other economies as well. Indeed, contagion within the financial system is rarely limited to domestic boundaries and it often threats the financial stability of the global economy. As an example of the cross-border implications of financial contagion, Degryse et al. (2010) find that in 2006 a shock wiping out 25% (35%) of US (UK) cross-border liabilities against non-US (non-UK) banks could lead to financial contagion eroding at least 94% (45%) of the recipient countries' banking assets. They also show that contagion risk and the "speed of propagation of contagion" have increased over time during the 1999–2006 period and that contagion is more widespread in geographical proximities.

In order to look at such geographical implications of financial contagion, we extend our analysis to a spatial framework in which economic and financial activities diffuse across space. We assume a continuous space structure to represent that the spatial economy develops along a linear city, as in Hotelling (1929). A similar approach has been recently used to characterize the spatial implications of economic growth and environmental degradation (Boucekkine et al., 2009; Brock and Xepapadeas, 2010; Capasso et al., 2010; La Torre et al., 2015, 2018). We denote with $n_{x,t}$, $s_{x,t}$ and $k_{x,t}$, respectively the share of non-speculative and speculative banks and per capita capital level in the position x at date t, in a compact interval $[x_a, x_b] \subset \mathbb{R}$, and we assume that there are no diffusion flows through the borders of $[x_a, x_b]$, that is the directional derivatives are null, $\frac{\partial n_{x,t}}{\partial x} = \frac{\partial s_{x,t}}{\partial x} = \frac{\partial k_{x,t}}{\partial x} = 0$, at $x = x_a$ and $x = x_b$. In this framework, any position x may be interpreted as a specific location while a set of adjacent locations as a region in the spatial economy; such a possibility to distinguish between different regions allows us to account for the existence of regional heterogeneity and to understand what this might imply for the entire spatial economy. Different from what discussed earlier for the a-spatial model, the outcome in the financial system cannot be fully characterized

by focusing only on the evolution of the degree of financial contagion, $i_{x,t}$ since the population of banks is spatially distributed, $B_t = \int_{x_a}^{x_b}[N_{x,t}+S_{x,t}]dx$, and thus it is not necessarily true that the shares of speculative and non-speculative banks sum to one in each location x (they do sum to one over the whole spatial domain). We need thus to analyze the evolution of the share of speculative banks and the share of non-speculative banks over time and across space, along with their implications on per capita capital, and the spatial model can be represented though a system of partial differential equation as follows:

$$\frac{\partial n_{x,t}}{\partial t} = \lambda\frac{\partial^2 n_{x,t}}{\partial x^2} + (b+\eta)s_{x,t} - (\theta k_{x,t}^{\epsilon} - d_x)s_{x,t}n_{x,t} \tag{15}$$

$$\frac{\partial s_{x,t}}{\partial t} = \lambda\frac{\partial^2 s_{x,t}}{\partial x^2} + (\theta k_{x,t}^{\epsilon} - d_x)s_{x,t}n_{x,t} - (\eta+b)s_{x,t} \tag{16}$$

$$\frac{\partial k_{x,t}}{\partial t} = \lambda\frac{\partial^2 k_{x,t}}{\partial x^2} + va n_{x,t}^{\beta}k_{x,t}^{\alpha} - (\delta+n)k_{x,t} \tag{17}$$

In the above equations the only difference with the respect to the a-spatial model earlier described is related to the introduction of a spatial characterization. In each location x economic and financial activities evolve over time exactly as we discussed before. However, such activities evolve also across space and $\lambda \geq 0$ represents the diffusion parameter which measures the speed at which per capita capital, and the shares of speculative and non-speculative banks spread across space, which without loss of generality is assumed to be same for all variables. The spatial diffusion of capital has been recently discussed in a number of macroeconomic geography papers, where capital diffusion represents the effect of trade between adjacent locations (Boucekkine et al., 2009). The diffusion of speculative and non-speculative banks can be similarly interpreted as the effects of business transactions (i.e., trade of assets) between banks operating in adjacent locations. All parameters in the above equations could be space-dependent but for the sake of simplicity we assume that only the probability of default is, d_x, meaning that different locations may be characterized by a different banks' default reflecting differences in local economic policy or legislation.

We wish to understand what are the possible implications of introducing a spatial structure in our model of financial contagion, and in particular whether trade across adjacent locations, what we will refer to as a *"spatial externality"*, might be beneficial for long run economic development. Explicitly analyzing systems of partial differential equations is cumbersome and goes well beyond the scope of this paper. It is however possible to characterize some conditions allowing to clearly understand the implications of the spatial structure. This is stated in the following proposition.

Proposition 3. *In a spatial Solow-type growth model with no diffusion ($\lambda = 0$), characterized by equations (15)-(16)-(17), in which the risk-transmission rate $\theta_t = \theta k_t^{\epsilon}$ is endogenous and $\epsilon = \frac{1-\alpha}{\beta}$, at each location x two outcomes are possible:*
(i) if $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}} \leq d_x+\eta+b$ then the local economy will converge to the non-speculative equilibrium characterized by $\overline{k}_x^1 = \left(\frac{va}{\delta+n}\right)^{\frac{1}{1-\alpha}}$, $\overline{s}_x^1 = 0$ and $\overline{n}_x^1 = 1$;
(ii) if $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}} > d_x+\eta+b$ then the local economy will converge to the speculative equilibrium characterized by $\overline{k}_x^2 = \left(\frac{va}{\delta+n}\right)^{\frac{1}{1-\alpha}}(1-\overline{s}_2)^{\frac{\beta}{1-\alpha}}$, $\overline{s}_x^2 = 1 - \frac{b+\sqrt{b^2+4\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}(\eta+d_x)}}{2\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}}$ and $\overline{n}_x^2 = \frac{b+\sqrt{b^2+4\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}(\eta+d_x)}}{2\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}}}$.

Proposition 3 identifies some (sufficient) conditions allowing to characterize the two possible outcomes in a given location in our spatial economy with no diffusion, which converges either to the non-speculative or the speculative equilibrium according to how the effective risk-transmission rate and the effective risk-exposure decay rate compare in each location. If there is no spatial externality ($\lambda = 0$) each location is independent from others and thus, exactly as in our previous a-spatial analysis, if its specific effective risk-transmission rate is larger (smaller) than its specific effective risk-exposure decay rate it will converge to the speculative (non-speculative) equilibrium. Note that while the non-speculative equilibrium is spatially homogeneous,

the speculative one is spatially heterogenous suggesting that economies with similar economic and financial fundamentals may achieve an equilibrium characterized by different levels of capital and different degrees of contagion. It is thus possible that some locations within the spatial economy converge to the speculative while others to the non-speculative equilibrium. In the presence of the spatial externality ($\lambda > 0$) each location's outcome depends on the outcomes in other locations as well, thus understanding where a given location within the spatial economy will converge to is not possible. In order to shed some lights on this, we rely upon some numerical simulations which allow to visually represent the possible outcomes. Let us consider a framework in which economies in different locations across the whole spatial domain are structurally identical (also in terms of initial conditions) apart from the probability of default, which is spatially heterogeneous. In particular, let us focus on a situation in which the probability of default is high (such that $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}} \leq d_x+\eta+b$ holds) in the lateral regions while it is low (such that $\theta(\frac{va}{\delta+n})^{\frac{1}{\beta}} > d_x + \eta + b$ holds) in the central region, thus from our previous analysis we expect that, in the absence of the spatial externality, the lateral regions will achieve a non-speculative equilibrium while the central region a speculative equilibrium, and thus that the presence of heterogeneity in the banks' probability of default will give rise to heterogeneous outcomes in the spatial economy (see Proposition 3). Understanding whether and how the presence of the spatial externality affects this type of conclusion is our main goal.

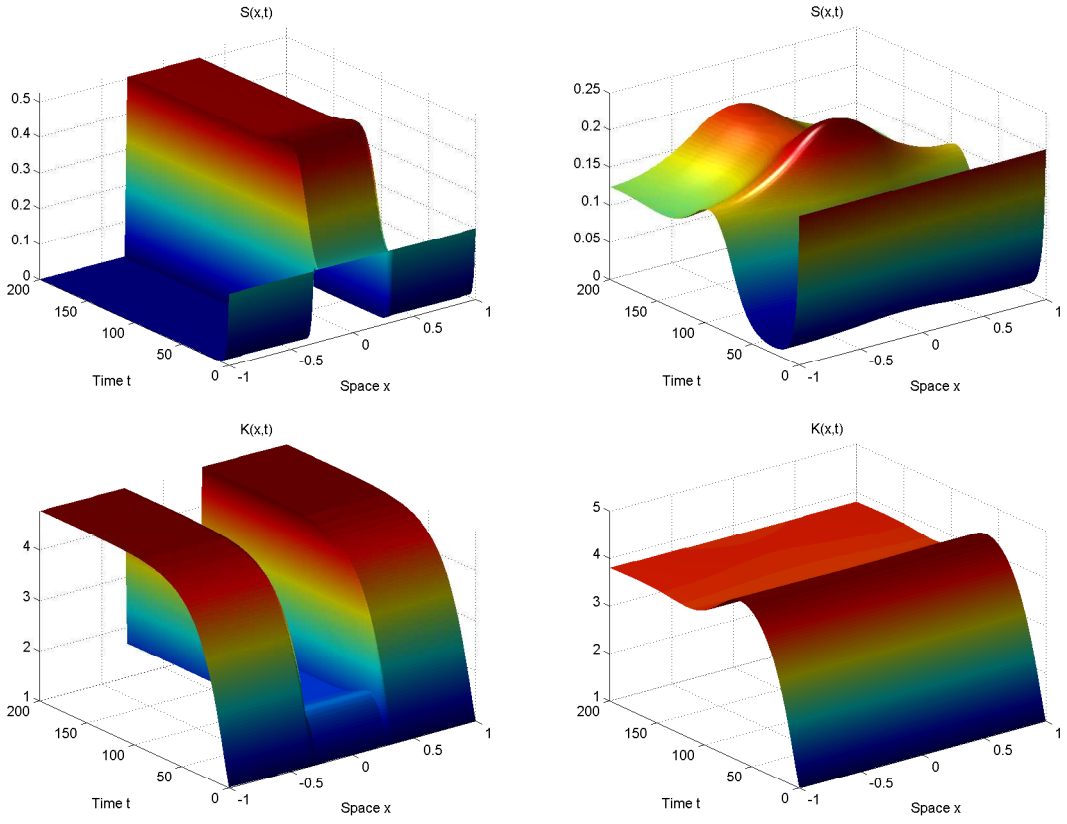

Figure 3: Evolution of the degree of financial contagion (top) and per capita capital (bottom), with no diffusion (left) and with diffusion (right). Probability of default relatively low in the lateral regions.

The implications of spatial externalities can be seen from Figure 3. The figure represents the spatio-temporal dynamics of the degree of financial contagion (top panels) and per capita capital (bottom panels) in the cases with no (left panels) and with (right panels) spatial externalities. The left panels represent the outcome in a situation in which $\lambda = 0$, meaning that the economy is spatially structured but any economy located in a specific venue is completely independent from economies located in other venues.

It is straightforward to note that, exactly as discussed earlier, the predictions of our a-spatial model are confirmed even in such a spatial framework: in the long run the lateral regions converge to a non-speculative equilibrium while the central region to a speculative equilibrium. Why this should be the case is obvious: the introduction of an exogenous spatial structure does not affect in any way the development experience of single economies since the spatial externality does not play any role and thus each region is completely independent from the others. The right panels represent instead the outcome in a situation in which $\lambda > 0$, meaning that the presence of diffusion due to trade implies that any economy located in a specific venue is affected by adjacent economies and thus each economy is no longer completely independent from all others. In this case we can note that the predictions of our a-spatial model are no longer confirmed, and also lateral regions converge to a speculative equilibrium. Since banks trade with each others independently of their specific location, contagion tends to spread across the entire spatial economy affecting the evolution of per capita capital. In particular the presence of diffusion tends to homogenize the long run spatial outcome, both in terms of financial and economic activities. With respect to the no diffusion case (left panels), the degree of contagion in the central region decreases and thus capital increases while contagion increases in the lateral regions and thus capital decreases. Overall, while the central region benefits from the spatial externality, the lateral regions suffer from trade spillovers. We can also note that with diffusion the dynamics of per capita capital and contagion become non-monotonic suggesting that trade can magnify the economic fluctuations induced by the interactions between the economic and financial sectors.

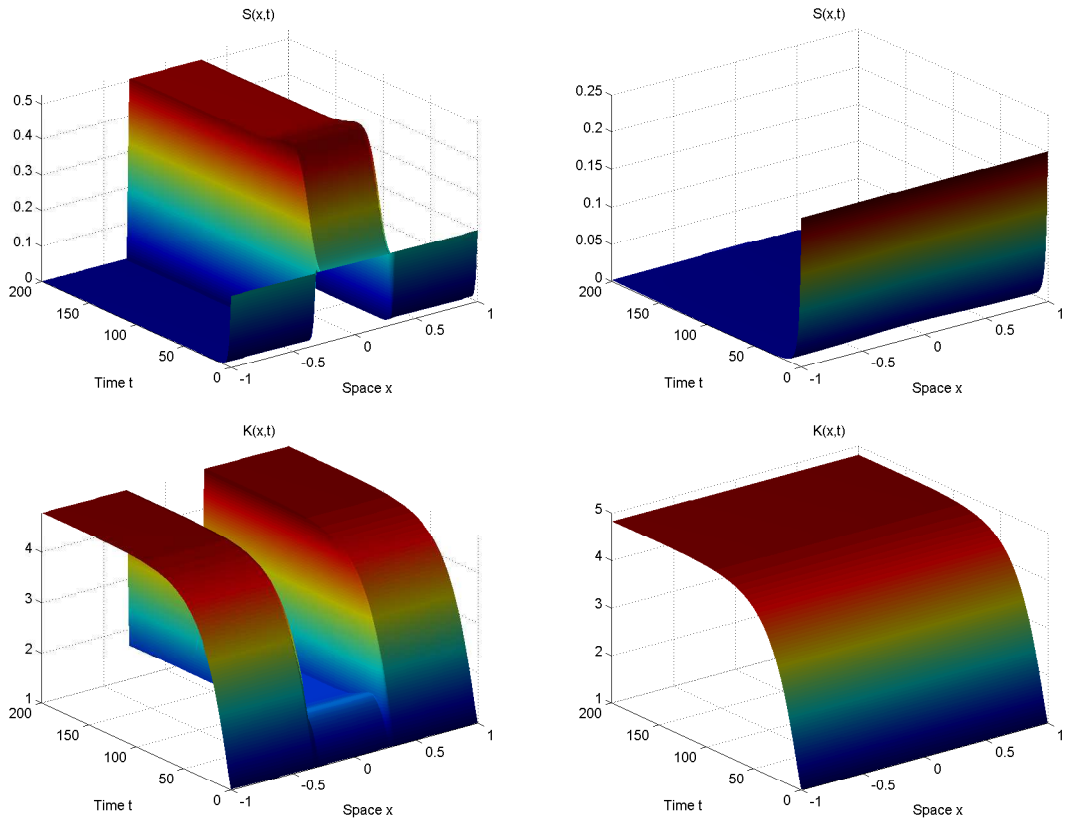

Figure 4: Evolution of the degree of financial contagion (top) and per capita capital (bottom), with no diffusion (left) and with diffusion (right). Probability of default relatively high in the lateral regions.

The above analysis suggests that because of the spatial externality, lateral regions suffer a reduction in income levels due to the fall in capital and rise in financial contagion. This occurs because the central region, due to its local economic policy and legislation, is not able to achieve a non-speculative equilibrium even in absence of trade spillovers. In such a case, by taking into account the presence of this detrimental effect due

to trade with the cental region, lateral regions can improve their long run outcome by relying on specific local policies targeting the effective risk-transmission rate or the effective risk-exposure decay rate. For example, they could intervene in order to increase the probability of banks' default. If the probability of default in the lateral regions increases enough to more than compensate for the negative effect induced by trade with the central region, then the entire spatial economy will monotonically converge to a non-speculative equilibrium (see Figure 4). This means that not only lateral regions will achieve a non-speculative equilibrium but also the central region will: an adequate change in economic policy in some (lateral) regions will benefit other (central) regions as well, improving the economic and financial outcomes in the entire spatial economy; this will also dampen the fluctuations in economic activity induced by the interactions between the economic and the financial systems, promoting a smooth process of economic development. These results suggest that policy coordination between different regions is essential in order to deal with financial contagion and the implications of spatial externalities.

Our analysis from Figures 3 and 4 allows us to state some interesting conclusions. Spatial externalities in the form of trade of assets between banks operating in different regions might be a source of economic instability: in case of localized shocks, contagion effects may propagate quickly across the entire spatial economy potentially giving rise to a global crisis, and policy decisions in a single economy are completely irrelevant (see Figure 3). In order to avoid a global spread of financial contagion, it is imperative that single economies cooperate with each other: if they do not, a too low probability of default in a single region may negatively affect other regions as well, meaning that economic and financial policies need to be mutually determined by taking into account the effects induced by trade spillovers (see Figures 4). We believe that these very simple and intuitive results can explain quite well the interactions between the economic and financial systems at world level during the recent global financial crisis.

5 Conclusion

The fact that the real and the financial systems are mutually related has been known for a long time, but how macroeconomic and financial activities affect each other is still an open question. This paper develops a simple framework to shed some light on such mutual feedbacks and in particular it tries to characterize the extent to which financial contagion arising from the financial system propagates to and is determined by the real economy. Specifically, we rely on an epidemiological approach to describe the transmission of risk within the banking sector which ultimately determines the degree of financial contagion; by allowing contagion to affect the TFP, introducing such epidemic dynamics in a Solow-type model of economic growth is straightforward; then, allowing economic development, measured by the level of per capita capital, to impact on the risk-transmission between banks permits us to close the loop. Such a framework allows us to derive some interesting conclusions: (i) the existence of multiple equilibria implies that economic policy can be effectively used to address the economy towards the speculation-free equilibrium, and this might simply require to promote banking efficiency by avoiding to rescue banks in distress; (ii) the complicated interactions between economic and financial systems imply that convergence to the speculative equilibrium might be characterized by cyclical fluctuations even in absence of random shocks, and such fluctuations naturally disappear if the economy is addressed to the non-speculative equilibrium. These results suggest that economic policy may play a fundamental role not only in improving long run (increasing the level of income per capita) but also short run (dampening the size of business fluctuations) macroeconomic outcomes. By extending the analysis to a spatial framework to account for spatial externalities we show that such conclusions are even more relevant in the presence of spatial heterogeneity, since regional policy if effective enough could improve the macroeconomic outcomes in the entire spatial economy.

To the best of our knowledge this is the first attempt to formally characterize the mutual links between financial contagion and macroeconomic activities. Therefore, we have tried to maintain the analysis as simple as possible in order to clarify the various mechanisms in place. In order to do so, the analysis has

been carried out in a purely dynamic setting without considering the optimal determination of economic and financial policy. Extending the analysis in order to characterize the associated optimal control problem may provide some further insights on how economic and financial systems are mutually related, both in an a-spatial and in a spatial framework; moreover, it may permit to quantify the welfare effects associated with economic and financial policy, allowing to assess thus the effective desirability of certain policies. These additional tasks are left for future research.

A A Generalization

In this appendix we show that the results discussed in the body text, based on some specific parametric restriction ($\epsilon = \frac{1-\alpha}{\beta}$), hold true also if we remove such a restriction. The cost of relaxing this condition is that we lose the explicit expression for the speculative equilibrium values of the degree of financial contagion and per capita capital. The equilibrium degree of financial contagion in the speculative equilibrium requires that equation (13) is verified. The following proposition states an existence and uniqueness result for the solution of this equation.

Proposition 4. *The equation (13) has a unique solution $s \in [0,1)$ if and only if $\theta(\frac{va}{\delta+n})^{\frac{\epsilon}{1-\alpha}} \geq d+\eta+b$.*

Proof. Equation (13) can be rewritten as

$$f_1(s) = \theta\left(\frac{va}{\delta+n}\right)^{\frac{\epsilon}{1-\alpha}}(1-s)^{\frac{\beta\epsilon}{1-\alpha}} = \frac{\eta+d}{1-s}+b = f_2(s) \tag{18}$$

The function f_1 is clearly strictly monotone decreasing, with $f_1(0) = \theta(\frac{va}{\delta+n})^{\frac{\epsilon}{1-\alpha}}$ and $f_1(1) = 0$. The function f_2 is monotone strictly increasing instead, with $f_2(0) = d+\eta+b$ and $f_1(1) = +\infty$. This implies that to have a unique solution to (13) is necessary and sufficient that $f_1(0) \geq f_2(0)$ which implies $\theta(\frac{va}{\delta+n})^{\frac{\epsilon}{1-\alpha}} \geq d+\eta+b$. ■

Whenever the condition in Proposition 4 is met, then a unique speculative equilibrium $E_2 = (\overline{k}_2, \overline{s}_2)$ exists. Specifically, the equilibrium value of capital is given by:

$$\overline{k}_2 = \left(\frac{va}{\delta+n}\right)^{\frac{1}{1-\alpha}}(1-\overline{s}_2)^{\frac{\beta}{1-\alpha}} = \frac{1}{\theta}\left[\frac{\eta+d}{1-\overline{s}_2}+b\right]$$

while the equilibrium value of the degree of contagion is the implicit solution to the following nonlinear equation:

$$\left[\theta\left(\frac{va}{\delta+n}\right)^{\frac{\epsilon}{1-\alpha}}(1-\overline{s}_2)^{\frac{\beta\epsilon}{1-\alpha}} - b\right](1-\overline{s}_2) = \eta+d$$

In order to analyze the stability properties of this equilibrium, we proceed via linearization. The Jacobian of the system of differential equations (11) - (12) evaluated at E_2 is given by the following expression:

$$J = \begin{bmatrix} (\theta\overline{k}_2^{\epsilon}-b)(1-2\overline{s}_2)-\eta-d & \epsilon\overline{s}_2\overline{k}_2^{\epsilon-1}(1-\overline{s}_2) \\ -va\beta(1-\overline{s}_2)^{\beta-1}\overline{s}_2^{\alpha} & va\alpha\overline{k}_2^{\alpha-1}(1-\overline{s}_2)^{\beta}-\delta-n \end{bmatrix}$$

It is straightforward to show that $J_{21} < 0$ and $J_{12} > 0$. The following calculations allow to determine the sign of J_{22} and J_{11}:

$$\begin{aligned} J_{22} &= va\alpha\frac{(1-\overline{s}_2)^{\beta}}{\overline{k}_2^{\alpha-1}}-\delta-n = va\alpha\left(\frac{\delta+n}{va}\right)-\delta-n = (\delta+n)(\alpha-1) < 0 \\ J_{11} &= -\overline{s}_2(\theta\overline{k}_2^{\epsilon}-b) = -\overline{s}_2\left(\frac{\delta+n}{1-\overline{s}_2}\right) < 0 \end{aligned}$$

These conditions imply that the trace of J is negative and the determinant of J is positive which leads to the conclusion that both eigenvalues of J are strictly negative, suggesting that E_2 is asymptotically stable. As discussed in the body text under the parametric restriction $\epsilon = \frac{1-\alpha}{\beta}$, also whenever this condition is not met a unique speculative equilibrium exists whenever a certain parametric restriction holds true, and whenever such an equilibrium exists this will be asymptotically stable. Not surprisingly, note that the condition in Proposition 4 ensuring existence and uniqueness of the speculative equilibrium is a generalization of the condition discussed in the body text, which indeed can be restored by setting $\epsilon = \frac{1-\alpha}{\beta}$. This discussion suggests that the results discussed in the body text apply even in more general situations. The evolution of per capita capital and financial contagion in the case in which $\theta(\frac{va}{\delta+n})^{\frac{\epsilon}{1-\alpha}} \geq d+\eta+b$ are illustrated in Figure 5, which shows that the qualitative behavior of the variables is identical to what discussed in the body of the paper; the only noticeable difference is related to the fact that the size of fluctuations in capital and contagion are much larger, suggesting that whenever ϵ is not restricted to take some specific value the macroeconomic effects of financial contagion can be substantially large.

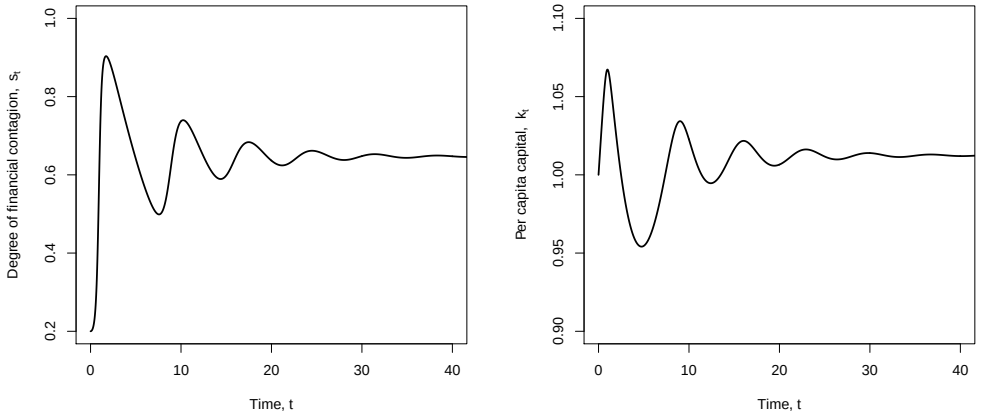

Figure 5: Evolution of the degree of financial contagion (left panel) and per capita capital (right panel) over time whenever $\theta(\frac{va}{\delta+n})^{\frac{\alpha}{\beta}} > d+\eta+b$, starting from initial conditions, $i_0 = 0.2$ and $k_0 = 1$. Parameter values: $\alpha = 0.33$, $a = 1$, $v = 0.2$, $\delta = 0.05$, $n = 0.02$, $\theta = 0.2$, $\eta = 0.1$, $\beta = 1$, $b = 0.03$, $\epsilon = 50$, with $d = 0.02$.

B A Different Formulation

We now present a different formulation of the relation between the financial and economic systems to show that even in more general setting our main results still hold true. We now assume that the average number of asset-transactions across banks required for risk to be transmitted increases no longer with per capita capital but with per capita income as follows: $\theta_t = \theta y_t^{\epsilon}$. The relation between financial contagion and capital accumulation in this case is summarized by the following equations:

$$\dot{s}_t = s_t\{[\theta a^{\epsilon}(1-s_t)^{\beta\epsilon}k_t^{\alpha\epsilon} - b](1-s_t) - \eta - d\} \tag{19}$$

$$\dot{k}_t = va(1-s_t)^{\beta}k_t^{\alpha} - (\delta+n)k_t. \tag{20}$$

As in the body of the paper, if $\epsilon = \frac{1-\alpha}{\beta}$, it is possible to explicitly find the equilibria. The speculative equilibrium E_2 is characterized by the following equilibrium values of capital and degree of financial contagion:

$$\overline{k}_2 = \left(\frac{va}{\delta+n}\right)^{\frac{1}{1-\alpha}}(1-\overline{s}_2)^{\frac{\beta}{1-\alpha}} \qquad 1-\overline{s}_2 = \frac{b+\sqrt{b^2+4\theta(\frac{va}{\delta+n})^{\frac{\alpha}{\beta}}(\eta+d)}}{2\theta(\frac{va}{\delta+n})^{\frac{\alpha}{\beta}}} \tag{21}$$

In this case, the economy converges to the non-speculative equilibrium whenever $\theta(\frac{va}{\delta+n})^{\frac{\alpha}{\beta}} \leq d+\eta+b$, while it converges to the speculative equilibrium whenever $\theta(\frac{va}{\delta+n})^{\frac{\alpha}{\beta}} > d+\eta+b$. Also in this case increases in the probability of banks' default (d) may either increase the speculative equilibrium capital level ($\overline{k}_2$) or allow the economy to achieve its maximal capital level ($\overline{k}_1$). The evolution of per capita capital and financial contagion are illustrated in Figure 6. We can observe that apart from some quantitative differences in the level of capital and contagion both during the transition and at equilibrium, from a qualitative point of view capital and contagion behave exactly as in our earlier formulation. This suggests that our main results hold true even in more general settings, and no matter the proxy of financial development (capital vs income) our model is able to effectively characterize the mutual links between the economy's financial and economic sides.

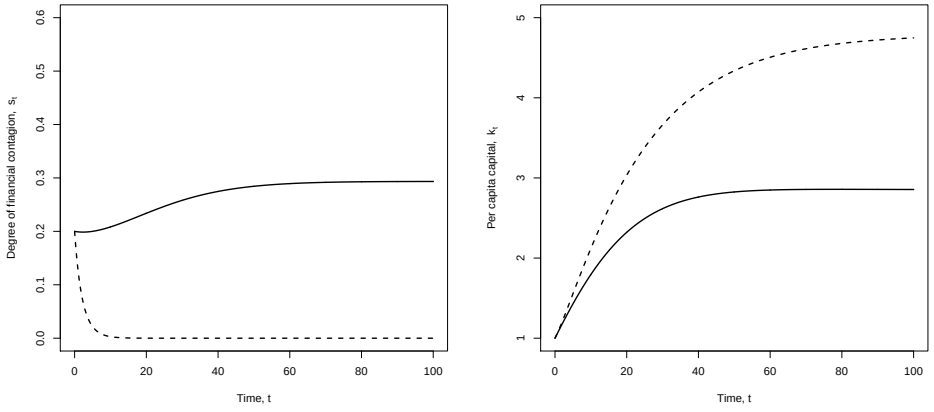

Figure 6: Evolution of the degree of financial contagion (left panel) and per capita capital (right panel) over time whenever $\theta(\frac{va}{\delta+n})^{\frac{\alpha}{\beta}} \leq d+\eta+b$ (dashed curves) and $\theta(\frac{va}{\delta+n})^{\frac{\alpha}{\beta}} > d+\eta+b$ (solid curves), starting from the same initial conditions, $i_0 = 0.2$ and $k_0 = 1$. Parameter values: $\alpha = 0.33$, $a = 1$, $v = 0.2$, $\delta = 0.05$, $n = 0.02$, $\theta = 0.2$, $\eta = 0.1$, $\beta = 1$, $b = 0.03$, $\epsilon = \frac{1-\alpha}{\beta}$, with $d = 0.08$ (dashed curves) or $d = 0.02$ (solid curves).

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