Pandemics of the Poor and Banking Stability*

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Abstract

We first develop a standard theoretical model that shows that the likelihood of a collapse of the banking industry of a developing country increases, as the joint prevalence of large pandemics such as AIDS and malaria increases. We also show that the optimal bank reserves increase as the prevalence increases. In the empirical part of the paper, we consider a large dataset of developing countries, and we exhibit a strong causality effect from combined prevalence to deposit turnover, as well as a strong causality effect from an increase of combined prevalence to an increase in bank reserves. This effect is strong for tuberculosis. Those empirical facts therefore strongly support our theoretical findings.

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*Thanks to be added later.

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1 Introduction

The spread of HIV/AIDS has been dramatic since the early 90s in many poor countries, and particularly in Sub-Saharan countries. For instance, the prevalence in the adult population in South Africa was about 20% in 2006 (UNAIDS [23]). Despite the impressive strain on public budget caused by the disease (up to 1.6 % of consolidated national expenditures in Nigeria in 2006 for instance, Hickey [10]), solely 15% of the infected population worldwide had access to public care in 2005 (Lampey et al. [13]). This HIV/AIDS pandemics often arose in countries with already high prevalence of other diseases such as malaria and tuberculosis (GFATM [7]). At the same time, large deposit withdrawals and relative banking fragility were observed in many of those developing countries (for instance for South Africa, see South African Savings Institute [22]). In this paper, we analyze the effects of the combined occurrence of those diseases on the banking stability of developing countries.

The potential causality effect from large-scale pandemics to banking stability is motivated by the fact that most of the treatments costs in developing countries are out-of-pocket health expenditures (Leoni [14, 15]). When experiencing significant morbidity symptoms requiring treatments, those patients may withdraw long-term bank investments (demand deposits for instance) to pay for their own care or that of their relatives. When withdrawals occur on a large national scale, because of high combined prevalence, bank reserves may be drained and long-term banking investments foregone. This implies that, as the prevalence in any of those diseases increases, the likelihood of a general banking collapse increases; this may occur even without runs, since the resulting withdrawal rate may exceed reserves, causing in turn a default from the banks. As a result, banks may have to increase reserves to alleviate the problems. We develop a theoretical support for those claims, and we support them with strong empirical evidence.

This paper has both a theoretical and an empirical part. We first develop a theoretical model, close to that of Diamond and Dybvig [5], which shows that the likelihood of a general collapse of the banking industry of a developing country increases, as the joint prevalence of those pandemics increases. We call *bank reserves* the ratio between liquid reserves and overall bank assets, which implies that our variable is always greater than mandatory reserves set by local regulators. When banks offer customers demand deposit contracts, as it is typically the case worldwide, we show that the optimal (equilibrium) bank reserves increase as the prevalence increases. In the empirical part of the paper, we consider a large dataset of developing countries, and we exhibit a negative causality between tuberculosis prevalence and out of pocket medical expenditures to banking deposits, banking turnover and private credit. Since we define banking turnover as credit over total assets, we thus obtain the same effect on banking reserves as predicted by the model. Those empirical facts therefore strongly support our theoretical findings.

We thus argue that the typical combination of those pandemics is a significant risk factor for the banking industry in developing countries with high prevalence, because of early withdrawals caused by the resulting morbidity. The fact that reserves run short in such circumstances sends negative signals to outside investors about the soundness of the banking industry, with the consequence of lower capital flows toward developing countries. The increase in bank reserves, and therefore the reduction of their long-term and most productive investments, also leads to an economic slowdown because of both credit and capital shortfall.

Several reasons at national level have been given so far to explain the already observed large turnover of deposits in developing countries. Those explanations are mostly associated with high unemployment and high inflation (see South African Savings Institute [22] for South Africa, and also Azemar and Debrides [2] for other potential determining factors). Leoni [15] was the first theoretical study to address

the causality effect from HIV/AIDS to banking stability, without addressing the adjustment of optimal bank reserves as a response to large pandemics, and without empirical support.

Other studies analyze the effects of large pandemics on the economic activities of developing countries. Haacker [8] gives a broad view of the macroeconomic effects of HIV/AIDS, whereas Johansson [12] studies more specifically the optimality of fiscal policies under high prevalence. Young [25] addresses the impact of high prevalence on growth; he argues that those pandemics have a detrimental impact on the accumulation of human capital, and that a large infection rate lowers fertility and increases the scarcity of labor. Young then concludes that this scarcity of labour is beneficial to survivors, even in reasonably large time horizon. In contrast, Santaeulalia-Llopis [20] argues that the resulting lack of accumulation of physical capital significantly delays the industrial transition that developing countries desperately need, with negative economic effects.

In a broader context, Ennis and Kiester [6] argues that bank runs are a significant deterrent to economic growth; their point is that their sole anticipation reduces capital accumulation and therefore future welfare. A corollary of our work leads to the same welfare conclusion, but it does not involve runs nor sunspots and the main explanation hinges on the increase in bank reserves leading to lower long-term investments.

In more details, we first develop a theoretical framework where standard deposit contracts and long-term investments are modelled as in Diamond and Dybvig [5]. The fraction of infected population is random, and it is unknown at the time the banks set the deposits rates and reserves. When an agent learns her infection, she needs to provide for her own care; if so, she may have to forfeit her long-term investments, should the cost of medications be too high as it is typically the case. There are two types of equilibria: either every agent anticipates a bank failure and thus does not deposit, or every agent deposits and a bank failure occurs with strictly positive

probability. This probability of failure is an increasing function, in the sense of first-order stochastic dominance, of the probability of infection. We also get the same corollary as in Leoni [15], where social welfare is greater in equilibria where agents deposit, despite the risk of banking failure. We also show that, as the likelihood of an infection increases in the sense of first order stochastic dominance, the optimal equilibrium banking reserves also increase.

We then test the model's predictions using annual data for 80 low income, lower middle income and upper middle income countries over the 1995-2009 period. Using system GMM, we estimate a series of structural VAR models measuring the impact of a shock in the incidence of tuberculosis and out of pocket medical expenditure on a set of banking stability variables. In line with the theoretical model, inspection of the model's impulse response functions suggests that a shock on the incidence of tuberculosis has a negative impact on banking deposits, financial system deposits, banking turnover and private credit; while out-of-pocket expenditures have a negative impact on turnover and private credit. Turning to the variance decomposition analysis, we find the effect of pandemics to be comparable to the impact of lagged constant per capita GDP, controlling for lagged financial variables.

The paper is organized as follows. In Section 2, we develop the model and carry out the theoretical analysis. In Section 3, we present the empirical part of the paper that strongly supports our theoretical findings. Section 4 contains some concluding remarks, and the technical proofs, figures and tables are given in the Appendix.

2 A model of banking collapse caused by large pandemics

We now formalize the fact that, in poor and emergent countries, an increase in out-of-pocket medical expenditures, combined with an increase in prevalence of one of the many diseases striking there, have a significant impact on banking stability. At theoretical model for now, we show that the combination of those two factors increases the deposit withdrawals levels, and it causes with strictly positive probability a banking failure independently of bank run. We also show that the optimal response of the banking industry to this phenomenon is to increase reserves, and therefore to reduce the most profitable long-term investments. We provide strong empirical support for those claims in the following sections.

2.1 The model

Our model is taken from Leoni [14] Ch. 4 and Leoni [15], and it is derived from that in Diamond and Dybvig [5]. For sake of simplicity, we will focus on the case where banks cannot suspend payments when too many individuals attempt to withdraw. A similar result holds in the other case, as pointed out in Leoni [14] Ch. 4.

The model has three periods ($T = 0, 1, 2$), and a single consumption good. In period 0, there is a continuum of non-infected individuals represented by the interval $[0, 1]$. Every individual is endowed with 1 unit of consumption good in period 0, which will be invested in various assets described later. There is also a continuum of banks, behaving competitively, in charge of long-term investments as described later as well.

In period 1, nature draws a type for every individual that corresponds to the occurrence or not of an infection caused by at least one disease (such as AIDS, tuber-

culosis). A type 1 individual is infected, and she learns her type in period 1. She will then have to finance her treatments $D > 0$, or else die in this period. In contrast, a type 2 individual is not infected. Every individual has the same probability of being infected in period 0.

In period 1, an individual of type 1 (resp. type 2) receives an endowment of $e_1 \geq 0$ (resp. $e_2 \geq 0$). Agents receive no endowment in period 2. The difference of endowments in period 1 may be explained by wage differential caused by drop in productivity because of an infection.

A fraction $t \in [0, 1]$ of the individuals becomes infected by at least one disease. The random number t is drawn in period 1 according to a probability distribution f over $[0, 1]$, which is continuous with $f(1) = 0$. The associated cumulative distribution F satisfies $F(t) < 1$, for all $t \in [0, 1)$; this last assumption captures the idea that a high fraction of infected people is relatively unlikely even if possible.

The individuals can use their period 0 initial endowment in three different manners. First, every individual can store consumption good in every period, in order to consume it in the next period. The storage is costless, and provides no return to the individuals. Second, the individuals have access to a competitive complete market for claims on future goods, which is open in every period.

Finally, individuals can deposit part of their endowment in a bank. With the deposit from the individuals, the bank uses a long-term investment with constant returns to scale. Formally, one unit of consumption good invested by the bank in period 0 yields $R > 1$ units of consumption good in period 2. If the investment is withdrawn in period 1, the salvage value to the bank will exactly be the value of the investment. In the financial market, it can be shown, as in Diamond and Dybvig [5], that the period 0 price of period 1 consumption is 1, and the period 0 price of period 2 consumption is R^{-1} to avoid arbitrage.

As in Leoni [15], the banks offer *demand deposit contracts* to their depositors. This

contract specifies a fixed claim of $r_1 \geq 0$ per unit deposited to customers withdrawing in period 1. The banks are mutually owned, and therefore period 2 withdrawers will equally share the face value of the remaining assets. Those contracts are the most common in practice, thus the focus here.

The banks also satisfy a *sequential service constraint*, where individuals withdrawing in period 1 are paid back in a first-come first-serve manner until the bank reserves in this period are exhausted. This assumption is essential since it allows for bank runs as an equilibrium. Formally, denote by A the total amount of deposits in period 0, and consider an individual $j \in [0,1]$ willing to withdraw in period 1. Let f_j denote the fraction of period 1 withdrawers arriving at the bank before individual j, and let V_1 denote the period 1 payoff per unit deposit to this individual j. When offering a rate $r_1 > 0$ to customers, the bank has in reserve $r_1^{-1}A$. The payoff V_1 per unit deposited, and withdrawn in period 1, is therefore $V_1(f_j, r_1) = r_1$ if $f_j < r_1^{-1}A$, and 0 otherwise.

Let now V_2 denote the period 2 payoff per unit deposit not withdrawn in period 1, and let f be the number of demand deposits withdrawn in period 1. There are 2 possible cases: the withdrawn funds have exceeded the bank reserves and the bank is bankrupt, otherwise the period 2 claimants share the profits. We thus have that $V_2(f, r_1) = \max\left[\frac{R}{1-f}(A - r_1 f), 0\right]$.

We next describe the payoff function of the agents. Let c_1 (resp. c_2) denote individual consumption of an individual in period 1 (resp. period 2), and let θ be the type of the individual. The utility derived from the consumption of the bundle (c_1, c_2), as a function of her type, is

$$U(c_1, c_2, \theta) = u(c_1 + c_2) \text{ for every } \theta, \tag{1}$$

where $u : \Re_+ \to \Re$ is twice continuously differentiable, strictly increasing, strictly concave, and satisfies the Inada conditions $u'(0) = \infty$, and $u'(\infty) = 0$. Similarly to

Diamond and Dybvig [5], solely for technical reasons and without a significant loss of generality, we assume that $-cu''(c)/u'(c) > 1$ for $c \geq 1$. We also normalize units so that $u(0) = 0$. Every individual is assumed to maximize the ex-ante expected utility $E[U(c_1, c_2, \theta)]$, where the expectation is taken according to F.

Consider now an infected individual, whose endowment e_1 in period 1 does not cover for her medical expenditures. This assumption is reasonable in practice, as pointed out in Leoni [14] Ch. 8. Assuming that she deposited in period 0, and was offered a rate $r_1 > 1$, she must then withdraw in period 1. She then gets an overall payoff $u(max[e_1 + V_1 - D, 0])$, where V_1 is defined above and it encompasses her position in the waiting line. The payoff to a type 2 individual, waiting until the last period to withdraw, is then $u(e_2 + V_2)$. The above remarks shows that, as long as $r_1 > 1$ and the out-of-pocket medical expenditures are high enough, every agent finds it optimal to deposit provided that no bank run is anticipated. If a bank run is anticipated in period 0, investment in the financial market is the only alternative.

2.2 Equilibrium concept

A strategy for the banks is the choice of $r \geq 0$, so as to maximize the ex-ante social welfare. This behavior is a standard argument caused by the competitiveness of the banking industry. Moreover, risk aversion will ensure that the optimal period 1 rate r is in $[1, R]$.

A strategy for an individual is to choose whether to deposit all of its endowment at a bank, for all possible interest rates the banks offer. We can easily rule out the possibility of partial deposit, by arbitrage considerations on the financial market. Also, for every possible interest rate and every deposit choice, an individual will choose either to withdraw in the first period or to wait, conditional on her type and others' strategies. Formally, an individual chooses (d, w), where d is a function from $[1, r]$ into $\{0, 1\}$ representing the decision to deposit, and w is a function from

$[1, R] \times \{0, 1\} \times \Theta$ into $\{0, 1\}$ representing the decision of withdrawing in period 1. The notation $d(r) = 1$ stands for the choice of depositing, and $w(r, d, \Theta) = 1$ means that she will withdraw in period 1.

We define the notion of banking reserve, central to our analysis.

Definition 1 *For any given deposit rate $r \geq 1$ offered by the bank, the banking reserve is given by $1/r$.*

The reserve represents the amount of liquid assets the bank has available for withdrawal in period 1. This amount therefore not invested in the long-term asset to hedge against the risk of larger than expected withdrawals.

Definition 2 *A bank failure occurs when the fraction of funds withdrawn in period 1 exceeds the bank reserve in this period.*

It is essential to make the distinction between bank run and bank failure as above. A bank failure occurs when are fully exhausted, and it may occur without mass panic or bank run. Every bank run will trigger a bank failure, but the converse is not true in general as shown later.

We next define our notion of equilibrium, to analyze deposit decisions and rate level.

Definition 3 *A (symmetric Nash) equilibrium is then r^* for the bank, and a common $(d^*, w^*$ for every individual such that $w^*(r, d, \Theta)$ is optimal for all (r, d, Θ), $d^*(r)$ is optimal for all r, and r^* is optimal taking as given individuals' strategies.*

The equilibrium above is symmetric since every agent shares the same strategy; this assumption can be easily justified since agents are all identical as to period 0. We refine this notion in order to encompass the possibility of bank runs, which is independent of bank failure as noted earlier. An *autarkic equilibrium* is an equilibrium

as above such that no individual decides to deposit in period 0. Such equilibria are typical of anticipated bank runs, where individuals take the precautionary measures of avoiding banking operations because of their expectations regarding a run.

2.3 Equilibrium behavior

We next characterize the symmetric equilibria of this game when the out-of-pocket medical costs increase, and we study their properties. We first show that such equilibria can be of two distinct types: either individuals avoid depositing (an autarkic equilibrium resulting from the anticipation of a bank failure), or they all deposit and non-infected individuals wait until the last period. For sake of simplicity, we carry out the analysis with the assumption that every fraction of infected individuals can be drawn by nature with strictly positive probability. This assumption can be relaxed in order to get the same result (see Leoni [15] for the details, and a closely related result).

Proposition 4 *Assume that $D > e_1 + 1$, then*

1. *an autarkic equilibrium exists, and*

2. *there exists another equilibrium where every individual deposits, every infected individual withdraws in period 1, every non-infected individual waits until period 2, and a bank failure occurs with strictly positive probability.*

Proposition 4 shows that, when out-of-pocket medical expenditures are high enough, either every agent anticipate a bank run and thus does not deposit, or otherwise every agent deposits and a bank failure may occur. Failure in this case is independent of any bank run, but stem from an unusually high withdrawal level because of the diseases. Moreover, this result shows that infected agents will withdraw and therefore

bank reserves increase because of the infection rate. Next section, we show that this result has strong empirical support.

The following result makes the link between optimal bank reserves and infection level.

Proposition 5 *Assume that $D > e_1 + 1$, and consider two distinct cumulative probability distribution F_1 and F_2 such that F_1 first-order stochastic dominates F_2. Consider also the (non-autarkic) equilibrium banking reserve $1/r_1^*$ (resp. $1/r_2^*$) associated with F_1 (resp. f_2) in every non-autarkic equilibrium. We have that $1/r_1^* \geq 1/r_2^*$.*

Proposition 5 shows that, when a bank run is not anticipated and thus individuals deposit, the equilibrium banking reserve increases as the likelihood of greater large-scale infection increases. This result is interesting because banking reserves are chosen so as to maximize social welfare. Therefore, it becomes optimal for banks to increase their reserves as the pandemics spread, which implies to forego long-term investments because of higher prevalence. This result has very strong empirical support, as shown in the empirical part of the paper. Both Proposition 4 and Proposition 5 will be proven in the Appendix.

The following result is a direct consequence of the proof of Proposition 4, and of Proposition 5.

Corollary 6 *Assume that $D > e_1 + 1$, and consider two distinct cumulative probability distribution F_1 and F_2 such that F_1 first-order stochastic dominates F_2. Consider also the (non-autarkic) corresponding equilibrium probabilities of bank failure P_1 and P_2. We have that $P_1 \geq P_2$.*

Corollary 6 shows that, as the combined prevalence increases, the greater the instability of the banking system.

3 Empirical validation

We now describe the dataset that we use to validate our theory, and our econometric analysis. We focus on tuberculosis, as the joint effect of all other diseases provide similar results.

3.1 Data

Our dataset is taken from the World Development Indicators database. Variables are selected with a view to maximize degrees of freedom. The selected banking sector variables are bank reserves defined as the ratio of banking credit to banking deposits (Turnover); the banking deposits to GDP ratio (Deposits); the financial system deposits to GDP ratio (System Deposits); and the private credit to GDP ratio (Private Credit). Health-related variables are the incidence of Tuberculosis per 100,000 people (Tuberculosis) and out of pocket health expenditure as a percentage of total health expenditure (Outpocket). We use constant per capita GDP as a control variable. We divide the sample by banking sector variable and develop four strongly balanced panels for the years 1995 to 2009 for all countries in the dataset, excluding high income countries. All variables are demeaned over the cross-sectional dimension prior to econometric investigation. A summary of the dataset is shown in Table 1.

INSERT TABLE 1 ABOUT HERE

3.2 Methodology

We adopt a panel structural VAR modeling approach to model the impact of an increase in out of pocket health expenditure and the incidence of tuberculosis on banking system deposits, private credit, bank reserves and financial system deposits.

Our approach can be outlined as follows. Consider the following structural VAR model:

$$\begin{cases} X_{it} &= A^*(L)X_{i,t-j} + \Gamma^{-1}\varepsilon_{i,t} \\ \varepsilon_{i,t} &= \nu_i + \nu_t + \nu_{it}, \end{cases} \tag{2}$$

where X_t is a vector of stationary variables, L is the lag operator and $A^*(L)$ is a transformed matrix of coefficients such as $A^*(L) = \Gamma^{-1}A(L)$, where Γ is the matrix of contemporaneous parameters and $A(L)$ the initial matrix of VAR parameters. Fixed time and individual effects (ν_i and ν_t) are included in the model to accommodate for unobserved individual heterogeneity. Errors ν_{it} have zero mean. The variance-covariance matrix of reduced form shocks $E(\nu_{it}, \nu'_{i,t}) = \Omega$ is real, symmetrical and positive definite. To derive impulse response from the model, this matrix is rewritten as:

$$\Omega = KDK'$$

where D is a diagonal matrix and K a lower triangular matrix. Letting $\mu_t = K^{-1}\nu_t$ be a vector of orthogonal residuals with

$$E(\mu_{it}\mu'_{it}) = E\left(K^{-1}\nu_{it}\nu'_{it}(K^{-1})'\right) = K^{-1}(KDK')(K^{-1})' = D,$$

the moving average representation is:

$$X_{it} = \sum_{h\geq 0} \phi_h K K^{-1}\nu_{i,t-h} = \sum_{i\geq 0} \Phi_h \mu_{i,t-h}, \tag{3}$$

with $\Phi_h = \phi_h K$ for every h.

Since the moving average form is obtained by inverting the VAR model, elements of Φ_h are a function of the initial VAR parameters. Orthogonal responses to shocks of X_{it} to innovations μ_{js} $(s \leq t)$ can be derived via the dynamic multiplicators

$$\frac{\partial X_{it}}{\partial \mu_{is}} = \Phi_{t-s}.$$

Structural error response functions are then $\Phi_{ij,h}$ for every $h \geq 0$. Error margins are computed by bootstrap with a 68% confidence interval (Sims and Zha, 1999). We use a Generalized Impulse Response Functions and Generalized Variance Decomposition framework in order to eliminate the compositional effects of the Choleski decomposition. The presence of lagged endogenous variable and individual fixed effects biases OLS and Within-Group estimator.[1] Parameters of the SVAR model are thus estimated via system GMM. We apply a Helmert transformation to our dataset by computing weighted deviations from forward means:

$$\begin{cases} X_{it}^{*} & = c_t \left[X_{it} - \frac{X_{i,t+1}+...+X_{iT}}{T-t} \right] \\ c_t & = \left(\frac{T-t}{T-t+1} \right)^{1/2}, \end{cases}$$

One key feature of this transformation is that real weights c_t preserve the variance of the dataset. In addition, the absence of serial correlation of error terms is preserved but transformed error terms become orthogonal to the untransformed variables. The latter are hence used as instruments (Arellano and Bover, 1995) in a system GMM framework.

3.3 Econometric investigation

The short time dimension poses a challenge for the analysis of our sample's time series properties. Most first and second generation panel unit root tests typically make large T, large N asymptotic distribution assumptions (e.g. Im et al., 2003; Maddala and Wu, 1999; Bai and Ng, 2004; Pesaran, 2003). However, Harris and Tzavalis (1999) showed that basing a unit root test on asymptotic T assumptions

[1] Nickell (1981) showed that this bias goes in the opposite direction on the relationship between exogenous variables and the lagged mean-differenced dependent variable. For instance, if an exogenous variable is negatively related to the lagged, mean differenced dependent variable, its estimated parameter will be biased upwards.

when T is fixed leads to underestimating the variance of the standardized test statistic. This has two potential adverse effects on statistical inference. First, this shifts the distribution of the test to the left, creating the possibility of an oversize bias. Second, this draws in the tails of the distribution and thereby diminishes the test's empirical size. Using Monte Carlo simulations, Harris and Tzavalis (1999) showed that the variance effect tends to dominate the mean-shift effect when fixed effects are included in the regression, resulting in a reduction in the size of the test and a significant decrease in power to reject the null hypothesis for values of $T < 50$. To correct for this bias, they derived an adjusted test statistic depending on the estimated parameters of the lagged endogenous variable and the known values of N and T. This test assumes a homogeneous within estimator across panels, which implies that the null hypothesis of a unit root needs to be rejected for all panels in order to conclude to stationarity. The results from the unit root test are shown in table 2 and point out that all variables are not stationarity in levels, but they are all stationary in first-difference.

INSERT TABLE 2 ABOUT HERE

Turning to cointegration tests, most residual-based cointegration tests require that the long-run parameters for the variables in levels be equal to the short run parameters for the first-differenced variable. This 'common factor' restriction causes over rejection of the no-cointegration null (Banerjee, Dolado and Mestre, 1998). To overcome this issue, Westerlund (2007) developed four new cointegration tests based on structural dynamics. Consider the following panel error-correction representation:

$$\Delta x_{it} = \alpha_i \left(x_{i,t-1} - \beta' y_{i,t-1} \right) + \sum_{j=1}^{p_i} \alpha_{ij} \Delta x_{i,t-1} + \sum_{j=-q_i}^{p_i} \gamma_{ij} \Delta x_{i,t-1} + e_{it} \qquad (4)$$

where the time-series and cross-sectional units are $t = 1, ..., T$ and $i = 1, ..., N$, respectively. The null hypothesis of no cointegration is stated as $H_0 = \alpha_i = 0$ for all i. Two pairs of test statistics are then derived for the alternative. The first pair

of tests (group-mean tests) $(G_\tau; G_\alpha)$ test H_0 against the alternative $H_1^g : \alpha < 0$ for at least one i. The second pair of tests (panel tests) $(P_\tau; P_\alpha)$ test H_0 against the alternative $H_1^p : \alpha < 0$ for all i. These tests are normally distributed . As shown in Table 3, for all models considered, the null hypothesis of no cointegration cannot be rejected by all the four tests (See Persyn and Westerlund (2008) for details on the tests computation and empirical properties). Therefore, the empirical properties of the variables examined imply that estimating the VAR in first differences since there exist no cointegration relationships between the variable. The models are estimated with four lags to fully capture the system's dynamics, results being robust to different lag intervals (we estimated the models with 1 to 4 lags).

INSERT TABLE 3 ABOUT HERE

Impulse responses functions are shown in Figure 1. We only report the impulse of interest for space-saving consideration (other impulse responses estimated from the models are available upon request). Inspection of the figure shows that a one standard deviation shock on the incidence of tuberculosis has a negative impact on banking deposits, financial system deposits, banking turnover and private credit; while a one standard deviation shock on out-of-pocket expenditures has a negative impact on turnover and private credit. Not surprisingly, for each model more than 95% of the forecast error variance of financial variables is driven by the lagged values. Figure 2 thus focuses the proportion driven by the health shock as compared to the GDP per capita control variable. We find that the effect of tuberculosis amounts to approximately one third of the effect of GDP per capita on bank reserves, banking deposits, financial system deposits and private credit. Turning to out of pocket expenditure, the effect ranges from being insignificant for banking deposits, financial system deposits and private credit, to about a half of the effect of GDP per capita in the case of bank reserves. Overall, these results give empirical support to the model's predictions.

INSERT FIGURE 1 ABOUT HERE

INSERT FIGURE 2 ABOUT HERE

4 Conclusion

We argue that the combined prevalence of various pandemics of the poor significantly affect the banking industry of developing countries. At theoretical level, we show that an increase in the likelihood of prevalence in the population triggers a greater risk of general banking failure, and that it forces banks to increase their reserves.

We then test the model's predictions by running a set of structural panel VAR models on a panel of 80 low income, lower middle income and upper middle income countries over the 1995-2009 period. Inspection of impulse response functions and variance decomposition analysis highlighted that increases in the prevalence of tuberculosis and related diseases, and in the rate of out of pocket medical expenditure, have the expected impact on banking deposits, financial system deposits, private credit, and above all banking reserves.

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A Appendix: Proofs of Theoretical Results

Before depositing, every individual must estimate the probability of arriving at the bank before it fails, conditional on an anticipated failure or run. Conditional on the fraction t of infected individuals in the population, this probability depends on the interest rate r offered by the bank, on the fraction s at which the bank suspends withdrawals (if allowed), and on the strategies chosen by the other individuals.

Since we focus on symmetric strategies, it is enough for an individual to estimate this probability 1) when all individuals withdraw in period 1, 2) when infected individuals only withdraw in period 1, and 3) when she alone withdraws in period 1 in addition to all of the infected individuals — these functions will be denoted by $\alpha_a(r,s|t)$, $\alpha_i(r,s|t)$ and $\alpha_1(r,s|t)$, respectively. We assume that for all $y = a, i, 1$, the function α_y is continuous with respect to t, is differentiable with respect to r and s with bounded partial derivatives, decreasing in s, and that

$$\int_0^1 \frac{\alpha_a(r|t)}{r} f(t)\mathrm{d}t = 1 \text{ for every } r.$$

The following proof closely follows that of Proposition 1 in Leoni [15]. To simplify matters, and without loss of generality, we normalize $e_1 = e_2 = 0$; in the same spirit, we set $D = 0$ to simplify matters.

A.1 Proof of Proposition 4

Since by assumption $D > e_1 + 1$, every infected individual that have deposited will withdraw in period 1 to pay for her medical needs. There are therefore four candidate strategies for a symmetric equilibrium:

1. No individual deposits, and non-infected individuals withdraw in period 1,

2. No individual deposits, and non-infected individuals withdraw in period 2,

3. Every individual deposits, and non-infected individuals withdraw in period 1,

4. Every individual deposits, and non-infected individuals withdraw in period 2.

It is easy to check that both Cases 1 and 2 above form an equilibrium for any possible interest rate offered by the bank – these two cases correspond to the autarkic equilibria in 4. Case 3 cannot be an equilibrium; this is true because any individual would prefer not to deposit in period 0 because bank failure is certain.

Thus, in order to prove Proposition 4, we need to show that Case 4 is an equilibrium. Assume now that the bank offers a rate $r \geq 1$, every individual deposits and non-infected individuals wait for the second period to withdraw. The expected utility for a non-infected individual is given by $\int_{[0,1]} u\left(\max\left\{R\frac{1-tr}{1-t}, 0\right\}\right) f(t)\mathrm{d}t$. If one non-infected individual decides to withdraw in period 1 when all of the other non-infected individuals withdraw in period 2, then her expected utility is given by $\int_{[0,1]} u(r)\alpha_1(r|t)f(t)\mathrm{d}t$.

Consider the set Ω of all rates that make worthwhile waiting until period 2 for a non-infected agent, in absence of run. This set is defined as

$$\Omega = \left\{ \; r \in [1, R] : \int_{[0,1]} u\left(\max\left\{R\tfrac{1-tr}{1-t}, 0\right\}\right) f(t)\mathrm{d}t \geq \int_{[0,1]} u(r)\alpha_1(r|t)f(t)\mathrm{d}t \; \right\}.$$

As long as $r \in \Omega$, Case 4 is a candidate for an equilibrium because there is no incentive for unilateral deviation for non-infected agents. From the above, a non-infected individual then receives $u\left(\max\left\{\Omega\frac{1-tr}{1-t}, 0\right\}\right)$, and an infected individual receives a utility of $\alpha_i(r|t)u(r)$, when the fraction of infected individuals is t. Thus, the ex-ante expected utility of any individual in this type of equilibrium is

$$U(r) := \int_{[0,1]} \left[t\alpha_i(r|t)u(r) + (1-t)u\left(\max\left\{R\frac{1-tr}{1-t}, 0\right\}\right)\right] f(t)\mathrm{d}t.$$

If an individual decides not to deposit, then her ex-ante expected utility is simply $\int_0^1 [tu(1) + (1-t)u(R)]\, f(t)\mathrm{d}t$.

Denote by $\Xi = \left\{ r \in [1, R] : U(r) \geq \int_0^1 [tu(1) + (1-t)u(R)]\, f(t)\mathrm{d}t \right\}$ the set of admissible deposit rate that makes deposit superior to autarky for potentially infected agent. The above shows that any individual will choose to deposit provided that r belongs to Ξ.

For Case 4 to be an equilibrium, it must be true that $\Xi \cap \Omega \neq \emptyset$. To be compatible with the maximizing behavior of the maximizing behavior, any r^* that maximizes U in the set $\Xi \cap \Omega$ must be an equilibrium strategy. The following lemma shows that such a r^* exists.

Lemma. *The function U has a maximizer in $\Xi \cap \Omega$.*

Proof. Note that the set $\Xi \cap \Omega$ is compact, and non-empty, since $r = 1$ belongs to $\Xi \cap \Omega$. The function U is a continuous function of r. Hence, there exists r^* that maximizes U in $\Xi \cap \Omega$. The proof is now complete.

We have shown so far that there are two types of equilibria: in one type nobody deposits, and in the other type everyone deposits, and non-infected individuals withdraw in period 2. In order to complete the proof of Proposition 4, we are left to show that in every equilibrium of the second type, we have $r^* > 1$ so that reserves may become exhausted for large enough withdrawals. Notice also that $r^* > 1$ implies a strictly positive probability of a bank failure, since the probability of a bank failure is exactly the probability that the fraction of individuals withdrawing in period 1 is at least $1/r^*$; this probability is simply given by $1 - F(1/r^*) > 0$.

Lemma. *In every symmetric equilibrium such that every individual deposits, and all non-infected individuals wait, we have $r^* > 1$.*

Proof. It suffices to show that there exists $\tilde{r} > 1$ such that $\tilde{r}$ belongs to $\Xi \cap \Omega$, and $U(\tilde{r}) > U(1)$, which in turn implies that $r^* > 1$ since social welfare increases in the deposit rate.

Let $\Omega L(r)$ (resp. $\Omega R(r)$) denote the left-hand (resp. right-hand) side of the inequality defining the set Ω. Since $\Omega L(1) = u(R) > u(1) = \Omega R(1)$, we conclude that there exists a ball B around 1 contained in Ω.

Note that $\Xi = \{r \in [1, R] : U(r) \geq U(1)\}$. Therefore, to prove existence of $\tilde{r} > 1$ such that $\tilde{r} \in \Xi \cap \Omega$, and $U(\tilde{r}) > U(1)$, it is enough to show that

$$\lim_{r \searrow 1} \frac{U(r) - U(1)}{r - 1} > 0. \qquad (*)$$

This is true, because whenever (*) holds, it cannot be the case that $\frac{U(r)-U(1)}{r-1} \leq 0$ for all $r > 1$ in the ball B around 1. Thus, there exist $\tilde{r} > 1$ in $B \subseteq \Omega$ such that $\frac{U(\tilde{r})-U(1)}{r-1} > 0$. This, in turn, implies that $U(\tilde{r}) > U(1)$, and $\tilde{r} \in \Xi$.

Let $g(r) = R(1 - tr)/(1 - t)$. We have that

$$\begin{aligned} & \frac{U(r) - U(1)}{r - 1} \\ = & \int_0^{1/r} \left[t \frac{u(r) - u(1)}{r - 1} + (1 - t) \frac{u \circ g(r) - u \circ g(1)}{r - 1} \right] f(t) \mathrm{d}t \\ + & \frac{1}{r - 1} \int_{1/r}^1 [t(\alpha_i(r|t)u(r) - u(1)) - (1 - t)u(R)] f(t) \mathrm{d}t. \end{aligned}$$

Since the following holds

$$\begin{aligned} & \frac{1}{r - 1} \int_{1/r}^1 [t(\alpha_i(r|t)u(r) - u(1)) - (1 - t)u(R)] f(t) \mathrm{d}t \\ \geq & -u(R) \frac{1}{r - 1} \int_{1/r}^1 f(t) \mathrm{d}t \\ = & -u(R) \frac{F(1) - F(1/r)}{r - 1} \to -f(1)u(R) = 0. \end{aligned}$$

We still need to prove that

$$\lim_{r \searrow 1} \int_0^{1/r} \left[t \frac{u(r) - u(1)}{r - 1} + (1 - t) \frac{u \circ g(r) - u \circ g(1)}{r - 1} \right] f(t) \mathrm{d}t > 0.$$

Note that $g'(r)u'(g(r)) = -u'(R)Rt/(1-t)$. Defining

$$h_r(t) = \begin{cases} \left[t\frac{u(r)-u(1)}{r-1} + (1-t)\frac{u\circ g(r)-u\circ g(1)}{r-1}\right] f(t) & \text{if } t \in [0, 1/r] \\ 0 & \text{otherwise.} \end{cases}$$

It is easy to check that $\lim_{r\to 1} h_r(t) = [u'(1) - Ru'(R)]tf(t)$. Thus, by Lebesgue Dominated Convergence Theorem, we have that

$$\begin{aligned} & \lim_{r\searrow 1} \int_0^{1/r} \left[t\frac{u(r)-u(1)}{r-1} + (1-t)\frac{u\circ g(r)-u\circ g(1)}{r-1}\right] f(t)\mathrm{d}t \\ = \quad & \lim_{r\searrow 1} \int_{[0,1]} h_r(t)\mathrm{d}t = [u'(1) - Ru'(R)] \int_{[0,1]} tf(t)\mathrm{d}t > 0, \end{aligned}$$

because $u'(1) > Ru'(R)$ (see Diamond and Dybvig [5], Footnote 2) and because $\int_{[0,1]} tf(t)\mathrm{d}t > 0$. The proof is now complete.

A.2 Proof of Proposition 5

With the same assumption as in the previous result, we know that equilibria where every agent deposits exists, and that the equilibrium deposit rates can be chosen among the maximizers of U in $\Xi \cap \Omega$. Moreover, the sets Ξ and Ω both depend on the underlying cumulative probability distribution for infection. Consider then two distributions F_1 and F_2, such that F_1 first-order stochastic dominates F_2. For $i = 1, 2$, denote by Ξ_i and Ω_i their corresponding sets. We next show that $\Xi_1 \cap \Omega_1 \subset \Xi_2 \cap \Omega_2$.

Since u is increasing, and that a linear combination of linear function is increasing, by definition of first-order stochastic dominance we have that $\Xi_1 \subset \Xi_2$ (see Mas-Collel et al. [17] p.197).

We next show that $\Omega_1 \subset \Omega_2$. Consider the function

$$\Gamma(r, t) = u\left(\max\left\{R\frac{1-tr}{1-t}, 0\right\}\right) - u(r)\alpha_1(r|t). \tag{5}$$

The function $\alpha_1(r|t)$ is assumed to be decreasing in t, and for every $r \geq 1$ the function $u\left(\max\left\{R\frac{1-tr}{1-t}, 0\right\}\right)$ is also decreasing. For every r such as $R\frac{1-tr}{1-t} \geq 0$, the function Γ

is therefore decreasing in t. By definition of first-order stochastic dominance, and by construction of the sets Ω, we thus have that $\int_{[0,1]} \Gamma(r,t)dF_2(t) \geq \int_{[0,1]} \Gamma(r,t)dF_1(t)$ for every $r \in \Omega_1 \cup \Omega_2$. This implies that $\Omega_1 \subset \Omega_2$, and in turn that $\Xi_1 \cap \Omega_1 \subset \Xi_2 \cap \Omega_2$.

Therefore, any equilibrium rate under F_1 belongs to $\Xi_2 \cap \Omega_2$. Since social welfare increases in the deposit rate, it follows that any equilibrium rate under F_2 is greater than that under F_1. This implies that equilibrium reserves under F_1 are greater than those under F_2, and the proof is now complete.

Table 1 Data description

Panel 1 : T=15; N=80	Obs	Mean	Std. Dev.	Min	Max
Turnover	1200	-.01107	.19298	-.83189	1.2393
GDP	1200	.09303	.20220	-.45182	.99493
Tuberculosis	1200	3.0917	60.240	-399	521
Outpocket	1200	-2.43e-08	5.0997	-22.0842	20.2731
Panel 2 : T=15;N=73	Obs	Mean	Std. Dev.	Min	Max
Deposits	1095	.02155	.08491	-.24973	.44789
GDP	1095	.08942	.1910	-.37845	.70965
Tuberculosis	1095	.76420	47.463	-280.25	373.75
Outpocket	1095	4.49e-08	5.1716	-22.0842	20.2731
Panel 3 : T=15;N=73	Obs	Mean	Std. Dev.	Min	Max
System Deposits	1095	.02084	.0846	-.24730	.45659
GDP	1095	.08942	.1910	-.37845	.70965
Tuberculosis	1095	.76420	47.463	-280.25	373.75
Outpocket	1095	4.49e-08	5.1716	-22.084	20.2731
Panel 4 : T=15;N=73	Obs	Mean	Std. Dev.	Min	Max
Private Credit	1095	.01744	.09467	-.317472	.72143
GDP	1095	.08942	.19106	-.37845	.70965
Tuberculosis	1095	.76420	47.463	-280.25	373.75
Outpocket	1095	4.49e-08	5.1716	-22.0842	20.2731

Table 2 Harris-Tzavalis panel unit root test

	Levels	1st difference
Variable		
Constant per capita GDP	0.996	0.149***
Out of pocket health expenditure	0.789	-0.091***
Tuberculosis prevalence	0.959	0.399***
Private credit	1.040	0.567***
Banking deposits	1.009	0.430***
Financial system deposits	1.005	0.424***
Banking turnover	0.833	0.050***

Table 3 Panel cointegration tests

Model 1 (*outpatient, gdp,credit*)			
Statistic	Value	Z-value	P-value
Gτ	-1.578	4.242	1.000
Gα	-2.357	9.220	1.000
Pτ	-12.407	2.159	0.985
Pα	-2.905	4.523	1.000
Model 2 (*outpatient, gdp,deposits*)			
Statistic	Value	Z-value	P-value
Gτ	-1.560	4.406	1.000
Gα	-2.507	9.014	1.000
Pτ	-7.601	6.813	1.000
Pα	-2.742	4.772	1.000
Model 3 (*outpatient,gdp,system*)			
Statistic	Value	Z-value	P-value
Gτ	-1.553	4.472	1.000
Gα	-2.501	9.023	1.000
Pτ	-8.863	5.591	1.000
Pα	-2.918	4.504	1.000
Model 4 (*outpatient,gdp,turnover*)			
Statistic	Value	Z-value	P-value
Gτ	-1.483	5.361	1.000
Gα	-1.891	10.316	1.000
Pτ	-5.974	9.053	1.000
Pα	-1.490	6.998	1.000
Model 5 (*tuberculosis,gdp,credit*)			
Statistic	Value	Z-value	P-value
Gτ	-2.511	-4.623	0.000
Gα	-1.119	11.416	1.000
Pτ	-3.635	11.318	1.000
Pα	-0.846	8.029	1.000
Model 6 (*tuberculosis,gdp,deposits*)			
Statistic	Value	Z-value	P-value
Gτ	-1.480	5.153	1.000
Gα	-1.365	10.571	1.000
Pτ	-3.839	10.456	1.000
Pα	-0.908	7.575	1.000
Model 7 (*tuberculosis,gdp,system*)			
Statistic	Value	Z-value	P-value
Gτ	-1.494	5.017	1.000
Gα	-1.488	10.403	1.000
Pτ	-3.912	10.386	1.000
Pα	-0.864	7.642	1.000
Model 8(*tuberculosis,gdp,turnover*)			
Statistic	Value	Z-value	P-value
Gτ	-1.483	5.361	1.000
Gα	-1.891	10.316	1.000
Pτ	-5.974	9.053	1.000
Pα	-1.490	6.998	1.000

Figure 1 Impulse response functions

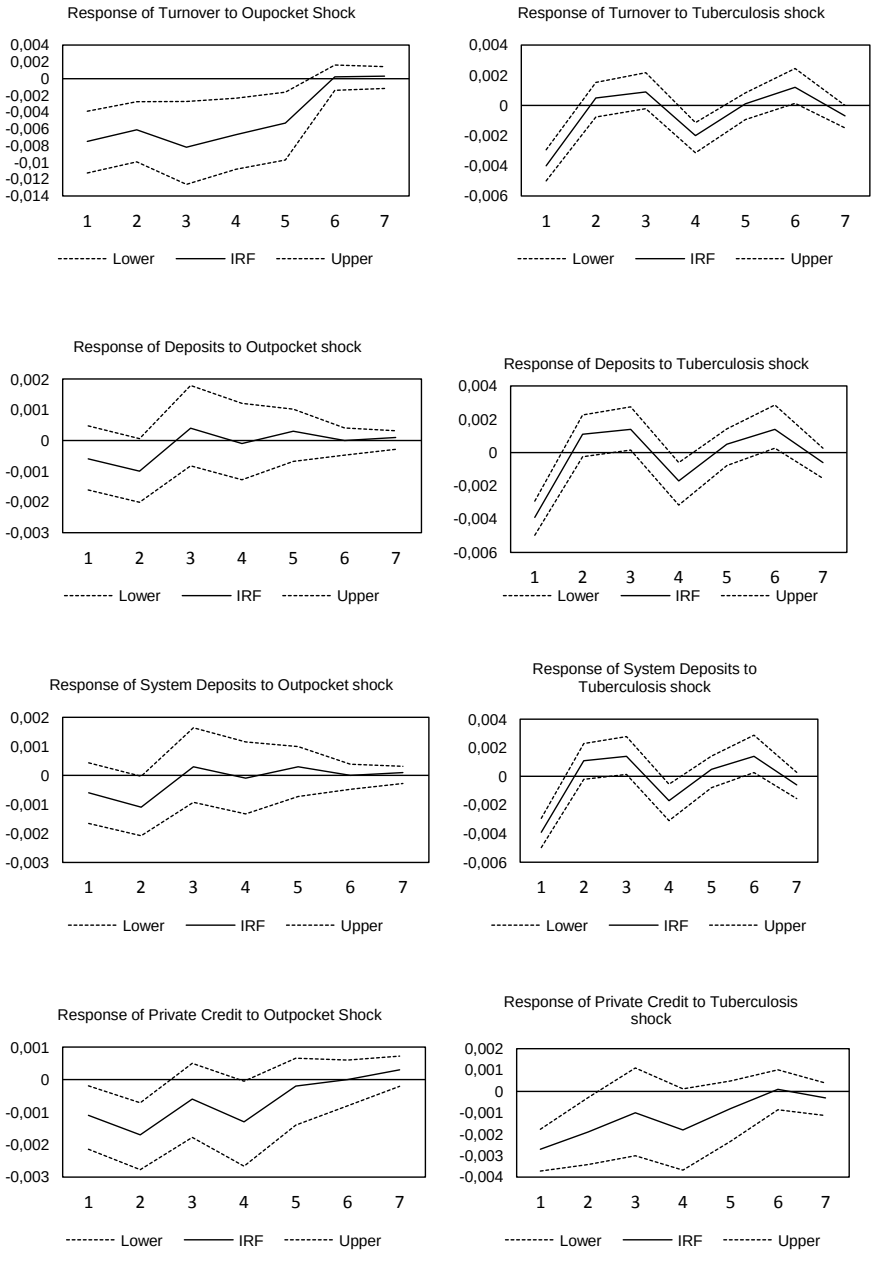

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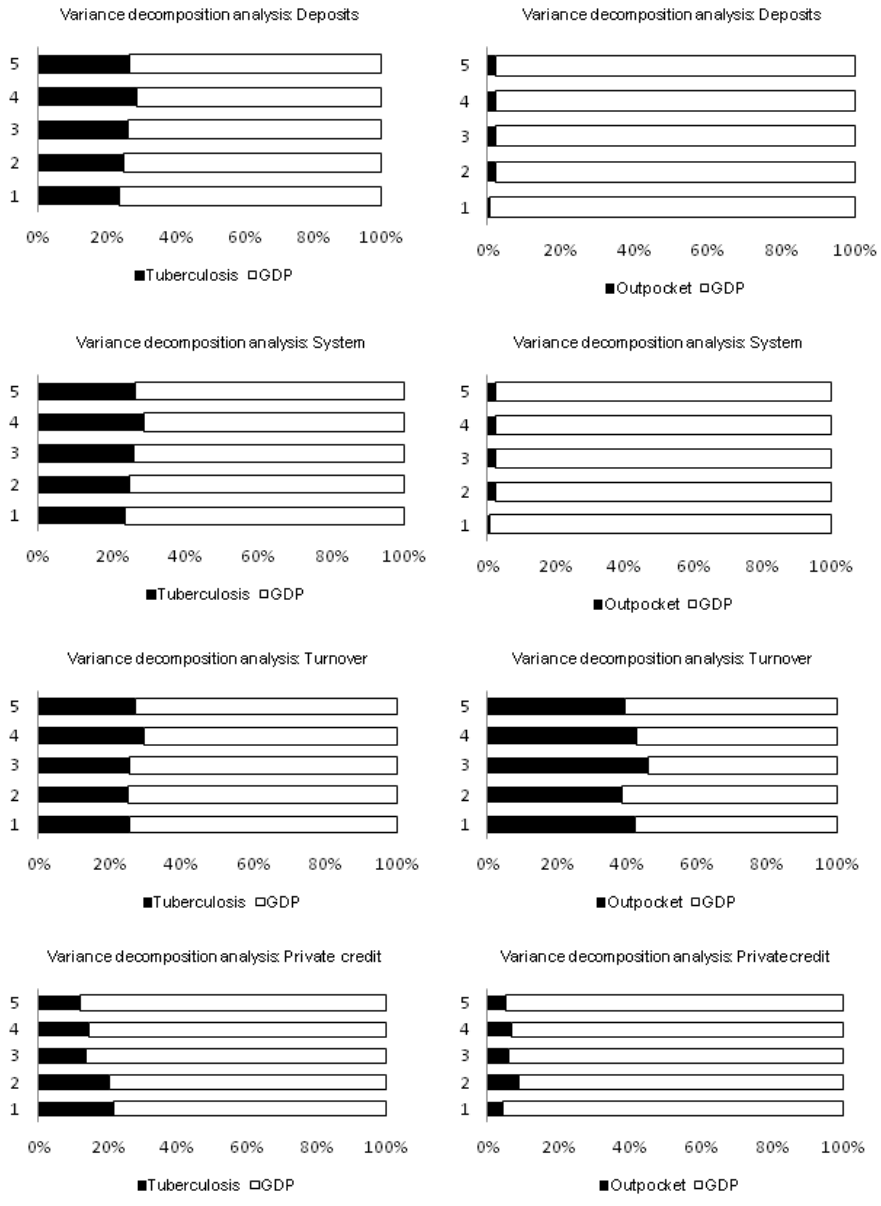
Figure 2 Variance decomposition analysis



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