Correlated Risks vs Contagion in Stochastic Transition Models

Patrick Gagliardini *

Christian Gouriéroux †

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*Swiss Finance Institute and University of Lugano.

†CREST, CEPREMAP and University of Toronto.

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Abstract

There is a growing literature on the possibility to identify correlation and contagion in qualitative risk analysis. Our paper considers this question by means of a model describing the joint dynamics of a set of individual binary processes. The two admissible values correspond to bad and good risk states of an individual. The risk correlation and its time dependence are captured by introducing a dynamic frailty, whereas the contagion passes through the effect of the lagged number of individuals in the bad risk state. We study carefully the dynamic properties of the joint dynamic process. Then, we focus on the limiting case of large populations (portfolios) and reconcile the microscopic and macroscopic dynamic views of the risk. The difficulty to identify in finite sample risk correlation and contagion is illustrated by means of Monte-Carlo simulations.

Keywords: Risk Dependence, Frailty, Systematic Risk, Contagion, Count Process, INAR Model, Compound Autoregressive Process, Affine Model, Credit Risk, Granularity Adjustment.
JEL classification: G12, C23.

1 Introduction

It has been recognized rather recently that large portfolios of individual risks, such as mortgages, credit default swaps, or life insurance contracts, cannot be fully diversified. This is due to the dependence between individual risks, which arises from exposure to common exogenous risk factors, called systematic risk (or frailty), but also from contagion phenomena. Let us discuss in this respect the literature for default risk [1], even if the literature on common risk factors and contagion is older [see Freund (1961), Oakes (1989)]. This literature distinguishes structural approaches, based on Merton's model for default risk [see e.g. Vasicek (1997), Hull, White (2001)], and reduced form approaches, which involve either copulas, or stochastic default intensities [2]. Similarly, we distinguish structural and reduced form modelling of contagion. The structural models will for instance consider the guarantees introduced in the credit contracts [Ebert, Luetkebohmert (2010), Gourieroux, Monfort (2010)], the structure of the debt of the borrowers [Rochet, Tirole (1996), Bolton, Scharfstein (1996), Bris, Welch (2005)], the liquidity shortages [Allen, Gale (2000)], or the counterparty risk [Jarrow, Yu (2001), Walker (2005), Egloff, Leippold, Vanini (2007), Brigo, Pallavicini (2007), Jorion, Zhang (2009)]. Such structural approaches require a detailed microeconomic knowledge of the debt structure. Since the interrelations between borrowers and lenders are complicated, this type of structural analysis can be applied to a small number of individual risks only. The reduced form models for contagion try to capture how the defaults of some individuals influence the default intensities of the individuals, who are still alive. They are either i) dynamic models written in continuous time, at the individual level [3] [see Azizpour, Giesecke (2008), a,b, Lando, Nielsen (2009)], based on the notion of mutually exciting point processes introduced by Hawkes (1971) a,b, Hawkes, Oakes (1974) or ii) based on the epidemic model introduced by Bailey (1953, 1957), Kendall (1956) [see the so-called infectious model used in a static framework by Davis, Lo (2001) a,b, Sakata, Isakado, Mori (2007), and its dynamic extension by Rulliere,

[1] Such a literature has also been developed for application in health insurance [see e.g. Gschlossl, Czado (2006)].

[2] See Li (2000), Schonbucher, Schubert (2002), Frey, McNeil (2003), Giesecke (2004) for reduced form models with copulas, and Duffie, Singleton (1999), Delloy, Fermanian, Shai (2005), Koopman, Lucas, Monteiro (2005), Das, Duffie, Kapadia, Saita (2007), Duffie, Eckner, Horel, Saita (2009) for stochastic intensity models.

[3] These models have to be distinguished from dynamic macroscopic models introduced to capture the volatility transmission [Gallo, Otranto (2007)], or the jump transmission [Ait-Sahalia, Cacho-Diaz, Laeven (2010)] between markets, even if they share common features with the microscopic models introduced for individual risks.

Dorobantu, Cousin (2010), for the application to credit risk]. However, it is still difficult to specify tractable models taking into account both systematic factor and contagion, and able to disentangle these two effects.

In our paper, we consider this problem for a transition model in discrete time, at a semi-aggregate level with respect to time and individual. Instead of following the dynamic of individual risks, we follow the dynamic of counts of individuals in the different classes of risk. Therefore, we introduce dynamic models for count processes, with both systematic factor and contagion. The analysis at a semi-aggregate level has two advantages. First, it is less demanding in terms of data confidentiality. Second, it allows for a modelling by means of affine processes, which are tractable for prediction purposes.

We consider in Section 2 an homogenous population of individuals with an endogenous dichotomous characteristic following a same homogenous Markov chain. We introduce the counting process defining the number of individuals in state 1 at each date, and show that this is a Markov process with a binomial autoregressive (BinAR) dynamics of order 1. For large population size, alternative limiting processes can be derived, depending whether we apply the Gaussian, or Poisson approximation of the binomial distribution. This leads to a Gaussian autoregressive [resp. Integer Autoregressive (INAR)] approximation. Section 3 explains how correlated risks and contagion can be introduced in a BinAR model, and in its two limiting counterparts. In Section 4 we present the results of some simulation experiments for a logistic model with frailty and contagion, and an INAR model with stochastic intensity. Section 5 concludes. Proofs are gathered in Appendices.

2 From the homogenous Markov chain to the Gaussian AR(1) and INAR (1) processes

2.1 Time-homogenous Markov chain

Let us denote by $(y_t, t \in \mathbb{N})$ a time-homogenous Markov chain with two states 0 and 1. The transition of this chain is characterized by the 2×2 matrix:

$$P = \begin{pmatrix} p_{00} & p_{01} \\ p_{10} & p_{11} \end{pmatrix}, \tag{2.1}$$

where $p_{ij} = P[y_t = i | y_{t-1} = j]$, for $i, j = 0, 1$. The transition probabilities are such that $p_{ij} \geq 0$, for any i, j, and $p_{0j} + p_{1j} = 1$, for $j = 0, 1$. The transition matrix admits the eigenvalues 1 and $\rho = p_{00} + p_{11} - 1$. Parameter ρ measures the within state stability of the chain. The stationary distribution of the chain $(1 - \mu, \mu)'$, say, is an eigenvector of transition matrix P associated with the unitary eigenvalue. Parameter μ is given by:

$$\mu = \frac{p_{10}}{p_{10} + p_{01}},$$

and is also equal to the probability to be in state 1 after a change of state.

The transition matrix can be equivalently written either in terms of the transition probabilities, or by means of the two parameters ρ and μ. Indeed, we have:

$$P = \begin{pmatrix} 1-\mu & 1-\mu \\ \mu & \mu \end{pmatrix} + \rho \begin{pmatrix} \mu & -(1-\mu) \\ -\mu & 1-\mu \end{pmatrix}. \tag{2.2}$$

The transition matrix of the chain at horizon h is:

$$P^h = \begin{pmatrix} 1-\mu & 1-\mu \\ \mu & \mu \end{pmatrix} + \rho^h \begin{pmatrix} \mu & -(1-\mu) \\ -\mu & 1-\mu \end{pmatrix}. \tag{2.3}$$

This highlights alternative interpretations of parameters μ and ρ, when $|\rho| < 1$. Parameter μ (or equivalently the stationary distribution) is a long run parameter since $\lim_{h\to\infty} P^h = \begin{pmatrix} 1-\mu & 1-\mu \\ \mu & \mu \end{pmatrix}$, whereas ρ provides the speed of adjustment towards this long run equilibrium. These parameters can be fixed independently: μ (resp. ρ) is constrained to be between 0 and 1 (resp. between -1 and 1).

2.2 The binomial autoregressive (BinAR) process

Let us now consider an homogenous population of individuals indexed by i, for $i = 1, \ldots, n$. We assume that the individual state histories $(y_{i,t})$, $i = 1, \ldots, n$ are independent time-homogeneous Markov chains with the same two-state transition matrix P. According to the type of application,

states 0 and 1 can have the following interpretations: low risk / high risk (for insured people), investment rating / speculative rating (for corporate bonds), holder of an insurance contract / not holder (for customers), ill/not ill (for individuals), low liquidity / high liquidity level [for banks, see e.g. Giesecke, Weber (2006)]. At each date t, we compute the count N_t of individuals in state 1 (resp. $n - N_t$ in state 0), and follow the structure of this homogenous population over time.

To analyse the transition distribution of process N_t, we can note that $N_t = N_{1t} + N_{0t}$, where N_{1t} (resp., N_{0t}) is the number of individuals in state 1 at date $t-1$ and staying in this state at t (resp., in state 0 at date $t-1$ and changing of state between $t-1$ and t). Conditional on past individual histories, variables N_{1t} and N_{0t} are independent, with conditional binomial distributions:

$$N_{1t} \sim \mathcal{B}(N_{t-1}, p_{11}), \quad N_{0t} \sim \mathcal{B}(n - N_{t-1}, p_{10}),$$

respectively. We deduce the proposition below.

Proposition 1: *Under the assumption of individual independent identically distributed Markov chains, the process (N_t) is a Markov process, with values on $\{0, 1, \ldots, n\}$ and transition distribution:*

$$\mathcal{B}(N_{t-1}, p_{11}) * \mathcal{B}(n - N_{t-1}, p_{10}),$$

where $*$ denotes the convolution operator.

In the probabilistic literature, this property is usually written by means of the binomial thinning operator [see Steutel, Van Harn (1979)], defined by

$$p \circ N = \sum_{i=1}^{N} u_i,$$

where u_i, for $i = 1, \ldots, n$, is a sequence of i.i.d. random variables admitting a Bernoulli distribution with parameter p, and $N \in \mathbb{N}$. With this notation, we have:

$$N_t = p_{11} \circ N_{t-1} + p_{10} \circ (n - N_{t-1}),$$

where the two components of the sum are independent conditional on N_{t-1}.

The conditional Laplace transform of count N_t is given by:

$$\begin{aligned}\psi_1(u) &= E_{t-1}[\exp(-uN_t)] \\ &= [1-p_{11}+p_{11}\exp(-u)]^{N_{t-1}}[1-p_{10}+p_{10}\exp(-u)]^{n-N_{t-1}} \\ &= \exp\left\{n\log[1-p_{10}+p_{10}\exp(-u)]+N_{t-1}\log\left[\frac{1-p_{11}+p_{11}\exp(-u)}{1-p_{10}+p_{10}\exp(-u)}\right]\right\}, \quad (2.4)\end{aligned}$$

where E_{t-1} denotes the conditional expectation given the past individual histories. It is defined for $u \geq 0$ and characterizes the transition of the nonnegative process (N_t) [see Feller (1968)]. The conditional Laplace transform is an exponential affine function of lagged count value N_{t-1}. Thus, process (N_t) is a compound autoregressive process of order 1 [CaR(1), see Darolles, Gourieroux, Jasiak (2005)]. [4]

The transition of process (N_t) at horizon h is $\mathcal{B}(N_{t-1}, p_{11}^{(h)}) * \mathcal{B}(n-N_{t-1}, p_{10}^{(h)})$, where $p_{ij}^{(h)}$ is the (i,j) element of matrix P^h. From equation (2.3), these elements are given by $p_{11}^{(h)} = \mu + \rho^h(1-\mu)$ and $p_{10}^{(h)} = \mu(1-\rho^h)$. In particular, the stationary distribution of (N_t) obtained for $h \to \infty$, is the binomial distribution $\mathcal{B}(n, \mu)$.

To summarize, count process (N_t) is such that the conditional distributions of both components N_{1t} and N_{0t} are binomial, and its unconditional (stationary) distribution is binomial too. This justifies the terminology binomial autoregressive [BinAR(1)] process of order 1. However, the transition of the BinAR(1) process is not binomial itself.

2.3 The limiting Gaussian AR(1) process

When the population size is large and the transition probabilities are fixed, the binomial distribution can be approximated by a Gaussian distribution. Equivalently, in terms of processes, the binomial autoregressive process can be approximated by a Gaussian process.

Proposition 2: *Let $n \to \infty$ and transition matrix P be fixed. Then, the process $X_{n,t} = \sqrt{n}(N_t/n - \mu)$, for $t \in \mathbb{N}$, converges in distribution to the Gaussian autoregressive process of order 1, denoted (ξ_t), such that:*

$$\xi_t = \rho\xi_{t-1} + \eta\varepsilon_t, \quad (2.5)$$

[4] The CaR processes are called affine processes in the continuous time literature, since the conditional log-Laplace transform is an affine function of the lagged value of the process [see e.g. Duffie, Filipovic, Schachermayer (2003)].

where $\varepsilon_t \sim IIN(0,1)$ *and* $\eta^2 = (1-\mu)p_{10}(1-p_{10}) + \mu p_{11}(1-p_{11}) = \mu(1-\mu)(1-\rho^2)$.

Proof: See Appendix 1.

The autoregressive coefficient is equal to the speed of adjustment of the BinAR(1) process. The innovation variance is such that the unconditional distribution of (ξ_t) is $N[0, \mu(1-\mu)]$.

2.4 The limiting INAR(1) process

The integer valued autoregressive process (INAR) has been initially introduced in Mc Kenzie (1985) and Al-Osh, Azaid (1987) [see also Mc Kenzie (1988) and Azaid, Al-Osh (1990)]. We derive it below as a limiting case of a binomial autoregressive process, when p_{01} is fixed, but p_{10} tends to zero when the population size n tends to infinity, such that $np_{10} \sim \lambda$, say, where $\lambda > 0$. Under these conditions, we get $p_{11} \sim \rho$ and $n\mu \sim \lambda/(1-\rho)$, and we can use the Poisson approximation of some binomial distributions. In particular, the stationary distribution of N_t is $\mathcal{P}[\lambda/(1-\rho)]$. It does not explode with n; thus, $n - N_{t-1}$ tends to infinity at speed n and the binomial distribution $\mathcal{B}(n - N_{t-1}, p_{10})$ is approximately Poisson $\mathcal{P}(\lambda)$, too. We deduce that the limiting process is the INAR(1) process defined below.

Proposition 3: *Let* $n \to \infty$ *and the transition probabilities be such that* $np_{01} \to \lambda$*, for* $\lambda > 0$*, and* p_{11} *is fixed. Then, the limiting process is an integer valued autoregressive process of order 1 [INAR(1)]. Conditionally on the past,* N_{1t} *and* N_{0t} *are independent with distributions* $N_{1t} \sim \mathcal{B}(N_{t-1}, \rho)$ *and* $N_{0t} \sim \mathcal{P}(\lambda)$*, respectively. The stationary distribution of* (N_t) *is* $\mathcal{P}[\lambda/(1-\rho)]$.

The conditional Laplace transform of count N_t at horizon 1 is:

$$\psi_1(u) = \exp\{-\lambda[1-\exp(-u)] + N_{t-1}\log[1-\rho+\rho\exp(-u)]\}. \tag{2.6}$$

The INAR(1) process is another example of CaR (affine) process of order 1 [Darolles, Gourieroux, Jasiak (2006)].

3 Correlated risks versus contagion

The basic models considered in Sections 2.2-2.4 assume a double homogeneity with respect to both individual and time. In this section, we still assume individual homogeneity, but consider time heterogenous Markov chains, i.e., chains with time dependent transition matrix P_t, say. We specify P_t in order to clearly disentangle correlated risks and contagion.

3.1 Modelling the correlation vs modelling the contagion

To illustrate the discussion below, let us interpret the states as not ill/ill. Then, p_{10} is the probability to get the disease for an individual currently in good health, whereas p_{01} is the probability to recover. We focus on a time dependent transition probability p_{10t} [5]. Thus, we get:

$$P_t = \begin{pmatrix} p_{00t} & p_{01} \\ p_{10t} & p_{11} \end{pmatrix}.$$

i) The dependence between individual risks to get the disease is generally introduced by considering a common stochastic intensity p_{10t}, or equivalently, by assuming that $p_{10t} = p_{10}(F_t)$, where F_t is an unobservable factor [6] [see e.g. Duffie, Singleton (1999), Schonbucher (2000), Delloy, Fermanian, Sbai (2005), Duffie, Eckner, Horel, Saita (2009) for credit risk applications]. This unobservable common factor is called common dynamic frailty in the credit risk literature by analogy with the terminology introduced by Vaupel, Manton, Stallard (1979) for application in demography [7].

[5] In other applications, such as low liquidity / high liquidity, both p_{10} and p_{01} can depend on time t [see e.g. Giesecke, Weber (2006)].

[6] For a homogenous population, any dependence structure can always be represented by means of such a factor representation [de Finetti (1931), Hewitt, Savage (1955)] with possibly an infinite dimensional factor. In particular, the copula based approach [see e.g. Li (2000), Schonbucher, Schubert (2002)] can be rewritten in this way.

[7] The terminology frailty has to be used carefully. In a general framework the transition probability $p_{10,i,t}$ can dependent on both individual i and time t, and different unobservable factors can be introduced, that are a pure individual effect F_i, a pure time effect F_t, or a joint unobservable effect F_{it}. The standard frailty terminology concerns models with independent individual effects F_i [see Greenwood, Yule (1920) for the first introduction of unobserved individual heterogeneity in the literature]. The aim is to account for omitted individual variables and explain the bias due to the fact that less fragile individuals will recover earlier. Joint individual and time effects have been introduced in the microeconometric literature to represent the effort to diminish risk by individuals and capture the moral hazard

ii) In a homogenous population the contagion effect explains how the number of ill people influences the probability to become sick for individuals currently in good health. This corresponds to a dependence of the type $p_{10t} = p_{10}(N_{t-1})$, where p_{10} is a deterministic function. This is a multivariate extension of Freund model for an homogenous population [Freund (1961)].

In the analysis of social effects, the literature distinguishes in a similar vein between endogenous effects, corresponding to contagion (also called peer effects, neighbourhood effects, herd behaviour, ...), and correlated effects, corresponding to the frailty [see Manski (1993)]. A dynamic binomial model with both risk correlation and contagion is defined by the following assumptions:

Assumption A.1: *The individual risk variables $y_{it}, i = 1, \ldots, n$ at date t are independent conditional on the past individual histories $\underline{y_{i,t-1}}, i = 1, \ldots, n$, and on the current and past factor values $\underline{F_t}$.*

Assumption A.2: *The conditional distribution of $y_{i,t}$ depends on individual histories by means of $y_{i,t-1}$ and N_{t-1} only, and on the factor path by means of F_t only. The corresponding transition matrix, conditional on $\underline{F_t}$ and $\underline{y_{i,t-1}}, i = 1, \ldots, n$, is:*

$$P_t = \begin{pmatrix} 1 - p_{10}(F_t, N_{t-1}) & p_{01} \\ \\ p_{10}(F_t, N_{t-1}) & p_{11} \end{pmatrix}.$$

Assumption A.3: *The conditional distribution of F_t given $\underline{F_{t-1}}, \underline{y_{i,t-1}}, i = 1, \ldots, n$, depends on F_{t-1} only, and admits the transition pdf $g(f_t|f_{t-1})$.*

Thus, the common factor has an exogenous Markovian dynamics and represents the external shocks influencing the probability to get the disease, such as environmental conditions, whereas contagion is an endogenous phenomenon.

In the model above, we have implicitly assumed that contagion arises with one lag. This recur-

phenomenon [see Gourieroux, Jasiak (2001) for an application to bonus-malus in motor insurance contracts]. In the framework above, similar to the standard one encountered in the credit risk literature, a stochastic time effect is introduced, that is, all individuals have at a given date the same degree of fragility. This explains the more precise terminology "common dynamic frailty" used in our framework [see also Duffie, Eckner, Horel, Saita (2009), p2096].

sive approach avoids the question of simultaneous contagion arising in discrete time models with $p_{10}(F_t, N_t)$ [see Manski (1993)] and in continuous time models [see e.g. Jarrow, Yu (2001)], as well as the associated identification problem, often called the reflection problem [Manksi (1993)]. We have also assumed that the influences of different sick people are the same. It would be possible to extend the model by assuming that contagion can arise with a limited number of neighbours [see e.g. Giesecke, Weber (2006)], or depend on individual characteristics of sick people.

The conditional distribution of N_t given $\underline{F_t}$, $\underline{N_{t-1}}$ becomes:

$$\mathcal{B}(N_{t-1}, p_{11}) * \mathcal{B}(n - N_{t-1}, p_{10}(F_t, N_{t-1})), \tag{3.1}$$

and the joint process (F_t, N_t) is Markovian. The conditional distribution of N_t given $\underline{N_{t-1}}$ only is derived by integrating out the unobservable factor path. More precisely, this conditional probability is given by:

$$P[N_t = n_t | \underline{N_{t-1}} = \underline{n_{t-1}}, F_0 = f_0] = \frac{\int \cdots \int \prod_{\tau=1}^{t} p[n_\tau | n_{\tau-1}, f_\tau] \prod_{\tau=1}^{t} [g(f_\tau | f_{\tau-1}) df_\tau]}{\int \cdots \int \prod_{\tau=1}^{t-1} p[n_\tau | n_{\tau-1}, f_\tau) \prod_{\tau=1}^{t-1} [g(f_\tau | f_{\tau-1}) df_\tau]}, \tag{3.2}$$

where $p(n_t | n_{t-1}, f_t)$ denotes the elementary probability of the conditional distribution (3.1). The factor integration creates a contemporaneous dependence between individual risks, but also an increase of the memory for count process (N_t), which is no longer Markovian.

Formula (3.2) shows that the effect of lagged counts $\underline{N_{t-1}}$ on the distribution of current count N_t has three origins: i) the dependence of N_t on N_{t-1} in the basic binomial autoregressive process; ii) the contagion effect, that is, the dependence of p_{10t} with respect to N_{t-1}; iii) the unobservability of the common dynamic frailty, which introduces the effect of $\underline{N_{t-1}}$ by means of the filtering distribution of F_t given $\underline{N_{t-1}}$. These different effects of $\underline{N_{t-1}}$ corresponding to the transition model, the contagion and the frailty filtering [8], respectively, can only be identified for special parameterized models.

Finally, in finite populations the affine property of process (N_t), and even of joint process (N_t, F_t), is not fulfilled in general. We describe below two limiting cases in which the affine property is (partially) recovered. They correspond to the limiting Gaussian AR(1) and INAR(1) processes, respectively.

[8] See e.g. Collin-Dufresne, Goldstein, Helwege (2003) for a discussion of the effect of the updating of beliefs.

3.2 The limiting models

The introduction of correlated risks and contagion in the limiting Gaussian autoregressive and INAR models is deduced by considering a large population and cross aggregating over the population. Thus, the limiting models explain how to pass from a microscopic analysis to a macroscopic one [Fournier, Meleard (2004)].

i) Limiting Gaussian autoregressive model

(*) Let us first consider a model with contagion effect only, that is, $p_{10,t} = p_{10}(N_{t-1}/n)$, say, where $p_{10}(.)$ is a given function. The conditional moments of N_t/n are:

$$\begin{aligned} E_{t-1}(N_t/n) &= p_{11}N_{t-1}/n + (1 - N_{t-1}/n)p_{10}(N_{t-1}/n), \\ V_{t-1}(N_t/n) &= \frac{1}{n}\left\{p_{11}(1-p_{11})N_{t-1}/n + p_{10}(N_{t-1}/n)[1-p_{10}(N_{t-1}/n)](1-N_{t-1}/n)\right\}. \end{aligned}$$

The conditional variance tends to 0, when n tends to infinity. This suggests that the variable N_t/n converges in quadratic mean to an equilibrium value μ, say, solution of the equation:

$$\mu = p_{11}\mu + (1-\mu)p_{10}(\mu), \tag{3.3}$$

whenever $p_{10}(.)$ is a continuous function. Moreover, if $p_{10}(.)$ is first-order differentiable, we get:

$$\begin{aligned} p_{10}(N_{t-1}/n) &\simeq p_{10}(\mu) + \frac{dp_{10}(\mu)}{d\mu}(N_{t-1}/n - \mu) \\ &= p_{10}(\mu) + \frac{1}{\sqrt{n}}\frac{dp_{10}(\mu)}{d\mu}X_{n,t-1}. \end{aligned}$$

This expansion modifies the basic limiting result in Proposition 2 [see Appendix 1]. We get the following result:

Corollary 1: *Let us consider a pure contagion model with $p_{10t} = p_{10}(N_{t-1}/n)$ and define $X_{n,t} = \sqrt{n}(N_t/n - \mu)$, where μ is the solution of equation (3.3). When $n \to \infty$, the process $X_{n,t}$ converges in distribution to a process ξ_t^* such that:*

$$\xi_t^* = \rho^*\xi_{t-1}^* + \eta^*\varepsilon_t^*,$$

where $\varepsilon_t^* \sim IIN(0,1)$ and:

$$\begin{aligned} \eta^{*2} &= \mu p_{11}(1-p_{11}) + (1-\mu)p_{10}(\mu)[1-p_{10}(\mu)], \\ \rho^* &= p_{11} - p_{10}(\mu) + (1-\mu)\frac{dp_{10}(\mu)}{d\mu}. \end{aligned}$$

Let us denote $\Psi_0(\mu) = p_{11}\mu + (1-\mu)p_{10}(\mu)$. Thus, the equilibrium value μ is a fixed point of function Ψ_0, whereas the limiting autoregressive parameter $\rho^* = \dfrac{d\Psi_0(\mu)}{d\mu}$ is the slope of function Ψ_0 at this point. Intuitively, the Gaussian limiting process is stationary if the fixed point is stable.

(**) In the general case with both correlated risks and contagion, we have $p_{10t} = p_{10}(F_t, N_{t-1}/n)$. By the same arguments as above, N_{t-1}/n tends to a limit μ_{t-1} when n tends to infinity, where μ_t satisfies the recursive equation:

$$\mu_t = p_{11}\mu_{t-1} + (1-\mu_{t-1})p_{10}(F_t, \mu_{t-1}), \tag{3.4}$$

which is the analogue of equation (3.3). Due to common factor F_t, the long run equilibrium at date t is now a dynamic stochastic equilibrium, which depends on the complete factor history. Corollary 1 becomes:

Corollary 2: *Let us consider a model such that $p_{10,t} = p_{10}(F_t, N_{t-1}/n)$ and define $X_{n,t} = \sqrt{n}(N_t/n - \mu_t)$, where the stochastic process (μ_t) of long run equilibria satisfies recursive equation (3.4). When $n \to \infty$, the process $(X_{n,t})$ converges in distribution to a process ξ_t^* such that:*

$$\xi_t^* = \rho_t^* \xi_{t-1}^* + \eta_t^* \varepsilon_t^*, \tag{3.5}$$

where $\varepsilon_t^* \sim IIN(0,1)$ and:

$$\begin{aligned} \eta_t^{*2} &= \mu_{t-1}p_{11}(1-p_{11}) + (1-\mu_{t-1})p_{10}(F_t,\mu_{t-1})[1-p_{10}(F_t,\mu_{t-1})], \\ \rho_t^* &= p_{11} - p_{10}(F_t,\mu_{t-1}) + (1-\mu_{t-1})\frac{\partial p_{10}}{\partial \mu}(F_t,\mu_{t-1}). \end{aligned}$$

We get a 3-dimensional nonlinear state space model, with state vector (ξ_t^*, μ_t, F_t). It is interesting to understand why the initial 2-dimensional state space (N_t, F_t) of the extended dynamic binomial process has been transformed into a 3-dimensional state space in the limiting case. In fact, we have:

$$N_t/n = \mu_t + \frac{1}{\sqrt{n}}\xi_t^* + o(1/\sqrt{n}). \tag{3.6}$$

Processes μ_t and ξ_t^* are providing the two first terms in the expansion of N_t/n in a neighbourhood of an infinite size n. They correspond to the cross-sectionally asymptotic (CSA) and granularity adjustment (GA) components, respectively, in the granularity approach developed in Basel 2 [see e.g. Gordy (2004), Gagliardini, Gourieroux, Monfort (2010)]. As noted before, μ_t provides the

dated long run equilibrium computed from a crystallized homogenous Markov chain with time independent transition matrix fixed at its current value. As seen from recursive equation (3.4), μ_t is a deterministic function of the current and lagged factor values. In particular, the sequence (μ_t) is stochastic, affected at each date by new shocks and does not converge when t tends to infinity. The cross-sectional limiting analysis shows that the sequence of granularity adjustments $(\xi_t^*, t \in \mathbb{N})$ has a simplified dynamics, which is linear Gaussian given the current and lagged values of F_t, μ_t.

Different specifications of probability function p_{10} can be introduced. For instance, we can consider a standard probit function with both frailty and lagged count as explanatory variables. This specification arises in the extension of the value of the firm model to contagion effects [see Rosch, Winterfeld (2008) for such an extension in a static framework]. A specification of p_{10} based on a logit function is analysed by means of simulation experiments in Section 4. Here we focus on two examples with pure contagion, and pure frailty effects, respectively.

Example 1: Logistic contagion

As an illustration, let us consider a pure contagion model with logistic contagion scheme:

$$p_{10t} = p_{10}(N_{t-1}/n) = \frac{1}{1 + \exp(-aN_{t-1}/n - b)}. \tag{3.7}$$

This logistic scheme is for instance considered for credit risk analysis in PortfolioView by Mc Kinsey. The long run equilibrium value [see (3.3)] is solution of the equation:

$$\frac{1-\mu}{\mu} \frac{1}{1 + \exp(-a\mu - b)} = 1 - p_{11}. \tag{3.8}$$

We prove in Appendix 2 that this solution $\mu = \mu(a, b, p_{11})$ exists, is unique and increasing with respect to parameters a, b, p_{11}. We also prove that the autoregressive coefficient $\rho^* = \rho^*(a, b, p_{11})$ in Corollary 1 is such that $|\rho^*| < 1$ for any values of $a \geq 0$, $b > 0$ and $p_{11} \in (0, 1)$. Hence the limit process (ξ_t^*) is stationary for any such parameter choice.

In Figure 1 we display the equilibrium value μ and the autoregressive coefficient ρ^* as functions of parameter a, for different values of parameters p_{11} and b.

[Insert Figure 1: Equilibrium value and autoregressive coefficient in the logistic model.]

The equilibrium value μ features an increasing pattern w.r.t. parameter a and approaches the maximum value $1/(2 - p_{11})$ when a gets large. The pattern of the autoregressive coefficient ρ^* can be

non-monotone w.r.t. parameter a, and ρ^* becomes negative for large a. The intuition for negative autoregressive coefficients is the following: For given values of p_{11} and b, when parameter a is sufficiently large the contagion probability $\pi_{10}(\mu)$ is such that $\pi_{10}(\mu) > p_{11}$. Then, at equilibrium the contagion probability is larger than the probability to remain sick. Suppose now we move N_{t-1}/n upward from equilibrium such that $N_{t-1}/n > \mu$. Then, the probability of contagion $\pi_{10}(N_{t-1}/n)$ increases, but the proportion of individuals $1 - N_{t-1}/n$ that can be contaged decreases. If the latter effect dominates, on average the proportion of sick individuals will be below the equilibrium, that is, $N_t/n < \mu$. Hence, a positive shock on $X_{n,t-1}$ is followed by a negative shock on $X_{n,t}$, which explains the negative autocorrelation coefficient.

Example 2: Pure frailty model

Let us consider the limiting model with frailty only. We have $p_{10}(F_t, \mu_{t-1}) = p_{10}(F_t) \equiv F_t^*$, where the transformed factor process F_t^* admits values in $(0, 1)$. The dynamic equation defining the sequence of equilibria becomes:

$$\begin{aligned} \mu_t &= p_{11}\mu_{t-1} + (1 - \mu_{t-1})F_t^* \\ &= (p_{11} - F_t^*)\mu_{t-1} + F_t^*. \end{aligned} \tag{3.9}$$

In particular, if factor (F_t^*) is a strong white noise, the sequence of dynamic equilibria satisfies a bilinear model of order 1 [see Granger and Andersen (1978), Pham, Tran (1981)]. [9] By recursive substitution, we get:

$$\mu_t = F_t^* + \sum_{h=1}^{\infty} \left[\prod_{k=0}^{h-1} (p_{11} - F_{t-k}^*) \right] F_{t-h}^*, \tag{3.10}$$

whenever the series in the right hand side exists.

Let us assume that process (F_t^*) in $(0, 1)$ is strictly stationary and ergodic. Then, the stationarity conditions for process (μ_t) can be derived from the results in Brandt (1986) [see also Pham, Tran (1981) and Bougerol, Picard (1992) when (F_t^*) is a strong white noise]. Specifically, process (μ_t) defined in (3.10) is the unique strictly stationary solution of the stochastic recursive equation (3.9)

[9] Process (μ_t) defined in (3.9) slightly differ from the definition of bilinear process of order 1 adopted in Pham, Tran (1981) since the shock in the stochastic autoregressive coefficient $p_{11} - F_t^*$ is equal to the innovation F_t^*, and not to its lagged value.

if:

$$E\left[\log|p_{11} - F_t^*|\right] < 0. \tag{3.11}$$

The latter condition is satisfied for any $p_{11} \in [0,1]$. Moreover, when $p_{11} < 1$, we have $\mu_t < 1$ a.s. and equation (3.9) can be solved for F_t^* to get $F_t^* = \dfrac{\mu_t - p_{11}\mu_{t-1}}{1-\mu_{t-1}}$. Hence, process (μ_t) is invertible and the information sets associated with the factor process (F_t^*) and the sequence of dynamic equilibria (μ_t) are the same.

ii) INAR model with correlated risks and contagion

When np_{10t} is equivalent to $\lambda_t = \lambda(F_t, N_{t-1})$, as n tends to infinity, we get a limiting INAR model with stochastic intensity. This type of model is especially simple if the stochastic intensity λ_t is an affine function of both F_t and N_{t-1}, and moreover (F_t) is itself an affine process. More precisely, let us assume:

Assumption A.4: $\lambda_t = c_0 + c_1 F_t + c_2 N_{t-1}$, *with* $c_0 > 0$, $c_1 \geq 0$ *and* $c_2 \geq 0$.

Assumption A.5: *The factor process is a positive compound autoregressive process with conditional Laplace transform:*

$$\psi_{1t}(u) = E[\exp(-uF_{t+1})|\underline{F_t}, \underline{N_t}] = \exp[-\alpha(u)F_t - \beta(u)], \tag{3.12}$$

for some positive functions α *and* β.

Since the conditional Laplace transform depends on F_t only, the factor features an exogenous dynamics.

Proposition 4: *Under Assumptions A.4-A.5, the INAR process with stochastic intensity is such that the bivariate process* (N_t, F_t) *is a CaR(1) process with conditional Laplace transform:*

$$\begin{aligned}
\psi_{1t}(u,v) &= E[\exp(-uN_{t+1} - vF_{t+1})|\underline{N_t}, \underline{F_t}] \\
&= \exp\Big\{ -c_0[1-\exp(-u)] - \beta\big(v + c_1[1-\exp(-u)]\,\big) \\
&\quad -N_t\,\big(c_2[1-\exp(-u)] - \log[1-\rho+\rho\exp(-u)]\,\big) - F_t\,\alpha\big(v + c_1[1-\exp(-u)]\,\big)\Big\}.
\end{aligned}$$

Proof: See Appendix 3.

The advantage of the specification above is the simple characterization of the case with correlated risks only (resp. contagion only), which corresponds to the restriction $c_2 = 0$ (resp. $c_1 = 0$). These hypotheses can be easily tested in practice once a parametric specification is chosen for the factor dynamics. For instance, it can be assumed that the factor is an autoregressive Gamma process (ARG) of order 1 [Gourieroux, Jasiak (2006)], that is, a time discretized Cox, Ingersoll, Ross process [Cox, Ingersoll, Ross (1985)]. The corresponding conditional Laplace transform is given by:

$$E[\exp(-uF_{t+1})|F_t] = \frac{1}{(1+\eta u)^{\delta}} \exp\left(-\frac{\gamma u}{1+\eta u} F_t\right), \tag{3.13}$$

that is,

$$\alpha(u) = \frac{\gamma u}{1+\eta u}, \quad \beta(u) = \delta \log(1+\eta u), \tag{3.14}$$

where $\gamma \geq 0$ and $\delta, \eta > 0$. The unconditional distribution of F_t is a gamma distribution and the component N_{0t} follows a Poisson distribution with gamma heterogeneity, that is, a negative binomial distribution. Thus, by introducing an ARG factor, we transform the initial process based on Poisson distributions in a process based on negative binomial distributions [see e.g. Bockenholt (1999)] and solve the standard overdispersion problem in a dynamic framework [Greenwood, Yule (1920)]. The sensitivity parameter c_1 in the intensity and the scale parameter η of the factor F_t cannot be identified separately. For instance, we can set parameter η such that $E[F_t] = 1$. Since $E[F] = \delta\eta/(1-\gamma)$ [see Gourieroux, Jasiak (2006)], we can assume $\eta = (1-\gamma)/\delta$.

The advantage of a CaR process is to provide easily nonlinear predictions at any horizon. More precisely, the conditional Laplace transform at horizon h is:

$$\begin{aligned} \psi_{h,t}(u,v) &= E[\exp(-uN_{t+h} - vF_{t+h})|\underline{N_t}, \underline{F_t}] \\ &= \exp[-a_{1,h}(u,v)N_t - a_{2,h}(u,v)F_t - b_h(u,v)], \end{aligned}$$

where $a_{1,h}$, $a_{2,h}$ and c_h are computed by recursion

$$\begin{aligned} a_{1,h}(u,v) &= c_2[1-\exp(-a_{1,h-1}(u,v))] - \log[1-\rho+\rho\exp(-a_{1,h-1}(u,v))] \\ a_{2,h}(u,v) &= \alpha\big\{a_{2,h-1}(u,v) + c_1[1-\exp(-a_{1,h-1}(u,v))]\ \big\}, \\ b_h(u,v) &= b_{h-1}(u,v) + c_0[1-\exp(-a_{1,h-1}(u,v))] + \beta\big\{a_{2,h-1}(u,v) + c_1[1-\exp(-a_{1,h-1}(u,v))]\ \big\}, \end{aligned}$$

where:

$$a_{1,1}(u,v) = c_2[1-\exp(-u)] - \log[1-\rho+\rho\exp(-u)], \quad a_{2,1}(u,v) = \alpha\big(v + c_1[1-\exp(-u)]\,\big),$$
$$b_1(u,v) = c_0[1-\exp(-u)] + \beta\,\big(v + c_1[1-\exp(-u)]\,\big).$$

Moreover, by considering the behaviour of functions $a_{1,1}(u,v)$ and $a_{2,1}(u,v)$ in a neighbourhood of $u = v = 0$, the stationarity conditions of the joint process (N_t, F_t) are directly deduced [see Darolles, Gourieroux, Jasiak (2006), Proposition 6.2]. More precisely, the joint process (N_t, F_t) is strictly stationary if and only if the modulus of the eigenvalues of the matrix:

$$\begin{pmatrix} \frac{\partial a_{1,1}}{\partial u}(0,0) & \frac{\partial a_{1,1}}{\partial v}(0,0) \\ \frac{\partial a_{2,1}}{\partial u}(0,0) & \frac{\partial a_{2,1}}{\partial v}(0,0) \end{pmatrix} = \begin{pmatrix} c_2+\rho & 0 \\ \gamma c_1 & \gamma \end{pmatrix}, \tag{3.15}$$

are strictly smaller than 1. We get the stationarity conditions:

$$c_2 + \rho < 1, \quad \gamma < 1. \tag{3.16}$$

The condition $\gamma < 1$ is the stationarity condition for the ARG process [see Gourieroux, Jasiak (2006)], while the condition $c_2 + \rho < 1$ involves the autoregressive parameter ρ of the INAR process and the parameter c_2 that describes the contagion effect in the stochastic intensity.

The first- and second-order moments of the stationary distribution of (N_t, F_t) are given in the next proposition, proved in Appendix 3.

Proposition 5: *When $\eta = (1-\gamma)/\delta$, the unconditional means, variances and covariances of process (N_t, F_t) are given by:*

$$E[N_t] = \frac{c_0 + c_1}{1-(c_2+\rho)}, \quad E[F_t] = 1,$$

$$V[N_t] = \frac{c_0+c_1}{1-(c_2+\rho)}\left[\frac{1-\rho^2}{1-(c_2+\rho)^2}\right] + \frac{c_1^2}{\delta}\frac{1}{1-(c_2+\rho)^2}\left[\frac{1+\gamma(c_2+\rho)}{1-\gamma(c_2+\rho)}\right], \quad V[F_t] = \frac{1}{\delta},$$

and:

$$Cov(F_t, N_t) = \frac{c_1}{\delta}\frac{1}{1-\gamma(c_2+\rho)}.$$

The stationary distribution of the count variable N_t features overdispersion, that is, $V[N_t] > E[N_t]$, when either $c_1 > 0$, or $c_2 > 0$ (or both). In the first case, overdispersion is due to the frailty effect,

while in the second case it is due to the contagion effect. The processes (N_t) and (F_t) feature a positive contemporaneous unconditional correlation when $c_1 > 0$.

Proposition 6 provides the autocorrelogram of the count process (N_t).

Proposition 6: *The autocorrelogram of process* (N_t) *is such that:*

$$Corr(N_{t+h}, N_t) = (1-\omega)(c_2+\rho)^h + \omega\gamma^h, \quad h \geq 0,$$

where $\omega = \dfrac{c_1\gamma}{\gamma-(c_2+\rho)}\dfrac{Cov(N_t,F_t)}{V(N_t)}$, *if* $\gamma \neq c_2+\rho$, *and:*

$$Corr(N_{t+h}, N_t) = (1+\tilde{\omega}h)\gamma^h, \quad h \geq 0,$$

where $\tilde{\omega} = c_1\dfrac{Cov(N_t,F_t)}{V(N_t)}$, *if* $\gamma = c_2+\rho$.

Proof: See Appendix 3.

The autocorrelogram of process (N_t) decays geometrically w.r.t. the lag. Indeed, as a consequence of the CaR(1) property of the joint process (N_t, F_t) in Proposition 4, the conditional mean of $(N_t, F_t)'$ given the past $(\underline{N_{t-1}}, \underline{F_{t-1}})$ is a linear function of the lag $(N_{t-1}, F_{t-1})'$, as in a bivariate VAR process (see Lemma A.1 in Appendix 3). The associated matrix of the autoregressive coefficients is the transposed of the matrix in equation (3.15), whose eigenvalues are $c_2+\rho$ and γ. Hence, when the eigenvalues $c_2+\rho$ and γ are distinct, the autocorrelogram of (N_t) is a linear combination of the autocorrelogram $(c_2+\rho)^h$, $h \in \mathbb{N}$, of the INAR process with pure contagion, and autocorrelogram γ^h, $h \in \mathbb{N}$, of the ARG process (F_t). When the two eigenvalues $c_2+\rho$ and γ are equal, the autocorrelogram of (N_t) involves also a multiplicative term that is linear in the lag.

Example 3: Pure correlated risks

When only correlated risks $\lambda_t = c_0 + c_1 F_t$ are introduced, we get a recursive system in which the factor dynamics is fixed exogenously, then driving the dynamics of the count process. This allows for computing nonlinear predictions in two steps, first by considering the conditional distribution

of N_{t+h} given $\underline{F_{t+h}}, \underline{N_t}$, then by reintegrating out the future factor path given $\underline{F_t}, \underline{N_t}$. We have:

$$\begin{aligned}
& E[\exp(-uN_{t+h})|\underline{F_{t+h}}, \underline{N_t}] \\
= \; & \exp\Big\{ -(\lambda_{t+h} + \rho\lambda_{t+h-1} + \ldots + \rho^{h-1}\lambda_{t+1})[1-\exp(-u)] + N_t \log[1-\rho^h + \rho^h \exp(-u)] \Big\} \\
= \; & \exp\Big\{ -\left[c_0 \frac{1-\rho^h}{1-\rho} + c_1(F_{t+h} + \rho F_{t+h-1} + \ldots + \rho^{h-1}F_{t+1}) \right][1-\exp(-u)] \\
& + N_t \log[1-\rho^h + \rho^h \exp(-u)] \Big\}.
\end{aligned}$$

We deduce that:

$$\begin{aligned}
E[\exp(-uN_{t+h})|\underline{F_t}, \underline{N_t}] \; = \; & \exp\left\{ -c_0 \frac{1-\rho^h}{1-\rho}[1-\exp(-u)] + N_t \log[1-\rho^h + \rho^h \exp(-u)] \right\} \\
& E\left[\exp\left\{ -c_1(F_{t+h} + \rho F_{t+h-1} + \ldots + \rho^{h-1}F_{t+1})[1-\exp(-u)] \right\} |F_t \right].
\end{aligned}$$

To conclude this computation, we have to explain how to compute recursively the nonlinear prediction of the smoothed future path $F_{t+h} + \rho F_{t+h-1} + \ldots + \rho^{h-1}F_{t+1}$. It is easily checked that :

$$E\{\exp[-v(F_{t+h} + \rho F_{t+h-1} + \ldots + \rho^{h-1}F_{t+1})]|F_t\} = \exp[-a_h(v)F_t - b_h(v)],$$

where a_h and c_h satisfy the recursive equations:

$$a_h(v) = \alpha[a_{h-1}(v) + v\rho^{h-1}], \quad b_h(v) = b_{h-1}(v) + \beta[a_{h-1}(v) + v\rho^{h-1}],$$

with initial conditions

$$a_1(v) = \alpha(v), \quad b_1(v) = \beta(v).$$

Thus, we get:

$$\begin{aligned}
E[\exp(-uN_{t+h})|\underline{F_t}, \underline{N_t}] \; = \; & \exp\Big\{ -c_0 \frac{1-\rho^h}{1-\rho}[1-\exp(-u)] + N_t \log[1-\rho^h + \rho^h \exp(-u)] \\
& -F_t \, a_h\left(c_1[1-\exp(-u)]\right) - b_h\left(c_1[1-\exp(-u)]\right) \Big\}.
\end{aligned}$$

4 Simulation experiments

In this Section we report the results of simulation experiments in two dynamic models with both contagion and correlated risks. The first model is a logistic specification admitting a limit Gaussian approximation. The second model is the INAR process with stochastic intensity.

4.1 Logistic model with contagion and correlated risks

The contagion probability admits a logistic specification:

$$p_{10}(F_t, N_{t-1}/n) = \frac{1}{1+\exp(-aN_{t-1}/n - b - cF_t)}, \tag{4.1}$$

where the parameters are $a = 5$, $b = -\log(9)$ and $c = 2$. The probability of staying in state 1 is $p_{11} = 0.5$. The factor F_t follows a Gaussian autoregressive process:

$$F_t = \gamma F_{t-1} + \sqrt{1-\gamma^2}\varepsilon_t, \tag{4.2}$$

where $\varepsilon_t \sim IIN(0,1)$. The factor process is standardized to have unconditional mean 0 and unconditional variance 1. The autoregressive coefficient is $\gamma = 0.5$. The number of individuals is $n = 100$.

In Figure 2 we display simulated paths for the factor F_t, the proportion N_t/n of individuals in state 1 and the stochastic equilibrium μ_t.

[Insert Figure 2: Simulated paths of factor, count and stochastic equilibrium in the logistic model with contagion and correlated risks.]

The path of N_t/n is close to that of the stochastic equilibrium μ_t, that is, the CSA approximation, although the path of μ_t is smoother. The dynamics of the equilibrium μ_t features regimes that are driven by factor F_t. When the values of factor F_t are close to zero, the equilibrium μ_t is close to 0.6. When the factor F_t features negative shocks, the equilibrium μ_t decreases sharply. Positive shocks on F_t are associated with rather small increases in μ_t. Hence, the reaction of the dynamic equilibrium μ_t to positive and negative shocks in F_t is asymmetric.

Figure 3 displays simulated paths for the standardized deviation from the equilibrium $X_{n,t} = \sqrt{n}(N_t/n - \mu_t)$, the autoregressive coefficient ρ_t^* and the volatility η_t^* of the Gaussian approximation.

[Insert Figure 3: Simulated paths of deviation from equilibrium, autoregressive coefficient and volatility of the Gaussian approximation for the logistic model with contagion and correlated risks.]

The path of $X_{n,t}$ features regimes in persistency, with both periods of positive autocorrelation and periods of negative autocorrelation. This is reflected in the dynamics of the autoregressive

coefficient ρ_t^* of the Gaussian approximation, that admits both values slightly above 1 and negative values. The path of volatility η_t^* is more stable than that of the autoregressive coefficient ρ_t^*, and features some sharp downward movements associated with the negative shocks on the factor F_t.

In order to assess the accuracy of the approximation (3.6), let us consider the standardized residuals:

$$\tilde{\varepsilon}_t^* = \frac{X_{n,t} - \rho_t^* X_{n,t-1}}{\eta_t^*}, \quad t \text{ varying},$$

that are the residuals for the autoregressive process in (3.5) computed from process $X_{n,t}$. If the approximation (3.6) is accurate, process $\tilde{\varepsilon}_t^*$ is close to a Gaussian white noise. We display some summary statistics for the unconditional distribution as well as some autocorrelation coefficients for process $\tilde{\varepsilon}_t^*$ in Table 1. They are computed by Monte-Carlo on a long simulated path of the process.

[Insert Table 1: Summary statistics and autocorrelogram of process $\tilde{\varepsilon}_t^*$.]

We consider different population sizes, that are $n = 25$, $n = 100$ and $n = 1000$. From Table 1 it is seen that process $\tilde{\varepsilon}_t^*$ gets closer to a white noise when n increases, which confirms that the accuracy of approximation (3.6) improves with the population size.

4.2 INAR model with stochastic intensity

Let us now consider an INAR model with stochastic intensity as in Assumptions A.4-A.5. The exogenous factor F_t follows an ARG process with autocorrelation parameter $\gamma = 0.5$ and shape parameter $\delta = 2$. The scale parameter η is set equal to $\eta = (1-\gamma)/\delta = 0.25$ to get $E[F_t] = 1$ [see Section 3.2 ii)]. The autoregressive parameter ρ of the INAR process is $\rho = 0.2$. Moreover, we consider four parameter sets for the intensity specification:

A) $c_0 = 2.4$, $c_1 = 0$, $c_2 = 0$,

B) $c_0 = 1.4$, $c_1 = 1$, $c_2 = 0$,

C) $c_0 = 1.2$, $c_1 = 0$, $c_2 = 0.4$,

D) $c_0 = 0.2$, $c_1 = 1$, $c_2 = 0.4$.

Parameters c_1 and c_2 are selected such that models A, B, C, and D correspond to specifications with constant intensity, pure frailty effect, pure contagion effect, and both frailty and contagion effects, respectively. The parameter c_0 is selected to have the same unconditional mean for count N_t across all specifications, which is equal to $E[N_t] = \dfrac{c_0 + c_1}{1 - (\rho + c_2)} = 3$.

i) Simulated paths

Let us first compare simulated paths of the process (N_t) for the different parameter sets A-D. For this purpose, it is useful to rewrite the model in the nonlinear autoregressive stochastic representation:

$$N_t = \inf\left\{ m \in \mathbb{N}, m \leq N_{t-1} : \sum_{j=0}^{m} \pi_{j,t}^{(1)} \geq U_{1,t} \right\} + \inf\left\{ m \in \mathbb{N} : \sum_{j=0}^{m} \pi_{j,t}^{(2)} \geq U_{2,t} \right\} \equiv a(N_{t-1}, F_t, U_t),$$

where $\pi_{j,t}^{(1)} = \dfrac{N_{t-1}!}{j!(N_{t-1}-j)!}\rho^j(1-\rho)^{N_{t-1}-j}$ and $\pi_{j,t}^{(2)} = e^{-\lambda_t}\dfrac{\lambda_t^j}{j!}$, with $\lambda_t = c_0 + c_1 F_t + c_2 N_{t-1}$, are the probability weights for the conditional binomial and Poisson distributions $\mathcal{B}(N_{t-1}, \rho)$ and $\mathcal{P}(\lambda_t)$ given N_{t-1} and F_t, respectively, and variables $U_t = (U_{1,t}, U_{2,t})$ are i.i.d. such that $U_{1,t}$ and $U_{2,t}$ are independent with uniform distribution $\mathcal{U}[0,1]$. The distribution of the factor (F_t) and shocks (U_t) is independent of intensity parameters c_0, c_1 and c_2. This allows us to compare the simulated paths of process (N_t) obtained from a same path of (F_t, U_t) and different intensity parameters c_0, c_1, c_2 as in sets A-D above.

The simulated path of (N_t) for parameter sets A-B and C-D are displayed in Figures 4 and 5, respectively.

[Insert Figure 4: Simulated paths of the INAR process with stochastic intensity (parameter sets A and B)]

[Insert Figure 5: Simulated paths of the INAR process with stochastic intensity (parameter sets C and D)]

ii) Conditional expectation

In Figure 6 we display the conditional expectation of N_t given $N_{t-1} = n_{t-1}$ as a function of lagged value n_{t-1} for parameter sets A-D.

[Insert Figure 6: Conditional expectation of N_t given N_{t-1} in the INAR process with stochastic intensity]

The conditional expectation is linear $E[N_t|N_{t-1}] = c_0 + (\rho + c_2)N_{t-1} = E[N_t] + (\rho + c_2)(N_{t-1} - E[N_t])$ for models with constant intensity, or pure contagion effects (parameter sets A and C). For models including frailty effects (parameter sets B and D), we compute the conditional expectation $E[N_t|N_{t-1}]$ by Monte-Carlo on a long simulated path of process (N_t). From Figure 6 it is seen that the conditional expectation is close to linear also for parameter sets B and D. Moreover, models A, B, C and D are ranked in order of increasing (linear, first-order) persistency. However, model C with pure contagion cannot be distinguished from a model with constant intensity and autoregressive INAR parameter $\tilde{\rho} = \rho + c_2$ based on the conditional expectation at lag one. Figure 6 suggests that this is hardly possible also for models B and D including frailty effects.

iii) Autocorrelogram

From Proposition 6 the autocorrelogram of (N_t) is a mixture of two power functions of γ and $c_2 + \rho$, respectively. Thus, we expect to better identify contagion and frailty effects from the ACF, at least when parameters γ and $c_2 + \rho$ are both non-zero and sufficiently different. More precisely, the log ACF is non linear if, and only if, $c_1 > 0$ and $\gamma > 0$, that is, there is a dynamic frailty effect. The nonlinearity is weak when the autocorrelation coefficient of the frailty γ is close to $c_2 + \rho$. There is no nonlinearity at all when the frailty is static.

In Figure 7 we display the autocorrelogram $\{Corr(N_{t+h}, N_t),\ h \in \mathbb{N}\}$ of process (N_t) for parameter sets A-D.

[Insert Figure 7: Autocorrelogram of the INAR process (N_t) with stochastic intensity]

For experiments A and C with no frailty effect, the log ACF is linear. At the opposite, some curvature of the log ACF is observed for experiment B with pure frailty, where $c_2 + \rho = 0.2$ and $\gamma = 0.5$. The log ACF is almost linear for experiment D with both frailty and contagion, where

$c_2 + \rho = 0.6$ and $\gamma = 0.5$.

iv) Conditional dispersion

Let us now investigate whether the analysis of higher-order moments of the conditional distribution of N_{t+1} given N_t can be useful for the purpose of identifying contagion vs frailty effects. The dispersion of the conditional distribution of N_{t+1} given N_t is defined as the ratio between conditional variance $V[N_{t+1}|N_t]$ and conditional mean $E[N_{t+1}|N_t]$. In Appendix 3 we show that:

$$V[N_{t+1}|N_t] = E[N_{t+1}|N_t] - \rho^2 N_t + c_1^2 \left(\gamma^2 V[F_t|N_t] + 2\frac{\gamma(1-\gamma)}{\delta} E[F_t|N_t] + \frac{(1-\gamma)^2}{\delta} \right), \quad (4.3)$$

where:

$$E[N_{t+1}|N_t] = E[N_t] + (c_2 + \rho)(N_t - E[N_t]) + c_1\gamma(E[F_t|N_t] - 1).$$

Hence, a model without frailty effect ($c_1 = 0$) features conditional underdispersion. The contribution of frailty to conditional dispersion is positive. It involves the conditional mean $E[F_t|N_t]$ and variance $V[F_t|N_t]$ of the unobservable factor F given the observable count N_t, as well as the unconditional variance $V[F_t] = 1/\delta$ of the factor and its autocorrelation parameter γ. When the sensitivity parameter c_1 is large enough, the positive frailty effect can dominate and yield conditional overdispersion (at least for some lag N_t).

In Figure 8 we display the conditional dispersion of N_{t+1} given N_t as a function of lagged value N_t for parameter sets A-D.

[Insert Figure 8: Conditional dispersion of N_{t+1} given N_t in the INAR process with stochastic intensity]

5 Conclusions

In this paper we analyze frailty correlated risks and contagion effects in large homogeneous populations. We consider a microscopic dynamic model in which individual risks can take two states (high and low), and the individual transition probabilities between states are time varying and stochastic. The frailty effect is modeled by means of a common unobservable factor F_t that impacts the

individual transition probabilities. The contagion effect is modeled through the dependence of the individual transition probabilities on the lagged count N_{t-1} of individuals in the high risk state. We derive macroscopic models for the count N_t as the limit of the microscopic model when the population size n tends to infinity. Different macroscopic dynamics are obtained according to whether the transition probabilities are assumed fixed w.r.t. n (Gaussian approximation), or the transition probability to the high risk state converges to zero when n increases (Poisson approximation). In the first setting, we derive an approximation for the dynamics of the proportion N_t/n in terms of a dynamic stochastic equilibrium driven by factor F_t plus a correction at order $1/\sqrt{n}$ involving a conditionally Gaussian autoregressive process. In the second setting, we carefully study the properties of the INAR process for count N_t with stochastic intensity driven by factor F_t and lagged count N_{t-1}.

An interesting question is to which extent it is possible to identify frailty and contagion effects from the macroscopic dynamics only. The analysis of the INAR model with stochastic intensity suggests that the identification of these two effects can be rather difficult when relying solely on summaries that capture linear dynamics, such as the conditional mean function or the autocorrelogram of process (N_t). Instead, nonlinear features of the conditional distribution of N_t given N_{t-1}, such as the conditional dispersion function, can be very useful to disentangle frailty and contagion effects.

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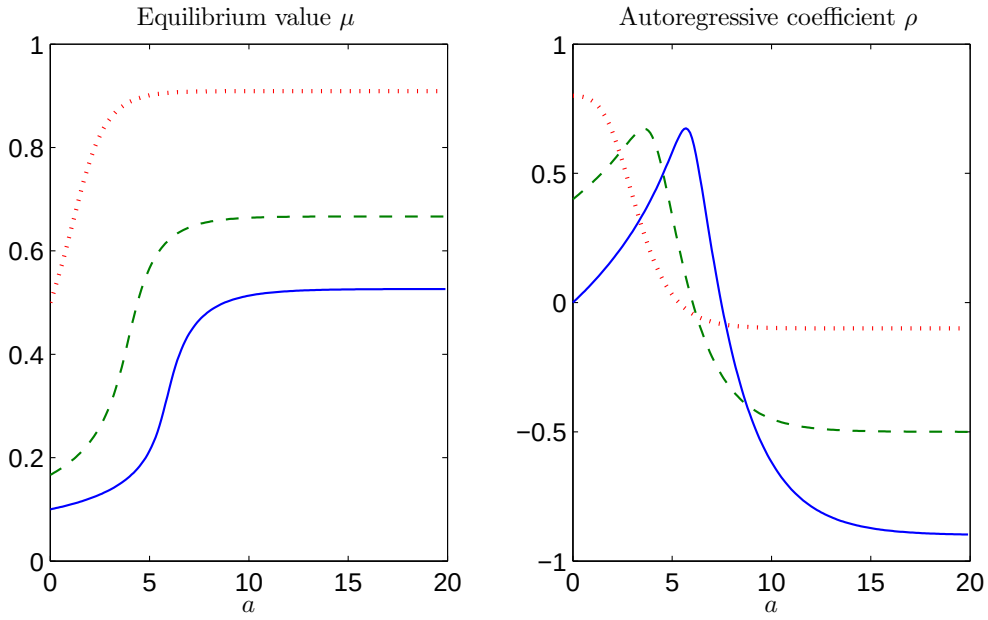
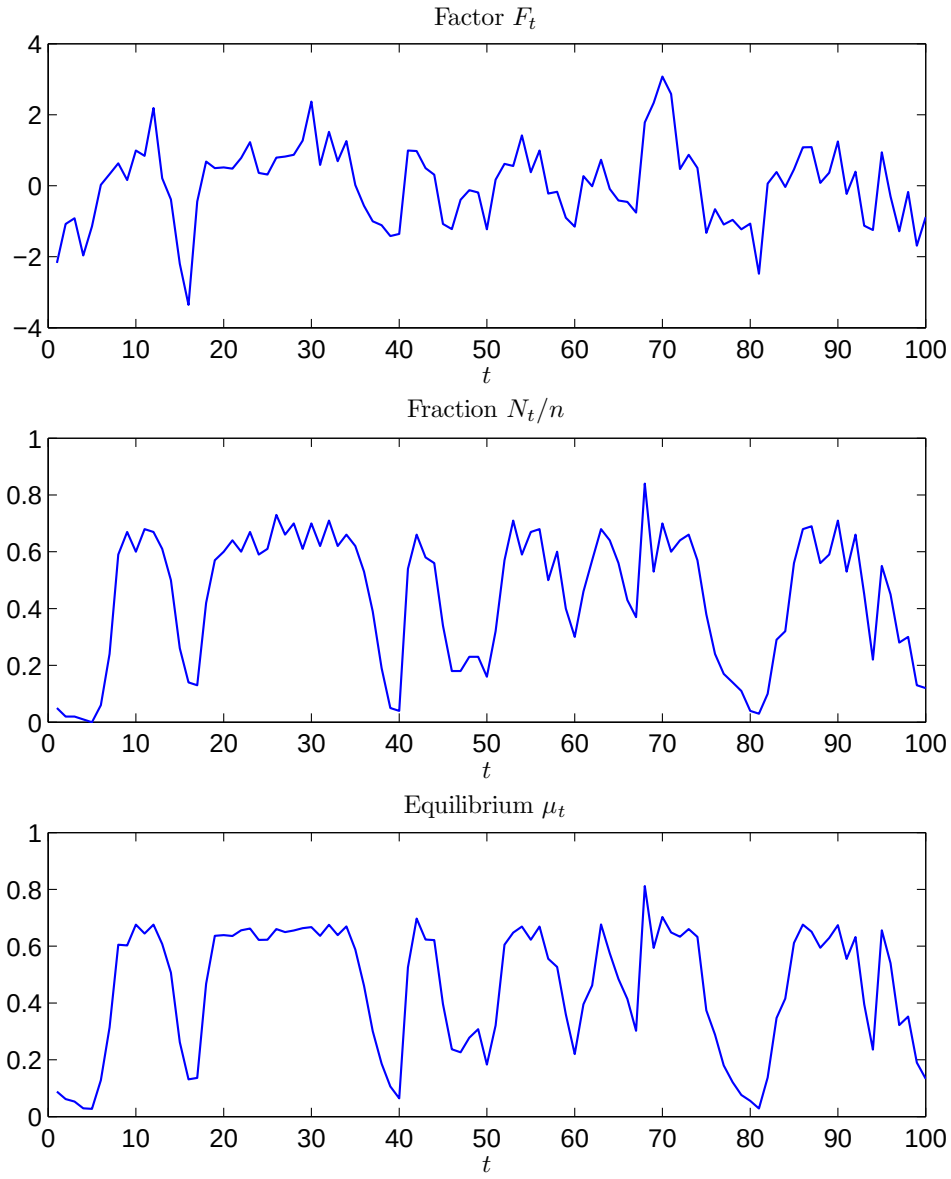
Figure 1: Equilibrium value and autoregressive coefficient in the logistic model.



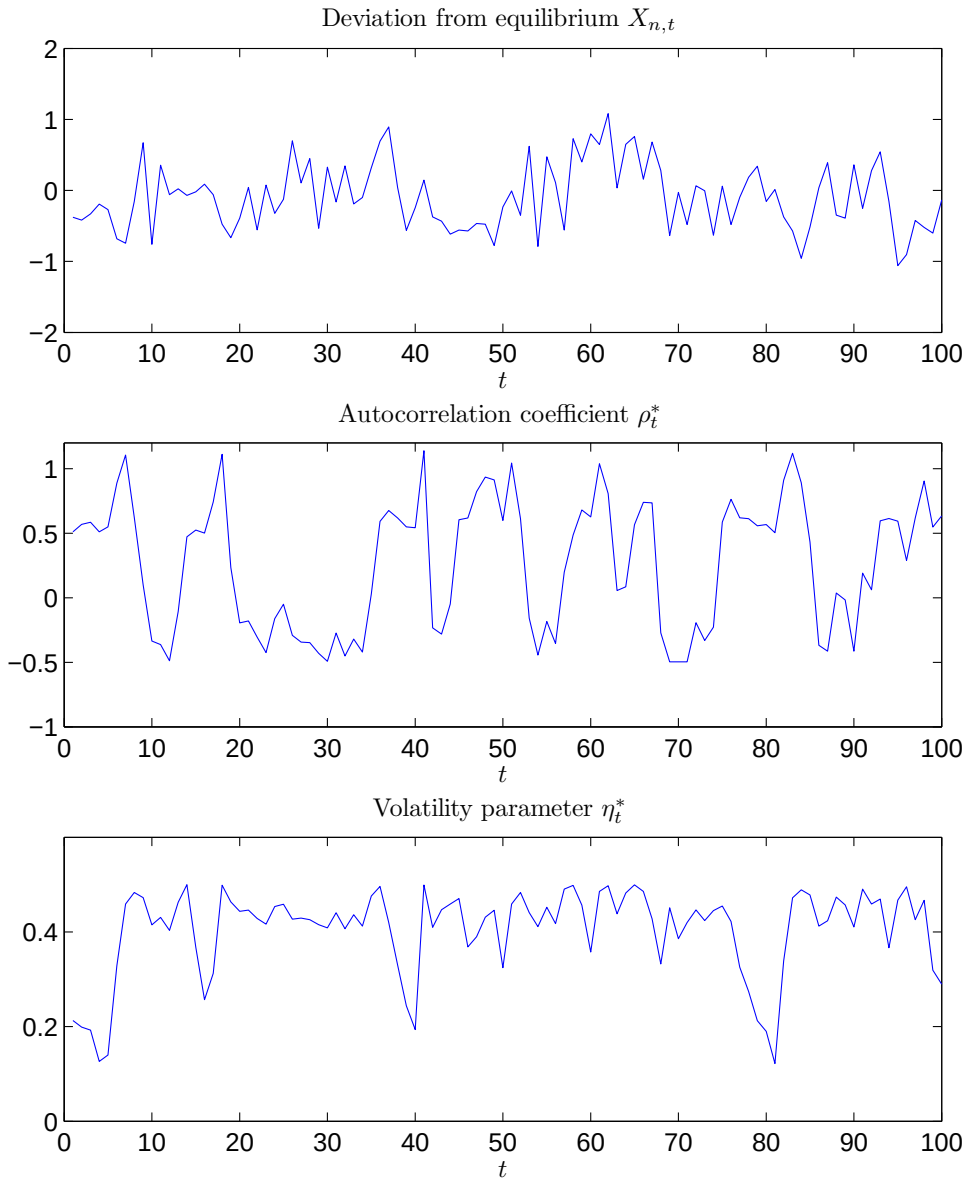
The left panel displays the equilibrium value μ as a function of parameter a, while the right panel displays the autoregressive coefficient ρ^* as a function of a. In each panel, the three curves correspond to different values of parameter p_{11}, that are $p_{11} = 1/10$ (solid line), $p_{11} = 1/2$ (dashed line) and $p_{11} = 9/10$ (dotted line). Parameter b is equal to $b = -\log(9)$.

Figure 2: Simulated paths of factor, count and stochastic equilibrium in the logistic model with contagion and correlated risks.



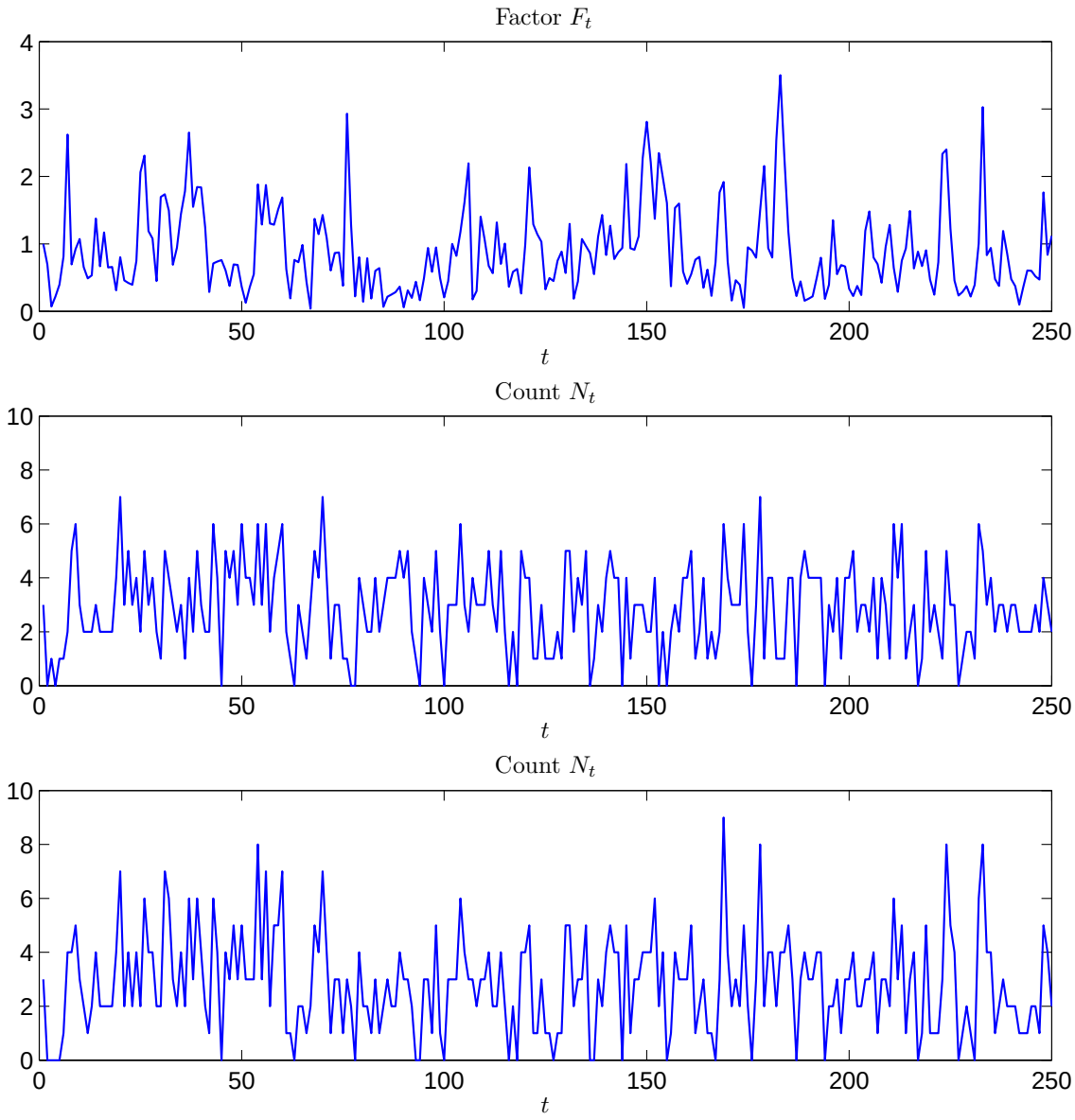
This Figure displays simulated paths for the logistic model with contagion and correlated risks (4.1)-(4.2). The parameter values are $a = 5$, $b = -\log(9)$, $c = 2$, $p_{11} = 0.5$ and $\gamma = 0.5$. The number of individuals is $n = 100$. The upper Panel displays the path of the factor F_t, the middle Panel displays the path of the fraction N_t/n of individuals in state 1, and the lower Panel displays the path of the stochastic equilibrium μ_t.

Figure 3: Simulated paths of deviation from equilibrium, autoregressive coefficient and volatility of the Gaussian approximation for the logistic model with contagion and correlated risks.



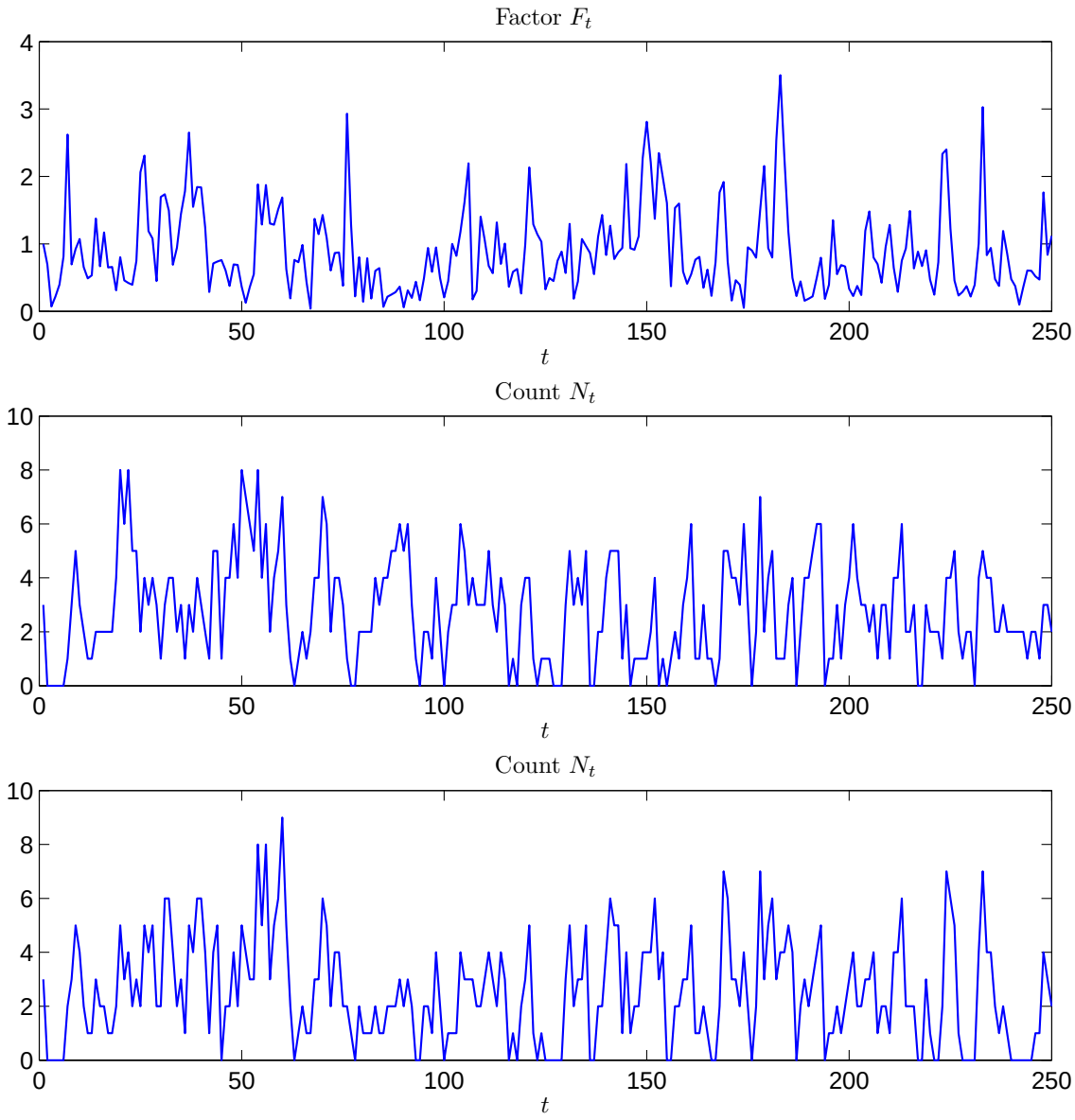
This Figure displays simulated paths for the logistic model with contagion and correlated risks (4.1)-(4.2). The parameter values are $a = 5$, $b = -\log(9)$, $c = 2$, $p_{11} = 0.5$ and $\gamma = 0.5$. The number of individuals is $n = 100$. The upper Panel displays the path of the standardized deviation from equilibrium $X_{n,t} = \sqrt{n}(N_t/n - \mu_t)$, the middle Panel displays the path of the autocorrelation coefficient ρ_t^* of the Gaussian approximation ξ_t^* in Corollary 3, and the lower Panel displays the path of its volatility parameter η_t^*.

Figure 4: Simulated paths of the INAR process with stochastic intensity (parameter sets A and B).



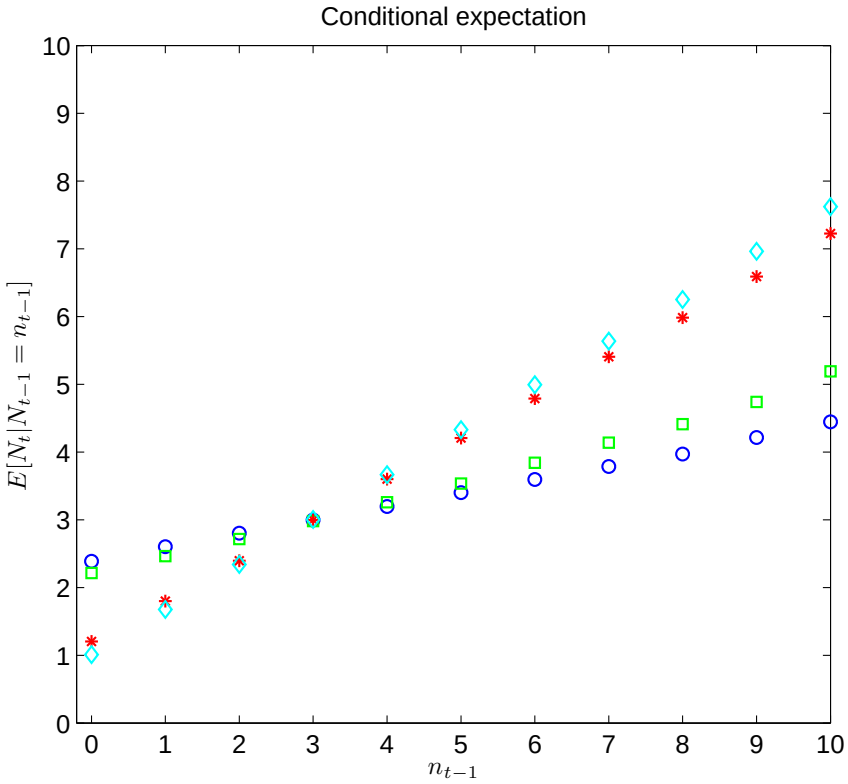
This Figure displays simulated paths for the INAR process with stochastic intensity as in Assumptions A.4-A.5. The factor F_t follows an ARG process with parameters $\gamma = 0.5$, $\delta = 2$ and $\eta = 0.25$. The autoregressive parameter ρ of the INAR model is $\rho = 0.2$. The upper panel displays the factor path. The middle and lower panels display the paths of the count N_t for models with intensity parameters c_0, c_1 and c_2 as in sets A and B, respectively.

Figure 5: Simulated paths of the INAR process with stochastic intensity (parameter sets C and D).



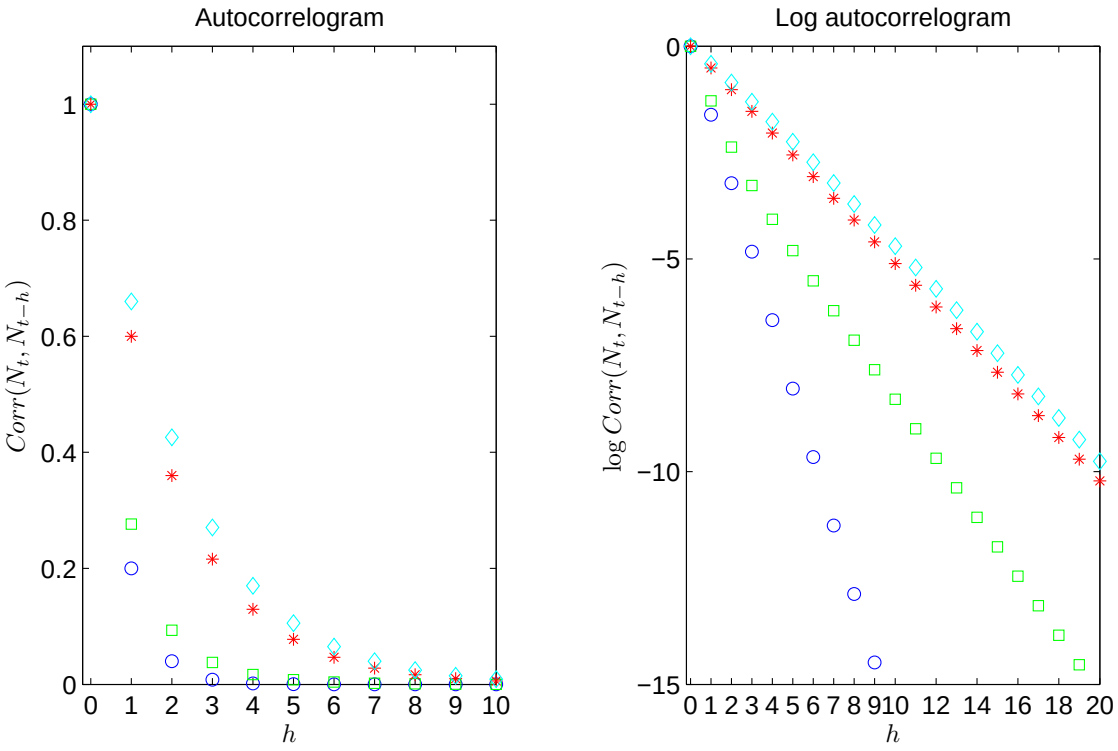
This Figure displays simulated paths for the INAR process with stochastic intensity as in Assumptions A.4-A.5. The factor F_t follows an ARG process with parameters $\gamma = 0.5$, $\delta = 2$ and $\eta = 0.25$. The autoregressive parameter ρ of the INAR model is $\rho = 0.2$. The upper panel displays the factor path. The middle and lower panels display the paths of the count N_t for models with intensity parameters c_0, c_1 and c_2 as in sets C and D, respectively.

Figure 6: Conditional expectation of N_t given N_{t-1} in the INAR process with stochastic intensity.



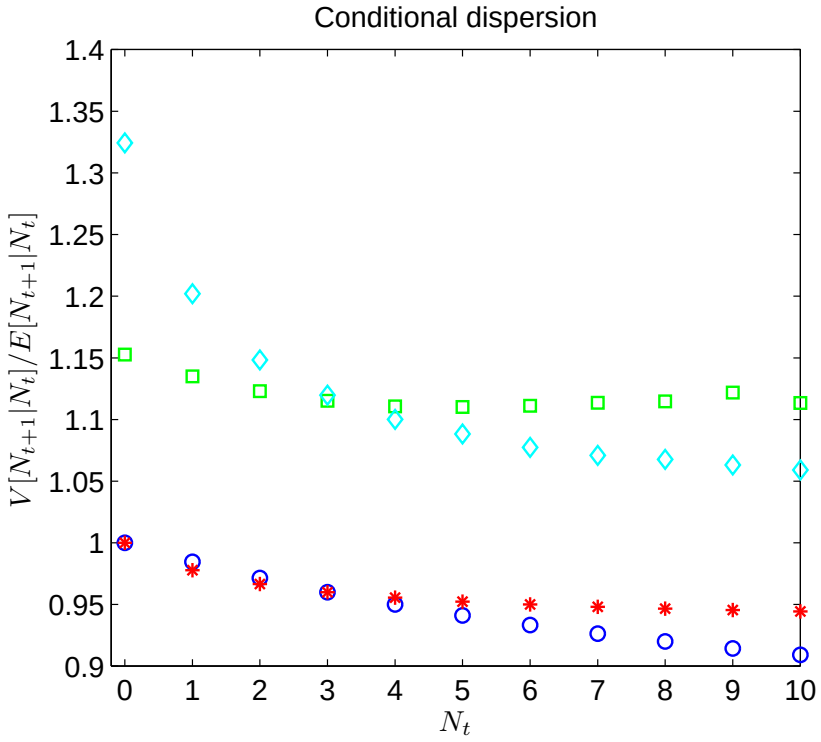
This Figure displays the conditional expectation of N_t given N_{t-1} for the INAR process with stochastic intensity as in Assumptions A.4-A.5. The factor F_t follows an ARG process with parameters $\gamma = 0.5$, $\delta = 2$ and $\eta = 0.25$. The autoregressive parameter ρ of the INAR model is $\rho = 0.2$. Circles, squares, stars and diamonds correspond to intensity parameters c_0, c_1 and c_2 as in sets A, B, C and D, respectively.

Figure 7: Autocorrelogram of the INAR process (N_t) with stochastic intensity.



This Figure displays the autocorrelogram (left panel) and the log autocorrelogram (right panel) of the INAR process (N_t) with stochastic intensity as in Assumptions A.4-A.5. The factor F_t follows an ARG process with parameters $\gamma = 0.5$, $\delta = 2$ and $\eta = 0.25$. The autoregressive parameter ρ of the INAR model is $\rho = 0.2$. Circles, squares, stars and diamonds correspond to intensity parameters c_0, c_1 and c_2 as in sets A, B, C and D, respectively.

Figure 8: Conditional dispersion function of the INAR process (N_t) with stochastic intensity.



This Figure displays the pattern of the conditional dispersion of N_{t+1} given N_t, as a funtion of N_t, for the INAR process (N_t) with stochastic intensity as in Assumptions A.4-A.5. The factor F_t follows an ARG process with parameters $\gamma = 0.5$, $\delta = 2$ and $\eta = 0.25$. The autoregressive parameter ρ of the INAR model is $\rho = 0.2$. Circles, squares, stars and diamonds correspond to intensity parameters c_0, c_1 and c_2 as in sets A, B, C and D, respectively.

Table 1: Summary statistics and autocorrelogram of process $\tilde{\varepsilon}_t^*$.

	$n = 25$	$n = 100$	$n = 1000$
Mean	-0.068	-0.035	-0.011
Median	-0.076	-0.041	-0.012
Std. deviation	1.001	1.001	1.000
Skewness	0.015	0.025	0.013
Kurtosis	3.156	3.042	3.000
$AC(1)$	0.016	0.006	0.000
$AC(2)$	0.002	0.002	0.001
$AC(3)$	0.002	-0.000	0.001
$AC(4)$	0.001	0.001	0.000
$AC(5)$	0.000	0.000	0.000

This Table displays summary statistics and the autocorrelogram of process $\tilde{\varepsilon}_t^* = \frac{X_{n,t} - \rho_t^* X_{n,t-1}}{\eta_t^*}$ for the logistic model with contagion and correlated risks (4.1)-(4.2). The parameter values are $a = 5$, $b = -\log(9)$, $c = 2$, $p_{11} = 0.5$ and $\gamma = 0.5$. We consider different population sizes that are $n = 25$, $n = 100$ and $n = 1000$. Statistic $AC(h) = Corr(\tilde{\varepsilon}_t^*, \tilde{\varepsilon}_{t-h}^*)$ denotes the autocorrelation of $\tilde{\varepsilon}_t^*$ of order h.

APPENDIX 1: Proofs of Proposition 2 and Corollary 1

From equation (2.4), the conditional log-Laplace transform of $X_{n,t} = \sqrt{n}(N_t/n - \mu)$ is:

$$\begin{aligned} & \log E_{t-1}[\exp(-uX_{n,t})] \\ = \; & N_{t-1}\log[p_{11}\exp(-u/\sqrt{n}) + 1 - p_{11}] + (n - N_{t-1})\log[p_{10}\exp(-u/\sqrt{n}) + 1 - p_{10}] \\ & + \sqrt{n}u\mu. \end{aligned}$$

By a Taylor expansion when $n \to \infty$, we get :

$$\begin{aligned} & \log[p_{11}\exp(-u/\sqrt{n}) + 1 - p_{11}] = \log[1 - p_{11}u/\sqrt{n} + p_{11}u^2/(2n) + o(1/n)] \\ = \; & -p_{11}u/\sqrt{n} + p_{11}(1-p_{11})u^2/2n + o(1/n), \end{aligned}$$

and similarly:

$$\begin{aligned} & \log E_{t-1}[\exp(-uX_{n,t})] \\ = \; & (\mu n + \sqrt{n}X_{n,t-1})[-p_{11}u/\sqrt{n} + p_{11}(1-p_{11})u^2/(2n)] \\ & + [(1-\mu)n - \sqrt{n}X_{n,t-1}][-p_{10}u/\sqrt{n} + p_{10}(1-p_{10})u^2/(2n)] + \sqrt{n}u\mu + o(1/n) \\ = \; & u\sqrt{n}[-p_{10}(1-\mu) - p_{11}\mu + \mu] \\ & - uX_{n,t-1}(p_{11} - p_{10}) + (u^2/2)[\mu p_{11}(1-p_{11})] + (1-\mu)p_{10}(1-p_{10})] + o(1). \end{aligned}$$

The first term of the right hand side is equal to 0 by definition of the long run parameter. The sum of the second and third terms is the log-Laplace transform of a Gaussian distribution with mean $(p_{11} - p_{10})X_{n,t-1} = \rho X_{n,t-1}$, and variance $\mu p_{11}(1-p_{11}) + (1-\mu)p_{10}(1-p_{10}) = \mu(1-\mu)(1-\rho^2)$. This proves Proposition 2.

Finally, if p_{10} is replaced by $p_{10t} = p_{10}(N_{t-1}/n) \simeq p_{10}(\mu) + \dfrac{1}{\sqrt{n}}\dfrac{dp_{10}(\mu)}{d\mu}X_{n,t-1}$, we get the additional term $-uX_{n,t-1}(1-\mu)\dfrac{dp_{10}(\mu)}{d\mu} + o(1)$ in the expansion. This provides the modification involved in Corollary 1.

APPENDIX 2: Gaussian model with logistic contagion

i) Let us first show that the solution $\mu = \mu(a, b, p_{11}) \in (0, 1)$ of equation (3.8) exists and is unique. Define the function:

$$\psi(\mu) = \frac{1/\mu - 1}{1 + \exp(-a\mu - b)}, \quad \mu \in (0, 1).$$

Since $\psi(0) = \infty, \psi(1) = 0$, the equation $\psi(\mu) = 1 - p_{11}$ for $p_{11} \in (0, 1)$ admits a solution $\mu \in (0, 1)$. The solution is unique, if function ψ is monotonically decreasing. The first-order derivative is given by:

$$\begin{aligned}
\frac{d\psi(\mu)}{d\mu} &= \frac{-\frac{1}{\mu^2}(1 + e^{-a\mu-b}) + (1/\mu - 1)(-a)e^{-a\mu-b}}{(1 + e^{-a\mu-b})^2} \\
&= -\frac{e^{-a\mu-b}}{\mu^2(1 + e^{-a\mu-b})^2}[e^{a\mu+b} - (a\mu^2 - a\mu - 1)].
\end{aligned}$$

Since $a\mu^2 - a\mu - 1 < 0$ for $\mu \in [0, 1]$, we get $\frac{d\psi(\mu)}{d\mu} < 0$ for $\mu \in (1, 1)$, and the conclusion follows.

ii) Let us now study the dependence of equilibrium μ on parameters a, b, p_{11}. We make explicit the dependence of function ψ on parameters a and b by writing $\psi(\mu) = \psi(\mu; a, b)$. Then:

$$\begin{aligned}
\frac{\partial\mu}{\partial a} &= -\frac{\partial\psi/\partial a}{\partial\psi/\partial\mu} = \mu\frac{\mu(1-\mu)}{e^{a\mu+b} - (a\mu^2 - a\mu - 1)} > 0, \\
\frac{\partial\mu}{\partial b} &= -\frac{\partial\psi/\partial b}{\partial\psi/\partial\mu} = \frac{\mu(1-\mu)}{e^{a\mu+b} - (a\mu^2 - a\mu - 1)} > 0, \\
\frac{\partial\mu}{\partial p_{11}} &= -\frac{1}{\partial\psi/\partial\mu} = \frac{\mu^2(1 + e^{a\mu+b})(1 + e^{-a\mu-b})}{e^{a\mu+b} - (a\mu^2 - a\mu - 1)} > 0.
\end{aligned}$$

iii) Let us finally show that $\rho^* = \rho^*(a, b, p_{11})$ is such that $|\rho^*| < 1$. We have from Corollary 1:

$$\rho^* = p_{11} - p_{10}(\mu) + a(1 - \mu)p_{10}(\mu)[1 - p_{10}(\mu)],$$

where $\mu = \mu(a, b, p_{11})$ is the equilibrium value. Then $\rho^* > -\pi_{10}(\mu) \geq -1$. Moreover, by using that $\mu = \mu(a, b, p_{11})$ solves equation (3.8), we get:

$$\begin{aligned}
& \rho^* = p_{11} - p_{10}(\mu) + a\mu(1 - p_{11})[1 - p_{10}(\mu)] < 1 \\
\Leftrightarrow \quad & (1 - p_{11})\{a\mu[1 - p_{10}(\mu)] - 1\} < p_{10}(\mu) \\
\Leftrightarrow \quad & a\mu[1 - p_{10}(\mu)] - 1 < \frac{\mu}{1 - \mu} \\
\Leftrightarrow \quad & a\mu(1 - \mu)[1 - p_{10}(\mu)] < 1. \qquad \text{(A.1)}
\end{aligned}$$

Now, by using that $1 - p_{10}(\mu) = e^{-a\mu-b}/(1+e^{-a\mu-b})$ and $a\mu e^{-a\mu} < 1$ for $a\mu \geq 0$, we deduce that the latter inequality in (A.1) is satisfied for any values of the parameters.

APPENDIX 3: INAR model with correlated risks and contagion

A.3.1 Proof of Proposition 4

We have:

$$\begin{aligned}
\psi_{1t}(u,v) &= E[\exp(-uN_{t+1} - vF_{t+1})|\underline{N_t}, \underline{F_t}] \\
&= E\left\{E[\exp(-uN_{t+1} - vF_{t+1})|\underline{N_t}, \underline{F_{t+1}}]|\underline{N_t}, \underline{F_t}\right\} \\
&= E\left[\exp(-vF_{t+1})\exp\left\{-(c_0 + c_1F_{t+1} + c_2N_t)[1-\exp(-u)]\right.\right. \\
&\quad \left.\left. + N_t\log[1-\rho+\rho\exp(-u)]\right\}|\underline{N_t}, \underline{F_t}\right],
\end{aligned}$$

by applying equation (2.6) and Assumption A.1. Therefore we get:

$$\begin{aligned}
\psi_{1t}(u,v) &= \exp\left\{-c_0[1-\exp(-u)] - N_t\left(c_2[1-\exp(-u)] - \log[1-\rho+\rho\exp(-u)]\right)\right\} \\
&\quad E\left[\exp\left\{-F_{t+1}(v + c_1[1-\exp(-u)])\right\}|F_t\right] \\
&= \exp\left\{-c_0[1-\exp(-u)] - \beta\left(v + c_1[1-\exp(-u)]\right)\right. \\
&\quad -N_t\left(c_2[1-\exp(-u)] - \log[1-\rho+\rho\exp(-u)]\right) \\
&\quad \left. -F_t\,\alpha\left(v + c_1[1-\exp(-u)]\right)\right\}.
\end{aligned}$$

A.3.2 Proof of Proposition 5

We use the following Lemma, which is proved at the end of this Appendix.

Lemma A.1: *The conditional moments of order* 1 *and* 2 *of the joint process* (N_t, F_t) *are given by:*

$$E_t\begin{pmatrix} N_{t+1} \\ F_{t+1} \end{pmatrix} = \begin{pmatrix} c_0 + \delta\eta c_1 \\ \delta\eta \end{pmatrix} + \begin{pmatrix} c_2+\rho & \gamma c_1 \\ 0 & \gamma \end{pmatrix}\begin{pmatrix} N_t \\ F_t \end{pmatrix}$$

and:

$$V_t\begin{pmatrix} N_{t+1} \\ F_{t+1} \end{pmatrix} = \begin{pmatrix} c_0 + \delta\eta c_1(1+\eta c_1) + [c_2+\rho(1-\rho)]\,N_t + (c_1\gamma + 2c_1^2\eta\gamma)\,F_t & \delta\eta^2 c_1 + 2\eta\gamma c_1 F_t \\ \delta\eta^2 c_1 + 2\eta\gamma c_1 F_t & \delta\eta^2 + 2\eta\gamma F_t \end{pmatrix}.$$

where E_t *and* V_t *denote conditional expectation and variance given the past* $(\underline{N_t}, \underline{F_t})$ *of the joint process.*

The conditional means, variances and covariances are linear functions of N_t and F_t since the joint process (N_t, F_t) is CaR(1) (see Proposition 4).

Let us now prove Proposition 5. The unconditional moments of process (N_t, F_t) are derived from the conditional moments in Lemma A.1 by applying the Law of Iterated Expectation and the variance decomposition formula. Specifically, from Lemma A.1 and the Law of Iterated Expectation we get:

$$E[F_{t+1}] = E\left[E_t(F_{t+1})\right] = \delta\eta + \gamma E[F_t].$$

By stationarity we have $E[F_{t+1}] = E[F_t]$, and we get:

$$E[F_t] = \frac{\delta\eta}{1-\gamma}, \tag{A.2}$$

[see also Gourieroux, Jasiak (2006) for the unconditional moments of the ARG process]. When $\eta = (1-\gamma)/\delta$, we get $E[F_t] = 1$. Then, from Lemma A.1 and $\delta\eta = 1-\gamma$:

$$E[N_{t+1}] = E[E_t[N_{t+1}]] = c_0 + \delta\eta c_1 + E[N_t](c_2+\rho) + \gamma c_1 E[F_t] = c_0 + c_1 + E[N_t](c_2+\rho),$$

which yields:

$$E[N_t] = \frac{c_0 + c_1}{1-(c_2+\rho)}.$$

Let us now consider the unconditional variance of F_t. From Lemma A.1 and the variance decomposition formula, we get:

$$\begin{aligned} V[F_{t+1}] &= V\left[E_t(F_{t+1})\right] + E\left[V_t(F_t)\right] = V[\delta\eta + \gamma F_t] + E[\delta\eta^2 + 2\eta\gamma F_t] \\ &= \gamma^2 V[F_t] + \delta\eta^2 + 2\frac{\delta\eta^2\gamma}{1-\gamma} = \gamma^2 V[F_t] + \delta\eta^2\frac{1+\gamma}{1-\gamma}, \end{aligned}$$

which yields:

$$V[F_t] = \frac{1}{1-\gamma^2}\delta\eta^2\frac{1+\gamma}{1-\gamma} = \frac{\delta\eta^2}{(1-\gamma)^2} = \frac{1}{\delta}. \tag{A.3}$$

Let us now consider the unconditional covariance between F_t and N_t. We have:

$$\begin{aligned} Cov(F_{t+1}, N_{t+1}) &= E\left[Cov_t(F_{t+1}, N_{t+1})\right] + Cov\left(E_t[F_{t+1}], E_t[N_{t+1}]\right) \\ &= E\left[\delta\eta^2 c_1 + 2\eta\gamma c_1 F_t\right] + Cov\left(\delta\eta + \gamma F_t, c_0 + \delta\eta c_1 + N_t(c_2+\rho) + \gamma c_1 F_t\right) \\ &= \delta\eta^2 c_1\frac{1+\gamma}{1-\gamma} + \gamma^2 c_1 V[F_t] + \gamma(c_2+\rho)Cov(F_t, N_t) \\ &= \frac{c_1}{\delta} + \gamma(c_2+\rho)Cov(F_t, N_t), \end{aligned}$$

which yields:

$$Cov(F_t, N_t) = \frac{c_1}{\delta} \frac{1}{1-\gamma(c_2+\rho)}. \tag{A.4}$$

Finally, let us consider the unconditional variance of N_t. We have:

$$\begin{aligned}
V[N_{t+1}] &= E\left[V_t(N_{t+1})\right] + V\left[E_t(N_{t+1})\right] \\
&= E\left[c_0 + \delta\eta c_1(1+\eta c_1) + N_t(c_2+\rho(1-\rho)) + F_t(c_1\gamma + 2c_1^2\eta\gamma)\right] \\
&\quad + V\left[c_0 + \delta\eta c_1 + N_t(c_2+\rho) + \gamma c_1 F_t\right] \\
&= c_0 + \delta\eta c_1(1+\eta c_1) + \frac{c_2+\rho(1-\rho)}{1-(c_2+\rho)}(c_0+c_1) + c_1\gamma + 2c_1^2\eta\gamma \\
&\quad + (c_2+\rho)^2 V[N_t] + \gamma^2 c_1^2 V[F_t] + 2(c_2+\rho)c_1\gamma Cov(N_t, F_t) \\
&= c_0 + \delta\eta c_1(1+\eta c_1) + \frac{c_2+\rho(1-\rho)}{1-(c_2+\rho)}(c_0+c_1) + c_1\gamma + 2c_1^2\eta\gamma \\
&\quad + (c_2+\rho)^2 V[N_t] + \frac{\gamma c_1^2}{\delta}\left[\gamma + \frac{2(c_2+\rho)}{1-\gamma(c_2+\rho)}\right].
\end{aligned}$$

We deduce:

$$\begin{aligned}
V[N_t] &= \frac{1}{1-(c_2+\rho)^2}\left\{c_0 + \delta\eta c_1(1+\eta c_1) + \frac{c_2+\rho(1-\rho)}{1-(c_2+\rho)}(c_0+c_1) + c_1\gamma + 2c_1^2\eta\gamma \right. \\
&\quad \left. + \frac{\gamma c_1^2}{\delta}\left[\gamma + \frac{2(c_2+\rho)}{1-\gamma(c_2+\rho)}\right]\right\}.
\end{aligned}$$

By using $\eta = (1-\gamma)/\delta$ and rearranging terms, we get the formula for $V[N_t]$ given in Proposition 5.

A.3.3 Proof of Proposition 6

Let $Z_t = (N_t, F_t)'$. Then, from Lemma A.1 we get $Cov(Z_{t+h}, Z_t) = A^h V(Z_t)$, for $h \geq 0$, where the matrix A is given by :

$$A = \begin{pmatrix} c_2+\rho & \gamma c_1 \\ 0 & \gamma \end{pmatrix}.$$

Now, for a generic triangular $(2,2)$ matrix we have:

$$\begin{pmatrix} a & b \\ 0 & c \end{pmatrix}^h = \begin{pmatrix} a^h & b\left(a^{h-1} + ca^{h-2} + ... + c^{h-2}a + c^{h-1}\right) \\ 0 & c^h \end{pmatrix}.$$

Thus, we get:

$$A^h = \begin{pmatrix} (c_2+\rho)^h & b(h) \\ 0 & \gamma^h \end{pmatrix},$$

where:

$$b(h) = \gamma c_1 \left[(c_2+\rho)^{h-1} + \gamma (c_2+\rho)^{h-2} + ... + \gamma^{h-2}(c_2+\rho) + \gamma^{h-1} \right].$$

When $\gamma = 0$, we have $b(h) = 0$, which implies $Cov(N_{t+h}, N_t) = (c_2+\rho)^h V(N_t)$ and thus $Corr(N_{t+h}, N_t) = (c_2+\rho)^h$. Let us now consider the case $\gamma > 0$. Then:

$$\begin{aligned} b(h) &= c_1 \gamma^h \left[1 + \frac{c_2+\rho}{\gamma} + \left(\frac{c_2+\rho}{\gamma} \right)^2 + ... + \left(\frac{c_2+\rho}{\gamma} \right)^{h-1} \right] \\ &= c_1 \gamma^h \frac{1 - \left(\frac{c_2+\rho}{\gamma} \right)^h}{1 - \frac{c_2+\rho}{\gamma}} = c_1 \gamma \frac{\gamma^h - (c_2+\rho)^h}{\gamma - (c_2+\rho)}, \end{aligned}$$

if $\gamma \neq c_2 + \rho$, and:

$$b(h) = c_1 h \gamma^h,$$

if $\gamma = c_2 + \rho$. It follows:

$$Cov(N_{t+h}, N_t) = (c_2+\rho)^h V(N_t) + b(h) Cov(N_t, F_t),$$

and:

$$Corr(N_{t+h}, N_t) = (c_2+\rho)^h + b(h) \frac{Cov(N_t, F_t)}{V(N_t)}.$$

The conclusion follows.

A.3.4 Conditional dispersion

From the Law of Iterated Expectation and the variance decomposition formula we have:

$$\begin{aligned} E[N_{t+1}|N_t] &= E[E(N_{t+1}|N_t, F_t)|N_t], \\ V[N_{t+1}|N_t] &= E[V(N_{t+1}|N_t, F_t)|N_t] + V[E(N_{t+1}|N_t, F_t)|N_t]. \end{aligned}$$

From Lemma A.1, we get:

$$E[N_{t+1}|N_t] = c_0 + \delta\eta c_1 + (c_2+\rho) N_t + \gamma c_1 E[F_t|N_t],$$

and:

$$V[N_{t+1}|N_t] = c_0 + \delta\eta c_1 (1+\eta c_1) + [c_2 + \rho(1-\rho)] N_t + \left(c_1\gamma + 2c_1^2 \eta\gamma \right) E[F_t|N_t] + \gamma^2 c_1^2 V[F_t|N_t].$$

By using $\eta = (1-\gamma)/\delta$, equation (4.3) follows.

A.3.5 Proof of Lemma A.1

The conditional moments are derived from the expansion of the log conditional Laplace transform in Proposition 4 around $u = v = 0$. At second order in u, v, we have:

$$\begin{aligned}
\log \psi_{1t}(u,v) &= -c_0[1-\exp(-u)] - \beta\big(v + c_1[1-\exp(-u)]\,\big) \\
&\quad -N_t\,\big(c_2[1-\exp(-u)] - \log[1-\rho+\rho\exp(-u)]\,\big) - F_t\,\alpha\big(v + c_1[1-\exp(-u)]\,\big) \\
&\simeq -c_0(u-u^2/2) - \beta\big(v + c_1[u-u^2/2]\,\big) \\
&\quad -N_t\,\big(c_2[u-u^2/2] - \log[1-\rho u+\rho u^2/2]\,\big) - F_t\,\alpha\big(v + c_1[u-u^2/2]\,\big) \\
&\simeq -c_0(u-u^2/2) - \beta\big(v + c_1[u-u^2/2]\,\big) \\
&\quad -N_t\,\big(c_2[u-u^2/2] + \rho u - \rho(1-\rho)u^2/2\,\big) - F_t\,\alpha\big(v + c_1[u-u^2/2]\,\big). \qquad \text{(A.5)}
\end{aligned}$$

By using the expansions at second order of functions α and β:

$$\alpha(u) = \frac{\gamma u}{1+\eta u} \simeq \gamma u - \gamma\eta u^2, \quad \beta(u) = \delta\log(1+\eta u) \simeq \delta\eta u - \frac{1}{2}\delta\eta^2 u^2,$$

we get:

$$\begin{aligned}
\alpha\big(v + c_1[u-u^2/2]\,\big) &\simeq \gamma(v + c_1 u - c_1 u^2/2) - \gamma\eta(v+c_1 u)^2, \\
\beta\big(v + c_1[u-u^2/2]\,\big) &\simeq \delta\eta\left(v + c_1 u - c_1 u^2/2\right) - \frac{1}{2}\delta\eta^2\,(v+c_1 u)^2\,. \qquad \text{(A.6)}
\end{aligned}$$

By replacing expansions (A.6) into expansion (A.5), and gathering the terms proportional to u, v, u^2, v^2 and uv, we get:

$$\begin{aligned}
\log \psi_{1t}(u,v) \simeq\ & -\left[c_0 + \delta\eta c_1 + N_t(c_2+\rho) + \gamma c_1 F_t\right] u - \left[\delta\eta + \gamma F_t\right] v \\
& + \frac{1}{2}\left[c_0 + \delta\eta c_1(1+\eta c_1) + N_t(c_2 + \rho(1-\rho)) + F_t(c_1\gamma + 2c_1^2\eta\gamma)\right] u^2 \\
& + \frac{1}{2}\left[\delta\eta^2 + 2\eta\gamma F_t\right] v^2 + \left[\delta\eta^2 c_1 + 2F_t\eta\gamma c_1\right] uv.
\end{aligned}$$

The conclusion follows.