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Uncertainty Due to Infectious Diseases and Stock–Bond Correlation

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Abstract: We study the non-linear causal relation between uncertainty-due-to-infectious-diseases and stock–bond correlation. To this end, we use high-frequency 1-min data to compute daily realized measures of correlation and jumps, and then, we employ a nonlinear Granger causality test with the use of artificial neural networks so as to investigate the predictability of this type of uncertainty on realized stock–bond correlation and jumps. Our findings reveal that uncertainty-due-to-infectious-diseases has significant predictive value on the changes of the stock–bond relation.

Keywords: artificial neural networks; Granger causality test; nonlinearity; uncertainty; infectious diseases; stock–bond correlation

JEL Classification: C45; C53; C58; G01; G11

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1. Introduction

Globalization has heightened the attention being paid to the international movements of people, goods, and information (Cash and Narasimhan 2000). In addition to expanding travel and trade, such massive movements accelerate the scale and speed of the transmission of infectious diseases. Indeed, the emergence of at least 30 new diseases has been witnessed during the last two decades, many of which are thought to be rapidly spread across countries (see, The world health report: Fighting disease, fostering development. Geneva, World Health Organization 1996). Two illustrative examples of the dispersal of pathogens in the contemporary globalized world are Human Immunodeficiency Virus Infection and Acquired Immunodeficiency Syndrome (HIV/AIDS) and the Severe Acute Respiratory Ayndrome Coronavirus 2 (SARS CoV-2) pandemic. Notwithstanding advanced biotechnology in (i) vaccines, (ii) diagnostics, (iii) therapeutics, and (iv) infection control measures, contagious diseases form the main cause of morbidity and mortality in a great number of areas. According to the Global Burden of Disease Study 2013, out of 2.5 billion disability-adjusted life-years provoked by the totality of diseases, 520 million disability-adjusted life-years were ascribed to contagious diseases in 2013 on a global scale (Murray et al. 2015). The abovementioned diseases, as well as tuberculosis, made up 5 of the top 12 causes of death internationally (Feigin 2016). Lately, 2,176,159 deaths due to coronavirus disease (COVID-19) have been reported to the World Health Organization (WHO) (reported in World Health Organization (WHO) Coronavirus Disease (COVID-19) Dashboard on 29 December 2020.) Moreover, antimicrobial resistance has rendered many formerly efficient therapies outdated, which, in conjunction with the slow-paced clinical evolution of new therapies, leads to a smaller number of treatment choices for several infections. It is hard to estimate and forecast the burden of antimicrobial resistance. It

might as well bring about at least 700,000 deaths annually. Still, this number could rise to 10 million deaths by 2050, and a decrease in the gross domestic product (GDP) of 2% to 3.5% can be observed in the absence of any measures to curtail such a menace (O'Neill 2014).

Epidemics can be linked to an abrupt soaring growth in morbidity and mortality in case health systems are insufficiently prepared while facing exorbitant costs due to a drop in economic transactions. Essentially, this stems from a self-protective mechanism of consumers, such as evading travel and the consumption of specific commodities, and the expectation of comparable changes in demand from the part of producers (Keogh-Brown et al. 2010). Several studies estimate that the global macro-economic effect of SARS is around USD 30–100 billion, or around USD 3–10 million per case (Hai et al. 2004; Hanna and Huang 2004; Lee and McKibbin 2004). Between 0.5% and 1.0% of the UK's GDP is estimated to be the impact related to Influenza A (H1N1) according to Smith et al. (2009). Owing to the COVID-19 pandemic, which was first reported in China in December 2019, except for the severe preoccupations on the remarkably rising casualties, the countries affected are required to grapple with grave and critical repercussions in all facets of daily life, ranging from economic recession to local and global curfew and lockdown.

The prior literature on contagious diseases has taken into consideration many concerns of great importance, which have affected modeling strategies in such a way that does not elucidate the overall structure of private choice and subsequent externalities in disease transmission (see Brito et al. 1991; Francis 1997; Geoffard and Philipson 1996; Kremer 1996; Ferguson et al. 2020, among others). Recently, Altig et al. (2020) studied a number of economic uncertainty indicators for the US and UK prior to and during the COVID-19 pandemic. They established huge uncertainty jumps shown by all indicators invariably in response to the pandemic and its economic fallout. Additionally, employing a data sample on internet searches and two representative surveys from the US, Fetzer et al. (2020) confirmed an upsurge in economic anxiety during and following the coronavirus outbreak. In citing a great diffusion of beliefs related to the pandemic risk factors of coronavirus, they showed a causal effect of such beliefs on market participants' economic anxieties. Yet, empirical evidence is still lacking when it comes to the potential of the uncertainty linked to epidemic and pandemic diseases to the economy and, in particular, to financial markets (see Meltzer et al. 1999; Bonds et al. 2010; Goenka and Liu 2012; Goenka et al. 2014; Brown et al. 2020; Corbet et al. 2020; Dontoh et al. 2020; Falato et al. 2020; Fetzer et al. 2020; Gates 2020; Goenka and Liu 2020; Ji et al. 2020; Sharif et al. 2020; Mavragani 2020; Mavragani and Gkillas 2020; Rubbaniy et al. 2020; Wu et al. 2020; Zhang et al. 2020; Zhu et al. 2020; Alghalith et al. 2021; Bouri et al. 2021; Guo et al. 2021, among others). Our goal is to analyze the externalities of infectious diseases on financial markets, focusing on realized correlation and jumps in realized correlation series (jumps, hereafter). Correlation is a key factor for understanding the dependence structure between assets. In the existing literature, there are several approaches to estimate correlation. The correlation literature has put in the spotlight realized covariances and correlations, since Andersen et al. (2001a) and Andersen et al. (2001b) established the realized correlation measure. The realized covariation matrix can construct the realized correlation, with the latter being modeled with the use of time-series techniques. Realized correlation measuring constitutes one of the handiest and most potent approaches to effectively integrate intra-day data with multivariate volatility estimation and forecasting. There has been a great challenge in whether a correlation series is a continuous process or not. It is of the essence that the literature lays more emphasis on jumps in this sequential movement of two-time series. The occurrence of such jumps entails major implications for risk management, diversification benefits, and asset allocation. These jumps induce the underestimation of the credit default probability of correlation since they decrease the accuracy of any model with respect to estimation, prediction, and forecasting correlation. This is explicable mainly because such models assume Gaussian conditional distributions. Jumps in correlation series are dissimilar to co-jumps (the synchronous jumps in volatility series of two assets are defined as co-jumps. Novotný and Urga (2018) were the first to study the notion of

co-jump, liming theory and the discrete sample properties of co-jumps), even when they aim at disclosing the breaks in the relation of volatilities between two assets. Additionally, Caporin et al. (2017) sought out systemic events influencing variances and correlations.

Here, we focus our interest on the relation between stock and bonds. It is a well-known fact that over time this relation is subject to changes. Even when making allowances for the changing correlation due to stock and bond market time-varying volatility, there was still a significant change in the conditional correlation between the US stocks and bonds during the post-war period (Scruggs and Glabadanidis 2003). Gaining insight into the time variation in the stock–bond correlation ensures essential repercussions for asset allocation and risk management since they form the two most important asset classes (Connolly et al. 2005; d'Addona and Kind 2006; Kim et al. 2006). The exploration of the various economic driving forces of the time-varying stock–bond correlation has been endeavored by several studies. In particular, Connolly et al. (2005, 2007) established a reduction in the future stock–bond correlation at higher daily frequency, parallel to rising stock market uncertainty in the US in addition to some more major markets, possibly owing to the flight-to-quality phenomenon. Kim et al. (2006) corroborated the comparable role of stock market uncertainty along these lines in a great number of European markets. Shedding more light on the economic drives which trigger the stock–bond correlation at lower, namely monthly, frequencies, Li (2002) claimed that not only the anticipated inflation uncertainty but also the real interest rate uncertainty tend to raise the correlation between stock and bond returns. On the contrary, d'Addona and d'Addona and Kind (2006) argued that the inflation volatility is inclined to decrease the correlation, despite the fact that the volatility of real interest rates may lead to a rise in the stock–bond correlation in G-7 countries.

Moreover, Boyd et al. (2005) and Andersen et al. (2007a) studied the impact of macroeconomic news announcements on stock and bond markets in periods of expansion and recession. They claimed that the cashflow effect is quite prevalent in the event of contractions, and they also stressed the significance of the discount rate effect in the event of expansions, thereby leading to positively correlated stock and bond returns during expansions and lower, possibly negative, correlations in the event of recessions. In the same vein, Ilmanen (2003) made a similar proposal. Nevertheless, Jensen and Mercer (2003) found a lower monthly correlation between stocks and bonds in periods of expansion than in recessions (despite this difference being statistically significant exclusively for small-cap stocks and not for large-cap stocks), and this finding is fundamentally incompatible with the flight-to-quality assertion. Furthermore, whether the inflation volatility results in a rise or a fall in the stock–bond correlation has yet to be established. Therefore, a great deal of controversy surrounds the issue of the way variable macroeconomic conditions, specifically at lower frequencies, determine the time variation in the stock–bond correlation.

In this study, the correlation between two-time series is thus depicted by estimating the realized correlation between these series. We also emphasize the detection of the presence of jumps (as breaks or discontinuities) in a continuous correlation series as well as the magnitude of jumps, showing the realized breaks (jumps) in correlation series, as identified directly in the correlation (see Gkillas et al. 2019b). A significant jump found in a realized correlation series will have a direct effect on the degree of relation. In particular, we use intraday data to compute daily realized correlation and jumps series between the stock and the bond to study whether uncertainty caused by infectious diseases can predict movements in the relation in the variables of interest. However, the first step which needs to be taken is to quantify the uncertainty linked to contagious diseases in such a manner that it functions as a proper input into an econometric model for predicting changes in the correlation between stock and bond. To this end, we make use of the uncertainty-due-to-infectious-diseases index proposed by Baker et al. (2020). For the analysis, we employ a nonlinear test of causality using artificial neural networks to discover non-linear relations between stock–bond realized correlation and jumps with the uncertainty-due-to-infectious-

diseases. Our findings reveal that uncertainty-due-to-infectious-diseases has significant predictive value on the changes of the stock–bond relation.

The rest of the paper is organized as follows: Section 2 reviews the relevant literature with regard to the time-varying nature of correlation. Section 3 presents the data used in this study. Section 4 discusses the econometric methods used. Section 5 provides the empirical results. Section 6 concludes the paper.

2. Relevant Literature

In the existing literature, there are two methods to estimate the correlation: (i) parametrically and (ii) non-parametrically. On the one hand, the Dynamic Conditional Correlation—Generalized Autoregressive Conditional Heteroskedasticity (DCC-GARCH) model is characterized as the best parametric estimation method. Many studies have used DCC-type models to investigate the nature of time-varying correlation and its drivers (see Tao and Green 2012; Lean and Teng 2013; Canh et al. 2019; Shiferaw 2019; Ghosh et al. 2020, among others). However, parametric or semi-parametric correlation estimations, the DCC-GARCH (see Missio and Watzka 2011; Aielli 2013), BEKK (named byYoshi Baba, Dennis Kraft and Ken Kroner) (Engle and Kelly 2012), and Markov-Switching Vector Autoregressive (VAR) models (see, e.g., Casarin et al. 2018) are employed as heavily parameterized models primarily for prediction instead of estimation. Furthermore, McAleer (2019) strongly criticized the DCC method, which encompasses algebraic non-existence, mathematical irregularity and non-asymptotic properties (see also Bouri et al. 2020).

The evidence above further motivates the use of realized correlation estimators. Indeed, realized correlation is regarded as an unconditional, assumptions-free measure of latent correlation (see Gkillas et al. 2018). The asymptotic distribution of the correlation is approximated directly by the realized correlation coefficient (Barndorff-Nielsen and Shephard 2004). Being constructed as quotients between realized covariances and products of realized standard deviations, realized correlations lead to a considerable improvement in the accuracy and performance of asset allocation, portfolio risk management and trading strategies (Audrino and Corsi 2010). The feasibility of predicting both in-sample and out-of-sample realized correlations is raised after the construction of realized correlations, low-parameterized and mostly linear models.

With an already small number of studies exploring the influence of realized correlation by economic/financial variables (see Christiansen and Ranaldo 2007; Aslanidis and Christiansen 2014; Aït-Sahalia and Xiu 2016; Bonato 2019; Gorgi and Koopman 2020; Demirer et al. 2020), our study departs from this body of research and aims to add to the literature on the drivers of realized correlation and jumps, investigating for the first time the predictive power of uncertainty due to infectious diseases. More specifically, Christiansen and Ranaldo (2007) studied the effects of macroeconomic announcements on the realized correlation between bond and stock returns, concluding that announcement effects are highly dependent on the sign of the realized bond–stock correlation. Aslanidis and Christiansen (2014) explored the impact of macro-finance variables on stock–bond correlation and found evidence that macro-finance variables are most useful in explaining stock–bond correlation. In particular, stock market variables and credit risk variables have a major impact on the correlation. In terms of the relationship now between correlation jumps and uncertainty, Aït-Sahalia and Xiu (2016) studied the informational content of co-jumps and pointed out that they often occur in response to the resolution of policy uncertainty. Demirer et al. (2020) studied the effect of non-cash flow factors over realized correlation jumps in financial markets and found evidence that non-cash flow factors can drive realized correlation jumps. As for stock returns correlation, Gorgi and Koopman (2020) analyzed realized correlations for several sets of stock returns. They found that the impact of past values of realized correlation on future values is at least 10% higher when stock returns are negative rather than positive. In the same vein, Bonato (2019) investigated realized correlations between oil and a set of commodities (i.e.,

agricultural commodities) and established that an increase in volatility transmission tends to precede the increase in correlations.

In the context of our analysis, the relation between stock and bonds is thus depicted by estimating realized correlation and correlation jumps between these series. A significant jump found in realized correlation will, in turn, have a direct effect on the degree of relation between stock and bonds. Therefore, it is economically important to proceed to a better econometric understanding of the time-varying behavior of realized correlation and its properties, namely jumps, in accordance with their drivers.

3. Data

Data concern stock Standard & Poor's 500 futures index (S&P 500 futures index) and T-bond futures markets (T-Bond 30-year futures) covering a sample period from 8 July 2002 to 28 August 2015, with a sampling frequency of 1-min. We use futures data, as futures price data have lower transaction costs linked to futures trading. Futures prices rather than spot prices constitute the focal point of our study, which is why this work is of interest for analysts and has practical applications in the framework of diversification and hedging. Boasting readiness for short selling and lower transaction costs, futures prices tend to have a more rapid response to new information than spot prices, revealing that it is in the futures market where price discovery can occur first and foremost (see, e.g., Demirer et al. 2018). The data were retrieved from the Pi-Trading Inc database (https://pitrading.com/, accessed on 3 December 2020). This sample period incorporates different market phases, such as major booms and crashes (e.g., the global financial crisis of 2008 and the European sovereign debt crisis). In contrast to the daily basis, high-frequency data reveal important information, such as intraday changes and market microstructures. According to Hansen and Huang (2016), daily realized volatility is more accurately estimated by employing high-frequency data. Realized volatility is a key element for constructing realized measures of correlation (see Section 4). Intraday data prove to be highly useful when estimating the transmission of volatility spillovers (see, e.g., Wu et al. 2005). In the following stage, and prior to the construction of the realized measures of interest, we resorted to several changes in the whole dataset of the raw intraday series. This is a common procedure when we are concerned with intraday data adopted by several studies, such as the work of Barndorff-Nielsen et al. (2009). To this effect, initially, we take into consideration overlapping trading hours exclusively. Secondly, we proceed to the exclusion of fixed and moving holidays, Christmas and New Year's Day included, as well as thin trading days (namely, days whose trading hours failed to cover the observation time window entirely) from the sample. Subsequently, we eliminate days with infrequent trades (less than 100 transactions at a 1-min time interval) from the sample (the trading of futures contracts on the CME Globex exchange is affected almost 24 h a day, from Sunday afternoon to Friday afternoon). The final total number of trading days under examination is 3964. Finally, in order to construct daily point estimates of the realized measures of interest (i.e., realized correlation and jumps in realized correlation), we make use of intraday 1-min log-returns—evidence reported in the existing literature recommends that the computation of intraday returns should be made at the highest frequency possible so that there is asymptotic convergence of volatility estimators towards the true conditional volatility following fixed domain asymptotics. The sampling frequency should be neither too high to trigger spurious jumps on account of market frictions nor too low to bring about poor data analysis (see Andersen and Bollerslev 1997, 1998; Taylor and Xu 1997). Therefore, we opt for the highest sampling frequency, since it is the optimal sampling frequency which diminishes the autocovariance bias (see Oomen 2001; Markellos et al. 2003; Oomen 2004; Degiannakis and Floros 2016; Degiannakis and Filis 2017; Sévi 2014; Gkillas et al. 2020, among others). The 1-minute frequency is eligible and plausibly adjacent to the optimum one, thus being regarded as the optimal sampling frequency in this empirical analysis.

Now, we focus our attention on the uncertainty-due-to-infectious—this measure of uncertainty is available for free to the public on a daily basis, while it can be retrieved

from http://policyuncertainty.com/infectious_EMV.html (accessed on 3 December 2020). Baker et al. (2020) developed this measure using a newspaper-based infectious disease Equity Market Volatility (EMV) tracker, available at a daily frequency from January 1985. In the development of Uncertainty-Due-To-Infectious-Diseases (EMVID), Baker et al. (2020) coined four sets of terms, namely, E: economic, economy, financial; M: stock market, equity, equities, Standard and Poors; V: volatility, volatile, uncertain, uncertainty, risk, risky; ID: epidemic, pandemic, virus, flu, disease, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) or coronavirus, middle east respiratory syndrome (MERS), severe acute respiratory syndrome (SARS), Ebola virus disease (EVD), highly pathogenic Asian avian influenza A (HPAI Asian H5N1) virus, influenza A virus subtype H1N1 (A/H1N1). Subsequently, daily counts of newspaper articles involving at least one term in each of E, M, V, and ID across approximately 3000 US newspapers are retrieved. Next, the raw EMVID counts account for the scaling of the count of all articles on the same day. In the end, Baker et al. (2020) proceed to the multiplicative rescaling of the series ensued to align the level of the CBOE Volatility Index (VIX), making use of the overall EMV index. Afterward, the scaling of the EMVID index is implemented to depict the ratio of the EMVID articles to total EMV articles. Considering the data availability of the two variables under investigation, our analysis spans the sample period of 8 July 2002 to 28 August 2015.

Table 1 reports basic statistics of realized stock–bond correlation, jumps, and the uncertainty-due-to-infectious-diseases. All series under consideration are on a daily basis. As we can see from this table, the mean of the realized stock–bond correlation series is equal to 0.3003, with a standard deviation equal to 0.6013. The skewness is positive equal to 0.6378, indicating that the tail of the right side of the distribution is longer. Additionally, the kurtosis is equal to 2.0587. As for the series of jumps, the mean is equal to 0.0660, with a standard deviation equal to 0.0396. The skewness is now positive and equal to 0.7744, and as expected the series is skewed to the right. Additionally, the kurtosis is equal to 4.5930, which further implies that the data exhibit heavy-tailed behavior or profusion of outliers. As for the uncertainty-due-to-infectious-diseases series, the mean is equal to 0.5466, with a standard deviation equal to 0.3100. The skewness is now positive and equal to 11.0101. Last but not least, the null hypothesis of normality is tested by the Jarque–Bera test in all series under consideration. In all cases, the null hypothesis of normality is rejected at a 1% significant level (the time-series of realized correlation and jump series are implicitly assumed to be stationary. Formal tests for a unit root in $RC_t^{(m)}$, $RCJ_t^{(RC)}$, and EMVID series easily reject the null hypothesis of non-stationarity).

Table 1. Basic statistics for realized correlation, realized correlation jumps and uncertainty-due-to-infectious-diseases index.

	Realized Correlation	**Realized Correlation Jumps**	**Uncertainty-Due-To-Infectious-Diseases**
	$RC_t^{(m)}$	$RCJ_t^{(RC)}$	EMVID
Mean	−0.3003	0.0660	0.5466
Median	−0.4933	0.0646	0.3100
Maximum	0.9903	0.3290	36.000
Minimum	−0.9989	0.0000	0.0000
Std. Dev.	0.6013	0.0396	1.1666
Skewness	0.6378	0.7744	11.0101

Table 1. *Cont.*

	Realized Correlation	Realized Correlation Jumps	Uncertainty-Due-To-Infectious-Diseases
Kurtosis	2.0587	4.5930	248.9896
J-B	423.7032 ***	815.4169 ***	10,282,870 ***
J-B Prob.	[0.0000]	[0.0000]	[0.0000]
Obs	3964	3964	3964

Notes: This table reports descriptive statistics for jumps in realized stock–bond correlation, jumps in realized correlation (in absolute values), and the uncertainty-due-to-infectious-diseases series. The following statistics are given: mean, median, maximum, minimum, standard deviation (Std. Dev), skewness, kurtosis, Jarque–Bera normality test (J-B), and the number of the observations. The null hypothesis that the series is normally distributed is also tested by the Jarque–Bera test. The *p*-values of the test are given below in brackets. *** indicates a rejection of the null hypothesis of normality at a 1% significance level.

4. Methods

This section describes in detail the non-parametric estimators of daily realized stock–bond correlation used, the detection scheme employed to detect daily jumps in realized stock–bond correlation, as well as the nonlinear causality method to discover complex non-linear causal relations from the uncertainty-due-to-infectious-diseases to realized stock–bond correlation and/or to jumps in realized stock–bond correlation. Previous studies on stock–bond correlation are mostly based on a parametric framework. Nonetheless, the non-parametric framework is a more natural way to estimate these series in terms of the strength of the data.

4.1. Realized Correlation Estimators

The literature abounds in the many available realized volatility estimators. The great number of estimators in existence notwithstanding, the realized variance is employed to construct realized correlation series. In what follows, we present the estimator of realized correlation that is used in this section. More specifically, we present the realized covariance estimator used divided by the square roots of the realized variations of the two assets under examination.

To begin with, we assume that for each day, with time confined in the unit interval $[0, 1]$, the observed intraday logarithmic asset prices follow the noise-contaminated process:

$$P_{t,m} = P^*_{t_i,m} + u_{t_i} \tag{1}$$

where p stands for the logarithmic price observed, P^* stands for the unobservable equilibrium logarithmic price, and u corresponds to the unobservable market microstructure noise. The time index t_i accounts for the ith observation in the $m+1$ intraday observations with a sampling frequency equal to $1/m$. The corresponding intraday returns (at the highest frequency of observation) correspond to $X_{i,m} = P_{t_i,m} - P_{t_i-1,m}$ and $X^*_{i,m} = P^*_{t_i,m} - P^*_{t_i-1,m}$. $e_{i,m} = u_{t_i} - u_{t_i-1}$ accounts for the difference of the noise component, and we assume that the equilibrium price evolves as a function of a stochastic volatility process, as follows:

$$P^*_{t_i} = \int_0^{t_i} \sigma_s dW_s + J_{t_i} \tag{2}$$

where σ_t denotes the stochastic volatility process, W_t denotes the standard Brownian motion, and J_J is the component to arise in the price process in the event of discrete jumps. The integrated volatility during a day is given by:

$$V_t = \int_0^1 \sigma_s^2 ds + \lambda_t \tag{3}$$

where $\lambda_t = \sum_{0<s\leq 1} \kappa_s^2$ stands for the part of the jumps into the volatility, while κ_s accounts for the size of the discrete jumps.

Now turning our attention to the case of two assets, their covariance matrix with the generic element $\sum_{a,b}$ is given by:

$$C_t = \int_0^1 \sum_{a,b} ds \tag{4}$$

where a and b are the assets considered (i.e., stock and bond). This covariance is latent. It is latent, and the realized volatility estimator on a multivariate level is considered the most appropriate estimator. The realized covariance and realized correlation estimator were proposed by Barndorff-Nielsen and Shephard (2004). The cross-products of the two 1-min asset return series over each trading day constitute the realized covariance:

$$RC_t^{(m)} = \sum_{i=1}^{m} X_{a,i,m} X_{b,i,m} \tag{5}$$

The theory of quadratic variation points to the probability limit of the correlation. Therefore, as $m \to \infty$, $RC_t^{(m)} \xrightarrow{p} C_t$ (see Jacod and Shiryaev 2003). Andersen et al. (2001b) also made reference to the aforementioned correlation. In the case of noise-free data, $RC_t^{(m)}$ is a consistent estimator of C_t as the sampling frequency increases.

The $RC_t^{(m)}$ divided by the square roots of the realized volatility estimators of the two assets ($RV_{t,a}^{(m)}$ and $RV_{t,b}^{(m)}$) result in the realized correlation emerges from:

$$RC_t^{(m)} = \frac{RC_t^{(m)}}{\sqrt{RV_{t,a}^{(m)}}\sqrt{RV_{t,b}^{(m)}}} = \frac{\sum_{i=1}^{m} X_{a,i,m} X_{b,i,m}}{\sqrt{\sum_{i=1}^{m} X_{a,i,m}^2}\sqrt{\sum_{i=1}^{m} X_{b,i,m}^2}} \tag{6}$$

where RV is constructed by the summation of intraday returns, and it has rapidly gained ground as a measure of daily volatility. According to Andersen et al. (2001b), when the length of the intra-day intervals goes to zero, RV is convergent to the true underlying integrated variance, proving to be an unbiased and highly efficient estimator (see also Barndorff-Nielsen and Shephard 2002).

Except for the realized correlation estimator, in this study, we make use of an additional estimator, namely the median realized variation correlation ($RC_t^{(MRV)}$). Both of them are derived from the same realized covariance estimate as to the realized correlation. This estimator differs from the realized correlation in the denominator, with the $RC_t^{(MRV)}$ being as follows:

$$\begin{aligned} RC_t^{(MRV)} &= \frac{RC_t^{(m)}}{\sqrt{RV_{t,a}^{(MRV)}}\sqrt{RV_{t,b}^{(MRV)}}} \\ &= \frac{\sum_{i=1}^{m} X_{a,i,m} X_{b,i,m}}{\sqrt{\lambda \sum_{i=1}^{m-1} med\left(|X_{a,i-1,m}|, |X_{a,i,m}|, |X_{a,i+1,m}|\right)^2}\sqrt{\lambda \sum_{i=1}^{m-1} med\left(|X_{b,i-1,m}|, |X_{b,i,m}|, |X_{b,i+1,m}|\right)^2}} \end{aligned} \tag{7}$$

where $\lambda = \left(\frac{\pi}{6-4\sqrt{3}+\pi}\right)\left(\frac{m}{m-2}\right)$. For further discussion on the median realized variance (MRV) estimator, see Andersen et al. (2012), while it is named as median realized variation correlation ($RC_t^{(MRV)}$). As we can from the previous equation, the $RC_t^{(MRV)}$ is equal to the standard realized covariance ($RC_t^{(m)}$) (as established by Andersen et al. 2001) divided by the square root of the MRV of the two assets ($RV_{t,a}^{(MRV)}$ and $RV_{t,b}^{(MRV)}$). MRV is one of the best alternative jump-robust estimators of realized variance. Specifically, MRV carries greater theoretical efficiency aspects than other variation measures (e.g., bipower variation and/or tripower variation). MRV turns out to be a jump-robust estimator of integrated variance, also considered to be a less biased estimator than other measures of RV in the event of jumps. The MRV estimating process enables the separation of jumps information (the returns variation caused by jumps) from the main model. In comparison

with other realized measures, MRV mitigates the effect of market-microstructure noise, thus displaying better finite sample properties even in the presence of noise and remaining unaffected by the sampling frequency (see also Gkillas et al. 2020; Gkillas et al. 2019a). For brevity, we do not include a table for the $RC_t^{(MRV)}$ measure. Results were qualitatively similar to those reported in the next section and are available upon request.

4.2. Jumps in Realized Correlation

The correlation-jump detection scheme employed in this study is based on a standard "realized volatility jump" detection scheme. Such schemes can also be employed for detecting realized correlation jumps (see also Demirer et al. 2020). More specifically, the jumps detection scheme based on Huang and Tauchen (2005); Andersen et al. (2007b), and Giot et al. (2010) is used to detect jumps in correlations in the following way:

$$RCJ_t^{(RC)} = RC_t \, I[RC_t > C_{1-a}] + RC_t \, I[RC_t < C_a] \tag{8}$$

where the RC_t is any of the realized correlation estimators (i.e., $RC_t^{(MRV)}$ or $RC_t^{(m)}$). The threshold value denoted by C can take different values with respect to α. The value of α used in this study is equal to 0.05. In the detection scheme of jumps in correlation, $RC_t^{(m)}$ is realized correlation series, but it can be also range-based realized correlation and/or realized range correlation estimators (see also Vortelinos 2010; Vortelinos 2011; Gkillas et al. 2018). An alternative jump detection scheme (see, e.g., Demirer et al. 2020) yields similar results. This scheme is described as follows $ZCJ_t^{(MRV)} = \frac{\left(RC_t^{(m)} - RC_t^{(MRV)}\right) RC_t^{(m)-1}}{\sqrt{\left(\xi_1^{-4} + 2\xi_1^{-2} - 5\right) \max\left\{1, RQ_t^{(MRV)} RC_t^{(MRV)-2}\right\}}}$, where $RQ_t^{(MRV)}$ is the integrated quarticity based on MRV, while it is given by $RQ_t^{(MRV)} = \lambda \sum_{i=1}^{m-2} med(|X_{i-1,m}|, |X_{i,m}|, |X_{i+1,m}|)^4$ and shows convergence in probability to integrated quarticity with $ZCJ_t^{(MRV)} \sim N(0,1)$. In this case, a jump is significant in case the test statistic is in excess of the appropriate critical value of the standard normal distribution, represented by Φ_a, at a level of significance; a 5% significance level is used in this study. The jump component ($CJ_t^{(MRV)}$) can be found as $RCJ_t^{(MRV)} = \left|RC_t^{(m)} - RC_t^{(MRV)}\right| I\left[ZCJ_t^{(MRV)} > \Phi_a\right]$, where $I\left[ZCJ_t^{(MRV)} > \Phi_a\right]$ is the indicator function of the $ZCJ_t^{(MRV)}$ statistic in excess of a given critical value of the Gaussian distribution Φ_a.

4.3. Nonlinear Granger Causality Test

Granger causality has been consolidated as a method for detecting causal relations. The implementation of Granger causality over the spectrum is to be effective at measuring the strength and direction of the causality, which could be variable over the frequencies. Granger (1969, 1980) introduced for the first time a spectral-density approach, as a means of illustrating causality in a richer and more concise way than a one-shot Granger causality measure applied throughout all periodicities. Therefore, the measurement of the bivariate Granger causality over the spectrum is more efficient than the one-shot test. In this light, two crucial issues to raise are: (i) the way causality changes with frequency as well as (ii) whether the significance and/or direction of the common Granger causality tests in the domain of time are subject to change once the causality test across frequency bands is adopted. However, Hinich and Patterson (1985), Scheinkman and LeBaron (1989), Brock et al. (1991), and Hsieh (1991), among others, found significant nonlinear dependence in stock returns. In the same vein, Hiemstra and Jones (1994) documented significant nonlinearities in aggregate trading volume. They noted that there was a nonlinear causal relation between stock prices and trading volume.

The problem with the linear approach to causality testing lies in the low power in locating a certain type of nonlinear causal relation. Put differently, linear causality tests, includ-

ing the Granger test, can be unable to reveal nonlinear predictability (Baek and Brock 1992; Hiemstra and Jones 1994). At this point, we should raise two issues connected to the statistical characteristics of the test. The first issue is related to the asymptotic distribution of the test when applied to the residuals of vector autoregression (VAR) models. Baek and Brock (1992) claimed that whether applied to consistently estimated residuals or to the mutually independent and individually independent and identically distributed errors of the maintained VAR, the asymptotic distribution of their variant of the test remains unchanged. Moreover, the above test version is considered to be nuisance-parameter-free (NPF) for such models. A comparable NPF result is expected for the modified Baek and Brock (1992) test employed in this study. Nonetheless, Hiemstra and Jones (1993) record Monte Carlo evidence that bears witness to the robustness of the modified test with regard to nuisance-parameter issues. Correspondence between the asymptotic and finite-sample statistical properties of the modified test, when applied to consistently estimated errors that stand for a given VAR model, was also found.

In order to tackle these challenges, we suggest using a nonlinear test of causality using artificial neural networks (ANNs) following Hmamouche (2020). This test allows us to handle non-linear relations between time series, and we put forward a non-linear extension of the Granger causality test employing feed-forward neural networks. The use of (ANNs) can be of high importance when computing causalities, particularly for a time series that follows a non-linear change in the course of time. We make avail of the features of ANNs and apply an extended version of the Granger causality test using a vector autoregressive neural network (VARNN) model. Prior to the description of this causality test, it is necessary to concisely discuss the VARNN model first. Then, we describe in detail the abovementioned non-linear Granger causality test, using the VARNN model.

Granger Causality Selection on Encoding

Let $x_t \in R$ denote a p-dimensional stationary time series and assume we have observed the process at T time points, $(x_1, \dots x_T)$. Let us also assume a training dataset which includes one target variable x and k predictor variables $\{x_1, \dots, x_k\}$. The VARNN(p) model constitutes a multi-layer perceptron (MLP) neural network model (also referred to as a "vanilla" NN, and it is one of the most common classes of feedforward ANNs) that considers the p lag values of the predictor variables and the target variable (x) so as to predict future values of x. The MLP is based on the "original" perceptron, suggested by Rosenblatt (1958), and Widrow and Hoff (1960). A brief description of the MLP is given as follows. An MLP contains at least three layers of nodes: (i) an input layer, (ii) a hidden layer and (iii) an output layer. Apart from the input nodes, each node corresponds to a neuron which makes use of a nonlinear activation function. MLP utilizes a supervised learning method, the so-called backpropagation for training. We refer to the study implemented by Aitkin and Foxall (2003) for more information regarding the statistical modeling of artificial neural networks employing MLP. We opted for the VARNN model to empower the prediction of each target variable with a certain set of predictors, as target variables do not obligatorily ensure the same predictors with the size of hidden layers equal to two for the univariate model (Equation (10)) and four for the bivariate model (Equation (11)). In the beginning, the model reorganizes the data by having a supervised learning form with regard to the lag parameter. The optimization algorithm employed for the update of the network's weights relies on the stochastic gradient descent (SGD) algorithm with learning rate equal to 0.1. Following Hmamouche (2020), the rectified linear unit (RELU) activation function is used in hidden layers, while the sigmoid function is used in the last layer. Moreover, Kingma and Ba (2015) supported that the Adam optimization algorithm (i.e., an extension to stochastic gradient descent) can find an implementation for updating the learning rate based on the SGD. For our empirical application, the SGD algorithm is used. The global function of the VARNN (p) can be expressed in the following way:

$$x_t = \psi_{nn}\left(x_{t-1}, \dots, x_{t-p}, \dots, x_{k(t-1)}, \dots, x_{k(t-k)}\right) + u_t \tag{9}$$

where ψ_{nm} and u_t stand for the network function and the error terms, respectively.

We focus now on the case of two variables, x_t and y_t. In the same manner, as in Granger causality, we consider two prediction models in order to investigate the causality effect of x_t on y_t; the first model takes into consideration the lag values of the target time series, while the second one takes into consideration the lag values of the target and the predictor time series:

$$y_t = \psi_{1,nn}\left(y_{t-1}, \ldots, y_{t-p}, \ldots, y_{k(t-1)}, \ldots, y_{k(t-k)}\right) + u_{1,t} \tag{10}$$

$$y_t = \psi_{2,nn}\left(y_{t-1}, \ldots, y_{t-p}, x_{t-1}, \ldots, x_{t-p}\right) + u_{2,t} \tag{11}$$

where $\psi_{1,nn}$ and $\psi_{2,nn}$ represent the network functions of the two models consider, respectively. Testing causality entails the use of F-tests to investigate whether lagged information on series x_t offers any statistically significant information about series y_t in the presence of lagged y_t. The F-test is used to test the null hypothesis that the series x_t does not cause series y_t.

It is essential to turn our attention to a crucial point regarding this causality. It is apparent that the computation of causalities by means of ANNs may confer the typical shortcoming of raising the computational time. Indeed, this is inaccurate occasionally, since, in the event of a great number of series, the computation of causalities between all variables is indispensable. Additionally, in the event of relationships between variables that change over time, the recalculation of causalities periodically or following each change is also indispensable. Note that the basic formulations of the conventional causality measures are lacking in further adaptation, which indicates that they do not enable the update of the new values by using the old ones. Conversely, with ANNs, the first computation of causalities may not be quick in comparison with the Granger test. However, in the case of new observations in the time series, the model becomes adaptive even faster on account of the learning features of ANNs. We refer to Hmamouche (2020) for more information regarding this issue.

5. Empirical Results

Now, we discuss the empirical results obtained from the VARNN(p) model to identify the non-linear causal relation between daily realized measures of stock–bond correlation ($RC_t^{(m)}$ and $RCJ_t^{(MRV)}$) used and uncertainty-due-to-infectious-diseases (EMVID). Our initial analysis showed that the standard linear causality test failed to detect any evidence of EMVID causing changes in the stock–bond relation. Complete results are available upon request. The estimation results are reported in Tables 2 and 3 for realized correlation and jumps, respectively.

Table 2 reports the nonlinear Granger causality tests between realized stock–bond correlation and uncertainty-due-to-infectious-diseases for a different number of lags p. In particular, we give the F-values and the p-values of the nonlinear Granger causality tests considering a different number of lags p, where $p = 1, \ldots, 7$. As we can see from this table, the null hypothesis is that the uncertainty due to infectious diseases does not cause realized stock–bond correlation, which is rejected for $p = 4$, at 1% level of significance as the value of the F-test is equal to 2.1018 and the corresponding p-value is equal to 6.51×10^5. The null hypothesis is also rejected for $p = 6$ and $p = 7$. This further implies that past values of the uncertainty due to infectious diseases collectively add explanatory power to the predictability of the stock–bond relation.

Now turning our attention to realized stock–bond correlation jumps and Table 3, the null hypothesis that the uncertainty due to infectious diseases does not cause realized stock–bond correlation is rejected for $p = 6$, at 1% level of significance, unlike the previous case where the null hypothesis was rejected for $p = 4$. The value of the F-test for this number of lags is equal to 1.949 and the corresponding p-value is equal to 5.97×10^5. The null hypothesis is also rejected for $p = 7$.

Table 2. Nonlinear Granger causality test for realized stock–bond correlation and uncertainty-due-to-infectious-diseases.

Lag Parameter	CGI	F-Value
	Granger causality index	
$p = 1$	2.13×10^{-4}	0.0389
		[1.0000]
$p = 2$	2.50×10^{-3}	0.3578
		[0.9992]
$p = 3$	9.37×10^{-3}	1.1062
		[0.3083]
$p = 4$	2.08×10^{-2}	2.1018 ***
		$[6.51\times 10^{-5}]$
$p = 5$	9.16×10^{-3}	0.7954
		[0.8370]
$p = 6$	1.97×10^{-2}	1.5179 ***
		$[9.92 \times 10^{-3}]$
$p = 7$	2.10×10^{-2}	1.4523 **
		[0.0145]

Notes: This table reports the nonlinear Granger causality test between realized stock–bond correlation and uncertainty-due-to-infectious-diseases for a different number of lags p. The maximum number of lags considered is 7 (one week). The F-values and the p-values of the nonlinear Granger causality test are reported. The p-values of the test are given below in brackets. The null hypothesis is that the uncertainty due to infectious diseases does not cause realized stock–bond correlation. ***, ** and * the rejection of the null hypothesis of no-causality at the 1%, 5%, and 10% level, respectively. The quantification of causality is realized with the evaluation of the variances of the errors of Equations (10) and (11). The Granger causality index (GCI) can be computed as $\log\left(\sigma_1^2/\sigma_2^2\right)$.

Table 3. Nonlinear Granger causality test for realized stock–bond correlation jumps and uncertainty-due-to-infectious-diseases.

Lag Parameter	CGI	F-Value
	Granger causality index	
$p = 1$	2.90×10^{-3}	0.5193
		[0.9679]
$p = 2$	4.62×10^{-3}	0.6481
		[0.9218]
$p = 3$	0.0000	-0.2838
		[0.9999]
$p = 4$	4.59×10^{-3}	0.4488
		[0.9989]
$p = 5$	4.37×10^{-3}	0.3704
		[0.9999]
$p = 6$	2.57×10^{-2}	1.9491 ***
		$[5.97 \times 10^{-5}]$
$p = 7$	2.35×10^{-2}	1.5917 ***
		$[3.04 \times 10^{-3}]$

Notes: This table reports the nonlinear Granger causality test between realized stock–bond correlation jumps and uncertainty-due-to-infectious-diseases for a different number of lags p. The maximum number of lags considered is 7 (one week). The F-values and the p-values of the nonlinear Granger causality test are reported. The p-values of the test are given below in brackets. The null hypothesis is that the uncertainty due to infectious diseases does not cause realized stock–bond correlation. ***, ** and * the rejection of the null hypothesis of no-causality at the 1%, 5%, and 10% level, respectively. The quantification of causality is realized with the evaluation of the variances of the errors of Equations (10) and (11). The Granger causality index (GCI) can be computed as $\log\left(\sigma_1^2/\sigma_2^2\right)$.

Trying now to identify the transmission channels of uncertainty in stock–bond correlation, it is important to reiterate that the nonlinear Granger causality runs from the uncertainty-due-to-infectious-diseases into the realized stock–bond correlation, yet, more importantly, the link is primarily in the realized correlation jump (discontinuous) component. Such evidence is highly recommended to investors who tend to rebalance their portfolios in order to adjust to the risk associated with jumps in realized stock–bond

correlation. From a practical perspective, our paper sheds light on the types of events that cause significant changes in the stock–bond relation as we attempt a more systematic characterization of the drivers that cause jumps in realized stock–bond correlation. The drivers of such jumps entail major implications for risk management, diversification benefits, and asset allocation. Although Donadelli et al. (2017) noted that media news on dangerous infectious diseases may have a positive sentiment effect among investors (especially companies which are likely to engage in the development of new vaccines), our study identifies that the disease-related fear does alter any positive climate to investors as it induces a significant underestimation of the credit default probability of stock–bond correlation in a sense that it entails a significant decrease in the accuracy of estimation, prediction, and forecasting correlation.

6. Conclusions

We study the causal relationship between daily stock–bond correlation and uncertainty-due-to-infectious-diseases. In order to uncover the predictability of the uncertainty-due-to-infectious-diseases on the stock–bond relation, we focus on the nonlinear Granger causality test by means of an artificial neural network method. By using the measure of uncertainty developed by Baker et al. (2020) and the realized measures of correlation and jumps for the period that spans from 8 July 2002 to 28 August 2015, we find strong evidence that time variation in the stock–bond relation can be linked to this kind of uncertainty. As noted by Connolly et al. (2005), "characterizing this time variation has important implications for understanding the economics of joint stock-bond price formation and may have practical applications in asset allocation and risk management." Thus, it is economically important to proceed in a better econometric understanding of stock–bond comovements in order to determine whether and how uncertainty-due-to-infectious-diseases is transmitted to financial markets. However, more importantly, our study contributes to the development of a deeper understanding of the types of events causing jumps in realized stock–bond correlation. The drivers of such jumps entail further implications for risk management, diversification benefits, and asset allocation as they decrease the accuracy of any model with respect to estimation, prediction, and forecasting correlation.

The prior literature suggested that stock market uncertainty generates significant cross-market pricing effects, as highlighted in Fleming et al. (1998); Kodres and Pritsker (2002); Chordia et al. (2001), among others, while Connolly et al. (2005) noted that "times of high stock uncertainty are also times with more frequent revisions in investors' assessments of both stock risk and the relative attractiveness of stocks versus bonds". Hence, stock market uncertainty can have a key role in understanding markets' states with negative stock–bond correlation (e.g., in stable inflationary periods). Contrary to the fundamental approach of Campbell and Ammer (1993)—in which the only feature that can induce a negative correlation between stock and bonds is based on the differential response to inflation expectations—Forbes and Rigobon (2002) mentioned that heteroskedasticity can induce time variation in correlation, but it cannot explain why two markets that normally exhibit a positive association occasionally exhibit a negative correlation, which, in turn, suggests that other factors may be important for understanding stock–bond comovements. Going one step further, in this study, we show that a different type of uncertainty associated with news on dangers of infectious diseases can drive important changes in this relationship since they can trigger jumps in the relation of stocks and bonds.

As part of future research, our analysis can be extended to investigate the impact of the uncertainty caused by the current COVID-19 pandemic on predicting changes in stock–bond relation for other financial markets around the world. Finally, from a methodological perspective, it is imperative that the method used here is extended to several major fields of financial applications, such as option pricing, stock market forecasting and bankruptcy prediction.

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