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Why Don't Blanchard-Kahn ever "Catch" Flu? And How it Matters for Measuring Indirect Cost of Epidemics in DSGE Framework

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Abstract

We attempt to apply a New Keynesian open economy model to simulate the economic consequences of influenza epidemic in Poland and measure the output loss (indirect cost) related to this disease. We introduce a negative health shock on the supply side of the economy and demonstrate that such a shock – implemented as a reduction in labour utilisation under unchanged labour cost – is not equivalent to negative labour supply shock. As expectational effects may hypothetically play a significant role in determining the economic cost of influenza, we attempt to endogenise the mechanism of epidemic in the model for the rational expectations solution algorithm to take account for the possibility of epidemic. This attempt has failed for the standard SIR model of epidemic and for the standard Blanchard-Kahn-like local solution methods, as the SIR block is only consistent with Blanchard-Kahn conditions under herd immunity of the population. In the deterministic simulation with the number of infected given exogenously, the output loss resulting from influenza-related presenteeism and absenteeism was estimated at 0.004% of the steady state level on average in the period 2000-2013. The simulated indirect cost in the New Keynesian model has turned out to be lower than the estimates that one could possibly obtain using the human capital approach. The reason for this discrepancy is the demand-oriented construction of the New Keynesian framework, and we treat this result as closer in notion to what the friction cost approach might suggest.

Keywords: indirect cost, influenza, DSGE, Blanchard-Kahn conditions, modelling epidemics

JEL Classification: C62, D5, I10, I19

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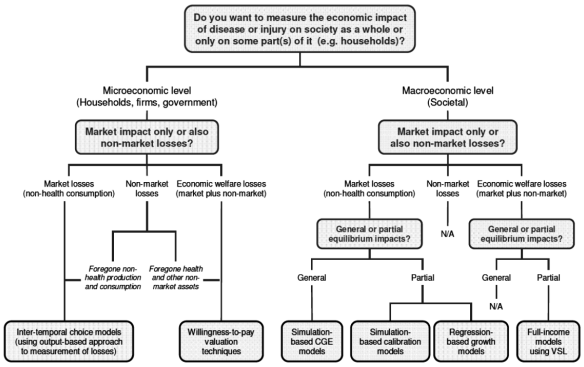
1 Introduction

Seasonal epidemic diseases (like common cold or influenza) are likely to constitute a significant burden to the welfare of the society in general and, specifically, to the economy. Its adequately structured description and measurement can be crucial from the perspective of health policymakers, as regards i.a. public expenditures on prevention and treatment.
Since Rice (1967), it is common to distinguish between two types of economic costs that arise from an illness:

> direct costs, i.e. spendings of public payer, as well as those of households (as out-of-pocket spendings or via private insurance schemes), that finance medical procedures, medicines, hospitalisations etc.; this is closely in line with the common, accounting-based understanding of the costs of illness;
>
> indirect costs, i.e. full macroeconomic alternative cost of an individual being sick; this is, above all, related to the fact that sick household members cease to supply labour for some time.

According to the characteristic features of an illness and its severity, one can distinguish between a few components of indirect costs. Firstly, one can be simply absent from work (an employee on a sick-leave or just interruption of work of a self-employed person) which is called absenteeism. Secondly, even though some sick workers do work, they are not fully productive due to their condition (presenteeism). Thirdly, should a disease cause a long-lasting or permanent damage to one's health, indirect costs stem from his or her inability to work. Fourthly, there can be a huge, multi-period output loss when a working-age individual happens to die of an illness. Fifthly, even healthy working age population may not supply labour if they are engaged in informal long-term care with other people, e.g. children or elderly people. According to World Health Organization (2009), economic implications of diseases from the macroeconomic or social perspective must be investigated possibly in a general equilibrium framework, dynamically, with a range of second-order effects included in the analysis. Specifically, WHO recommends to use general equilibrium models in order to measure the macroeconomic impact of market losses (see Figure 1). In practice, however, such frameworks are still missing in applied policy analyses, largely due to methodological gaps. WHO itself does not specify the methodology suitable for the measurement of economic welfare losses in general equilibrium, i.e. losses in both market labour and consumption terms, as well as non-market, pure utility terms (cf. Figure 1).
In this paper we attempt to challenge this gap using the example of seasonal influenza epidemics in Poland. A few specific features of this disease should be taken into account when performing example calculations. First of all, it is an epidemic disease, i.e. it is characterised by strong surges in the number of infected people in seasonal terms (see Figure 2). Moreover, it is a recurrent epidemic, i.e. economic

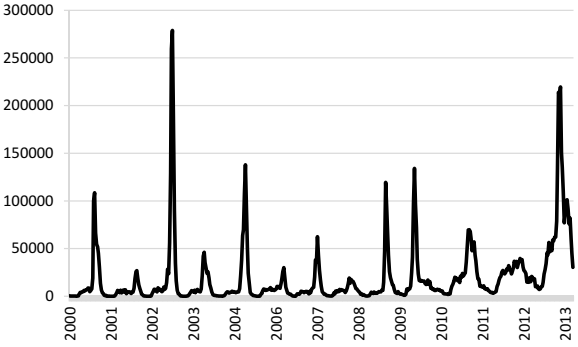
Figure 1: Methodological frameworks for the indirect cost of illness evaluation by WHO



Source: World Health Organization (2009).

agents can take it into account in making their decisions and assessment of their future consequences. Infection periods are relatively short, but flu can cause much discomfort (and hence effectively prevent from any work). Given the presence and construction of Polish public health insurance scheme, this output loss is redistributed between the worker himself (lower wage), the employer and the public finance (the sickness benefit). However, also severe cases can happen, resulting in complications (permanent disability) or even decease. Usually, 0.5-2 millions of people in Poland catch influenza in annual terms (or, more precisely, during an epidemic season that lasts from September to August next year).

Figure 2: Recurrent epidemics of influenza in Poland: number of infected people, 2000-2013 (weekly data)



Source: Narodowy Instytut Zdrowia Publicznego - Państwowy Zakład Higieny (compiled from weekly epidemic updates).

Given the abovementioned expectational effects, as well as a recommendation to take account of a wide range of second-order effects, we decided to apply a New Keynesian dynamic stochastic general equilibrium model to track the economic consequences of an epidemic. We directly apply the model as described by Torój (2010), proposing necessary extensions to account for health and illness considerations. We demonstrate the techniques to modify the utility and production function, and what implicit assumptions they impose. We also discuss these assumptions in the context of characteristic features of influenza. The imposed assumptions imply a clear differentiation between a negative labour supply shock (which primarily affects labour costs in the New Keynesian setup) and a proposed negative "health" shock.
The ultimate aim of this analysis is to measure the indirect cost of influenza in Poland. As an attempt to provide a preliminary answer, we run a deterministic simulation with the specified and extended model. The calibration of the shock series is based on empirical data regarding the number of influenza cases, as published by the National Institute of Public Health (see Figure 2).
The rest of the paper is organised as follows. Section 2 discusses the theoretical aspects of utility and production functions that should be considered in the discussion about health shocks. Section 3 explains why it is impossible to simply create an endogenous "epidemic" block within a DSGE model, and then run stochastic simulations, while avoiding the assumption about agents' perfect foresight in the deterministic framework. Section 4 presents the results of the deterministic simulations. Section 5 concludes and suggests directions for further research.

2 Putting health shocks into the New Keynesian model: theoretical considerations

This Section aims to develop the theoretical extensions to a standard set of micro-foundations in a general equilibrium model, allowing to take account of health and its economic implications. We start with utility function of a household, then proceeding to the producer side.

2.1 Health and utility function

A concise and tractable proposal to incorporate various cost-of-illness aspects into households' behaviour can be found in Bardhan and Udry (1999). They propose the following general specification of a household's felicity function (the corrected by World Health Organization, 2009):

$$U\left[C^{nh}, L, S, H\left(C^{h}\right)\right] \tag{1}$$

where:

U– one-period utility;

C– stream of consumption, divided into consumption of non-health goods (C^{nh}) and health goods (C^h);

S– consumption of non-market, self-produced goods;

L– leisure;

H– the stock of a household's health.

There are four sources of utility: leisure, consumption of non-health goods (acting as the standard utility-yielding consumption in DSGE models), home production and the stock of health. Consumption of health goods does not yield utility directly. It is rather the stock of health that households derive their utility from, and consumption of health goods can be seen as a "production factor" of health. It is important to note that consumption of health goods (privately financed direct costs of illness) is not desirable, as long as the stock of health permits, and a household can attribute resources to other, non-health consumption goods or shift back their budget constraint by supplying less labour. H and S may not directly influence economic variables, but could be crucial when estimating the social impact of non-market losses due to an illness (see Figure 1).
Occurrence of a disease, such as an epidemic of influenza, may be seen as a negative health shock ε^H that affect households in the following ways:

directly decreases the health stock, $H = H\left(C^H, \varepsilon^H\right)$;

generates redistribution of resources from the non-health to health sector; in a resource-constrained, closed economy without capital and investment, the constraint is $Y = C^{nh} + C^h$, so increasing the demand for C^h must decrease C^{nh} as long as Y remains unchanged (which is not the case under the occurrence of indirect costs, so C^{nh} must then decrease even more);

deprives households of time spent on being sick; this time can be split into lost working time (which normally yielded disutility, anyway) and into lost leisure time L^{nh}; this leaves only the utility-yielding amount of leisure $L = 1 - N - L^{nh}$ (N is the labour supply and 1 is the numeraire for the amount of time in a given period).

To see the economic impact of decreasing leisure, consider the standard CRRA utility function with demand shock ε_t^D, risk aversion σ, labour supply shock ε_t^L and Frisch elasticity of labour supply ϕ. If we assume that time spent working and time spent being sick should be deduced from leisure homogeneously, we can write the following:

$$U_t\left(C_t, N_t\right) = \varepsilon_t^D \frac{\left(C_t - hC_t\right)^{1-\sigma}}{1-\sigma} - \varepsilon_t^L \frac{\left(N_t + L_t^{nh}\right)^{1+\phi}}{1+\phi} \tag{2}$$

Note that by defining (2) we make two strong assumptions. First, the same Frisch elasticity ϕ applies to time spent working and time spent being sick (whereby only

the first one is a decision variable). Secondly, when running simulations, we must be able to define how much leisure is absorbed by the disease (e.g. time spent visiting doctors, queuing, going to pharmacies or just feeling bad). Another problem is how to treat time that is spent on a sick leave – depending on the degree of symptom severity, the illness may even increase leisure.

If we take account of decreasing leisure in the utility function (2), one must be aware that we thereby also decrease consumption, at least in the short run. This stems from the first order conditions of consumer optimisation problem, that encompass i.a. the labour supply equation:

$$\frac{W_t}{P_t} = mrs_t = \frac{\frac{\partial U}{\partial C}}{\frac{\partial U}{\partial L}} \tag{3}$$

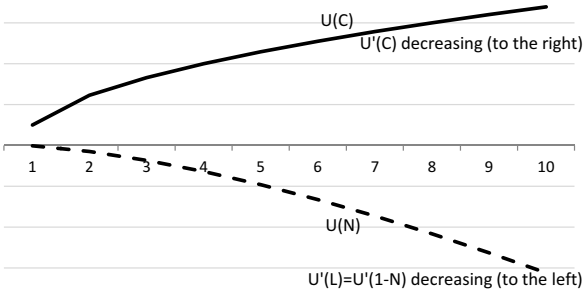
Here, the real wage W_t/P_t must be equal to the marginal rate of substitution between consumption and leisure. Under a flexible labour market, this can be seen as a wage-setting equation. However, if W_t/P_t is inflexible in the short run, one might expect C to adjust to decreasing L. The mechanism of this adjustment is illustrated in Figure 3.

With standard assumptions about decreasing marginal utility from consumption (solid line) and leisure, we get increasing marginal loss in utility from working time (as additional work deprives the household from increasingly valued and more scarce leisure hours). By adding a positive amount L_t^{nh} to (2), we move to the right along the dashed line. Thereby we decrease L, thus making it more valued in marginal terms. In order to keep mrs_t constant, we have to make $\partial U/\partial C$ more valued as well, i.e. move along the solid line to the left. This implies reducing the level of consumption. In other words, leisure-deprived households could attempt to partly defend their previous leisure by reducing their effective labour input, income and finally consumption.

While this may seem unintuitive, we should bear in mind that we only speak of pure leisure effect. Households are also deprived of their working time, which hits their income and consumption. Under that circumstances, further adjustment in consumption may be unnecessary or agents could even be willing to supply more labour by reducing their leisure, to defend their previous level of consumption. The final consumption-leisure choice should be the composite of both effects. Its outcome is ambiguous and crucially depends on the level of L_t^h.

In the simulation analysis discussed further, we do not incorporate effects stemming from leisure and health-related utility for three reasons. Firstly, our focus in this paper is on the economic effects (macroeconomic indirect costs), which excludes categories such as general welfare ranking or losses. Analysing health-related utility would require a lot of further, possibly controversial assumptions and discussion, such as the specification of "health production function" from direct costs or defining qualitative and quantitative properties of marginal utility from health. Secondly, we do not

Figure 3: Decreasing marginal utility from consumption and leisure



believe that the above-discussed considerations apply to the specific case of influenza: households are unlikely to make consumption-leisure decisions based on an infection lasting a few days, under contracts lasting for months or years. Thirdly, we avoid making extremely arbitrary assumptions about the amount of leisure lost because of influenza and whether such a loss of leisure is equivalent, in terms of utility, to leisure given up by supplying work.
Nonetheless, one must be aware that – by leaving the utility function as it is in the analysis that follows – we make the implicit assumption that an infection or a sick leave does not impact the amount of leisure at the disposal of a household, either positively or negatively.

2.2 Health and production function

Traditional approaches to measuring indirect cost of illness, such as human capital approach or friction cost approach, are focused on the producer side rather than on the household side.
The most widespread human capital approach (see e.g. Wrona *et al.*, 2011) assumes a simple linear relationship between lost hours of work and lost output. The scaling factor is labour productivity, measured by the hourly wage rate or adequately transformed *per capita* GDP. This approach is atheoretical in the sense of ignoring the existence of a multi-factor production function, non-linear specifications of this function, and various forms of behavioural adjustment, i.a. transferring hours of work between periods.
Koopmanschap *et al.* (1995) criticise the human capital approach because it disregards the economic alternative of temporarily hiring new workers in the place of the sick ones. If that alternative was always feasible, the indirect cost would be reduced to the resources devoted to administrating the temporary replacement and training the new worker. The underlying assumption is the existence of a quantitatively and qualitatively unlimited stock of unemployed population, willing to accept the short-term replacement job and able to perform it perfectly well. One

also has to assume that hiring new workers recruited from the stock of unemployed has no impact on wages. Naturally, in this "friction cost" approach, the cost of hiring and training a temporary replacement is incomparably lower than lost productivity over the entire period of another employee's illness.

Pauly *et al.* (2002) indicate possible nonlinearities at the micro (firm) level that may provide a more nuanced picture than any of the above-mentioned approaches. A quasi-insurance scheme of hiring additional workers just to smooth the impact of diseases is only feasible for big enterprises as an element of economies of scale. Also, this may not be an adequate description mechanism should an epidemic like influenza occur, as risks are probably highly dependent. Pauly et al. also refer to different micro-level production functions that have qualitatively different implications on the macro level as regards the indirect cost, such as "key workers" model with heterogeneity (highly productive leaders and less productive co-workers) and "teamwork" model, where the absence of a single team member substantially and disproportionately hinders the entire production process.

We propose a hybrid of the two mainstream approaches – the human capital and the friction cost – taking the standard Cobb-Douglas production function as a starting point (and assuming that it describes sufficiently well the above-mentioned nuances at the aggregate, macro level):

$$Y_t = A_t \cdot K_t^{1-\alpha} N_t^{\alpha} \cdot \varepsilon_t \tag{4}$$

where A – total labour productivity, K – capital stock, ε_t – supply shock and α (calibrated in further simulations at 0.65) – the Cobb-Douglas exponent for labour.

Under this specification, average labour productivity is clearly higher than marginal labour productivity, that is a better proxy of the impact that an epidemic exerts when pushing some workers out of the labour market for some time. Lower marginal productivity of labour is broadly in line with the argument risen by Koopmanschap *et al.* (1995) that a sick worker can be replaced by others, although – formally – it is rather decreasing technical equipment of labour that is responsible for the diminishing marginal labour productivity under Cobb-Douglas.

For a disease like influenza, where the period of absence or low productivity is relatively short, one can argue that the employer is faced with a stochastic, health-related variation in effective labour input rather than actual hiring and firing decisions:

$$Y_t = A_t \cdot K_t^{1-\alpha} \left(H_t \cdot N_t\right)^{\alpha} \cdot \varepsilon_t \tag{5}$$

With healthy labour force, $E\left(H_t\right) = 1$, but an epidemic forces H_t below unity. With the stochastic factor H_t, the marginal product of labour should be extended to:

$$MPN_t = \frac{\partial Y_t}{\partial N_t} = A_t \cdot K_t^{1-\alpha} \alpha N_t^{\alpha-1} \cdot \varepsilon_t \cdot H_t^{\alpha} \tag{6}$$

After log-linearisation:

$$mpn_t = a_t + (1-\alpha)\, k_t + (\alpha - 1)\, n_t + \epsilon_t + \alpha h_t \tag{7}$$

This leads to a real marginal cost equation (mc_t) different from the standard New Keynesian real marginal cost (mc_t^{NK}) by $-\alpha h_t$:

$$mc_t = w_t - p_t - (a_t + (1-\alpha)\, k_t + (\alpha - 1)\, n_t + \epsilon_t + \alpha h_t) = mc_t^{NK} - \alpha h_t \qquad (h_t < 0) \tag{8}$$

Note that in this New Keynesian, demand-driven framework, the supply side is finally represented by the Phillips curves for individual sectors along with their real marginal cost equations (as in (8)). Quantities are hence determined on the demand side which is not shocked directly (but only via lower disposable income, should this be the case). As a consequence, output in all the sectors calculated from the market clearing conditions is a function of consumption (domestic and foreign), as well as all relevant relative prices.

2.3 Health and labour supply: IRF analysis

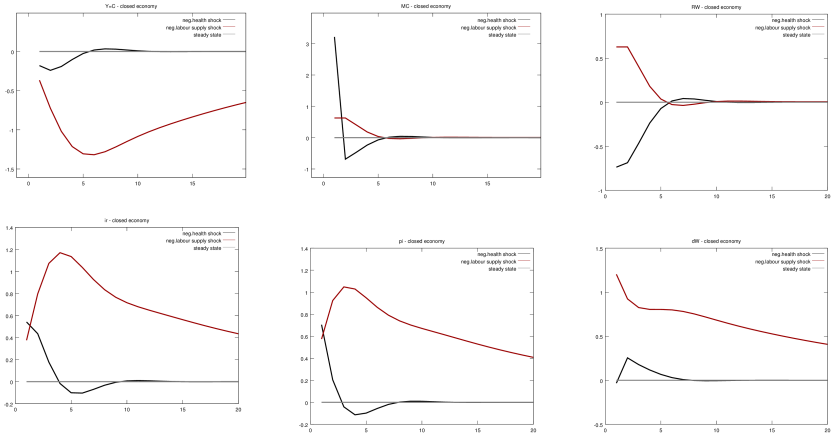
Having specified the labour shock as H_t, we investigate its economic implications by analysing the impulse response functions. In particular, we focus on the difference between the health shock and what might be seen as its more common counterpart, i.e. negative labour supply shock ε^L (from equation (2)).

The specification of the model is based on Torój (2010). In its open economy version, there are 2 countries with a calibrated degree of openness and size. In both, there are 2 sectors: tradable (T) and nontradable (NT). The closed economy version obviously implies just one country and one, nontradable sector. Households and producers solve the same optimization problems, i.e. maximizing the discounted present and future stream of utility and profits, respectively. Monetary policy operates via Taylor rule with smoothing. There are nominal and real rigidities, such as Calvo price- and wagesetting (Calvo, 1983) with backward looking price indexation (Gali and Gertler, 1999) and partial wage indexation (Erzeg *et al.*, 2000), as well as consumption habits (a la Fuhrer, 2000). The capital stock is assumed to be constant, as compared to (4), which – in this simulation – implies that the health shock is unlikely to impact capital and investment in the short run. The calibration is fully based on the posterior means obtained in the Bayesian estimation by Torój and Konopczak (2012).

In the closed economy setup, health shock has a less persistent effect than labour supply shock in terms of output and consumption (see Figure 4). This is a consequence of the fact that the health shock only occurs on the supply side, i.e. in the equations of real marginal costs in all sectors. On the contrary, the labour supply shock appears also in the labour supply equation, and hence visibly impacts on wagesetting (see the IRF for nominal wage dynamics, *dw*). As a result, real wages diverge between the two shocks, and the impact on real marginal cost lasts longer in the case of labour supply shock. This persistence produces humped-shaped responses of inflation and interest rates, as opposed to health shock, where all the output-negative factors remain short-lived.

Turning to the open economy case (Figure 5) does not change any qualitative

Figure 4: Impulse-response functions to negative labour supply shock versus negative health shock: closed economy



Y– output, C – consumption, MC – real marginal cost, RW – real wage, ir – nominal interest rate, pi– inflation, dW – wage inflation.

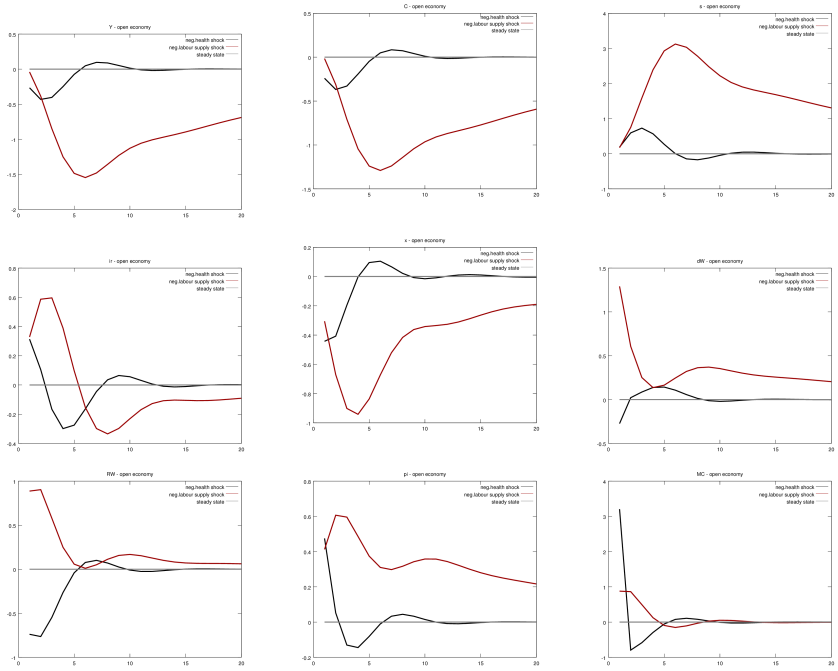
conclusion. One should only notice that a negative health shock has a moderate impact on terms of trade appreciation (and hence competitiveness loss) as compared to negative labour supply shock. This is also originally related to nominal wage dynamics.

3 Endogenising the epidemic with a SIR model

Epidemics of influenza (J10 and J11 by ICD-10 codes), but also of a common cold (J00), share specific features that motivate treating the mechanism of epidemic as an inherent, endogenous feature of the model. First of all, they are roughly seasonal and, also roughly, expected. This seasonality, however, is not deterministic, and not even stochastic in the sense of e.g. time-varying SARIMA patterns. Even with quarterly data, one cannot be sure that the epidemic curve shall peak in the fourth quarter, or in the first quarter (sometimes even moving non-negligibly into quarter two). However, once agents realise that the last quarter of a calendar year is characterised by an accentuated and above-average surge in the number of infected people, one can also expect quarter one to be exceptionally severe.

Why should expectations matter here? When indirect costs occur, resource-constrained households consume less having less output available. In an open economy and under international risk sharing, one could also consider current-account implications thereof. In both cases, consumption-smoothing agents could prepare for an epidemic while lowering their consumption earlier. Also, producers could adjust their labour demand decisions to the fact that the effective labour productivity

Figure 5: Impulse-response functions to negative labour supply shock versus negative health shock: open economy



Y– output, C – consumption, s– (log) terms of trade, x – (log) internal terms of trade, dW – wage inflation, RW – real wage, pi– inflation, ir – nominal interest rate, MC – real marginal cost.

(i.e. after taking account of influenza-based productivity losses) is lower. This is of particular importance under wage and other labour market rigidities.

Although the economic consequences thereof seem to be predictable on a month-to-month and even quarter-to-quarter basis, huge epidemics cannot be accurately predicted many years in advance as they remain in line with mutations in the virus structure (see Brydak, 2008, for more on mutation mechanism and historical sizes of epidemics or pandemics). This is why applying a stochastic, epidemic-generating shock and an endogenous propagation mechanism within a rational expectations model seems to be preferable when taking account of economic expectational effects due to influenza rather than applying an exogenous path illustrating the number of infected people. The latter makes perfect-foresight agents aware of future epidemic sizes and could be admissible as a first-order proxy when no unexpectedly sizeable epidemics happen.

We attempt to endogenise the epidemic using a SIR model (Susceptible-Infected-Removed), in the version presented by Brauer (2008, chapter 12). Its continuous-time

version is presented below:

$$\frac{dS}{dt} = -\beta S\left(I + \delta A\right) \tag{9}$$

$$\frac{dL}{dt} = \beta S\left(I + \delta A\right) - \frac{1}{\phi}L \tag{10}$$

$$\frac{dI}{dt} = p\frac{1}{\phi}L - \frac{1}{\alpha}I \tag{11}$$

$$\frac{dA}{dt} = (1-p)\frac{1}{\phi}L - \frac{1}{\eta}I \tag{12}$$

$$\frac{dR}{dt} = \frac{f}{\alpha}I + \frac{1}{\eta}A \tag{13}$$

where t denotes time and:

S – the number of susceptibles, i.e. those could potentially be ill but are not so far;

L – the number of latents, i.e. those who already were exposed to the virus and can either become infected or go through influenza asymptomatically;

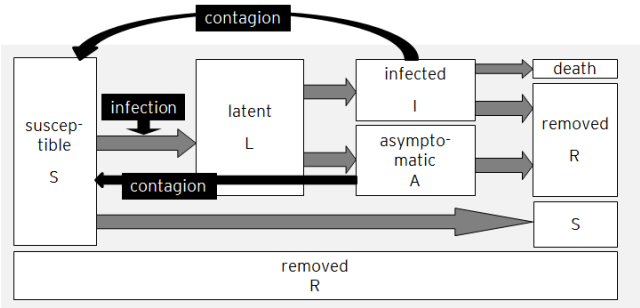
I – the number of infected, i.e. the cases of influenza;

A – the number of asymptomatics, i.e. those who were exposed to the virus (and hence latent) but did not develop symptoms and hence are not officially counted as the cases of influenza;

R – the number of removed, i.e. individuals that used to be infected or asymptomatic in the same season and became immune for the rest of the season, or individuals who were immune to influenza from the very beginning of the season.

The mechanics of the system is illustrated with Figure 6. An epidemic begins with introducing a number of infected agents into a population that is initially split into susceptible and immune people (marked as removed from the beginning). Susceptible agents become exposed to the virus when interacting with other agents, with the intensity of contacts in the population in a given unit of time calibrated as β. Agents remain latent for some time (with a mean duration ϕ), then moving either into the infected group with probability p or to the asymptomatic group with probability $1-p$. The mean periods of remaining in these groups are denoted respectively as α and η. By assumption, ϕ, α and η are means from exponential distribution. Both groups infect other people (while the infectivity of asymptomatics is reduced by a factor of $\delta < 1$). Ultimately, a small fraction of infected individuals die, and the rest of infected and asymptomatic people become immune – and move to the "removed" group – for the rest of the season.

Figure 6: Flows between subgroups of population within the SIR model



Two remarks should be made at this stage. Firstly, as regards the specification of this model, we skip the possibility of vaccinations and hence apply the version of this model where agents are homogeneous in their susceptibility. In general, it is possible to adjust the model to account for the dichotomy between the vaccinated and unvaccinated agents. It might be crucial for policy analyses, but – given a negligible fraction of Poles vaccinated against influenza (3.75% in 2012) – we skip this extension for clarity of presentation. Under such a low fraction, however, the subsequent results remain almost unchanged.

Secondly, the model should be put to a discrete-time version in order to be included in a New Keynesian setup. The SIR model is usually calibrated at daily frequency (cf. 1), which is far above the usual New Keynesian setup, usually calibrated and estimated in quarterly terms. When we move to a higher frequency, we should adjust the model parameters: β, α, η and ϕ appropriately. Also, if we treat the described population as the labour force, we can say that there is no feedback from the economy into the block described above, so it can be at first analysed alone at a daily frequency. All the subsequent conclusions hold regardless of the frequency in question.

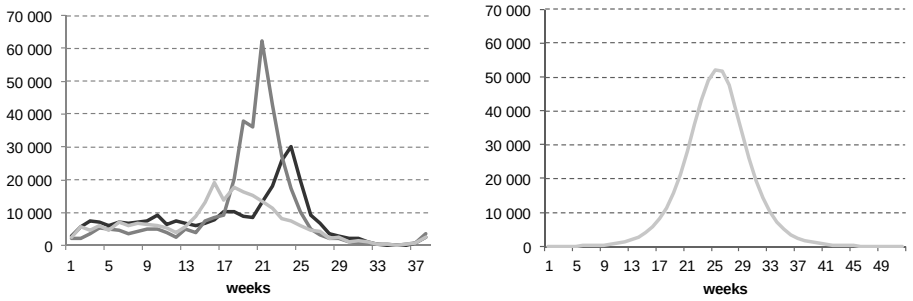
Table 1: Calibration of SIR model (daily version)

Parameter	**Notation**	**Calibration**	**Source**
population	N	38200037	CSO
mean transmission rate	β	$8 \cdot 10^{-8}$	ML estimate
fraction of susceptibles	s_0	12.4%	ML estimate
mean infection period	α	4.1	Longini (2004)
mean asymptomatic period	η	4.1	Longini (2004)
mean latent period	ϕ	1.9	Longini (2004)
probability of asymptomaticness once latent	$1-p$	0.66	Brauer (2008)
reduced infectivity of asymptomatics	δ	0.7	Brauer (2008)
probability of decease once infected	$1-f$	$5 \cdot 10^{-5}$	Molinari (2007)

ML estimates involve empirical fit of the theoretical epidemic curve to historical influenza epidemics in Poland, over the decade 2000-2010.

It can be demonstrated that, with the calibration presented in Table 1, the SIR model replicates the shape of epidemic curves observed for influenza in Poland over the period 2000-2013 (see Figure 7). Although it is a model of non-recurrent epidemic, the recurrent behaviour could be implemented in a few ways: via stochastic shocks, regime switching behaviour, or modelling smooth transitions from the stock of removed to the stock of infected over the summer quarters.

Figure 7: Cases of influenza



Source: Narodowy Instytut Zdrowia Publicznego - Państwowy Zakład Higieny (compiled from weekly epidemic updates) and own calculations.

The attempt at building a block representing the epidemic and including it in the New Keynesian model has failed, however. The standard Blanchard-Kahn conditions, checked upon solution of a log-linear model, were not fulfilled. Systematically, the number of unstable eigenvalues of the linear system exceeded the number of non-predetermined variables. In such circumstances, no stable equilibrium exists and it is impossible to find any solution to the system under rational expectations. Interestingly, there is a pattern in this in instability that has to do with inherent properties of the SIR model.

To see this, note that equations (9)-(10) state the flow from S to L, $\beta S\left(I+\delta A\right)$, as a function of cross-product variables SI and SA. This specification generates the hump-shaped epidemic curve presented in Figure 7. Initially, the stock of susceptibles is high (large S) and the number of infected (and asymptomatic) people – low (small I and A). As the epidemic develops, large S is decreasing slowly (in percentage terms), but I – increasing rapidly (in percentage terms), making the epidemic spread quickly. This is when the surge in the number of infected individuals slopes almost vertically. This is happening until S decreases strongly enough to make its percentage drops outperform the increases in I in their product. This is where the epidemic curve peaks. In other words, the turning point is reached when there are already so many infected people in the society that the chance of meeting and infecting a susceptible is sufficiently low for the outflows from I to R to dominate. This is when the curve starts to slope downwards.

Consider now the discrete-time version of equation (9), as well as its linear-quadratic approximation:

$$s_t - s_{t-1} = -\beta \bar{S} i - \beta s i - \beta \delta \bar{S} a - \beta \delta s a \tag{14}$$

The log-increment of S is now linearly dependent on i and a, but also on the cross-products si and sa. Note that the standard log-linear approximation only contains the former, so it is impossible to capture the hump-shaped behaviour of the epidemic curve. $\bar{S} = s_0 N$ denotes here the steady state number of susceptibles in the population and is the only non-negative steady state value in the block of epidemics (the part of the removed people that are immune by nature is not modelled here). Technically, to ensure the return to a steady state, an equation has been added that transfers the removed people back to the susceptible stock at a very slow pace. The "depreciation rate" of immunity against influenza is a matter of discussion, and so is the number of susceptibles in the steady state. This – even in the absence of Blanchard-Kahn conditions violation – would require making strong assumptions when implementing the endogenous epidemic mechanism into the model.
A key, reduced-form parameter in the SIR model is the basic reproduction number, BRN, defined as (cf. Brauer, 2008):

$$BRN = N s_0 \beta \left[p\alpha + \delta \left(1 - p\right) \eta \right] \tag{15}$$

It is a straightforward function of the deep parameters in the model and represents the number of people that, on average, become infected by one sick individual. This parameter provides a synthetic description of the epidemic behaviour in the model. In general, three states are possible:

1. $BRN > 1$: this is the "normal" situation in which the explosive behaviour of an epidemic looks like in Figure 7 and a hump-shaped pattern occurs. This situation also arises under the parameters from Table 1.

2. $BRN = 1$: a hypothetical situation in which the population of infected people remains flat and replicates itself exactly from period to period.

3. $BRN < 1$: the reverse situation, in which the shape from Figure 7 no longer applies. With basic reproduction number lower than unity, the epidemic spreads more slowly than people get cured and move to the "removed" group. As a consequence, the number of infected people - once some of them get introduced into the population – is slowly, gradually and monotonically decreasing. This version is referred to as "herd immunity" and would not be empirically plausible as a valid description of seasonal influenza propagation in Poland, given historical data (Figure 2).

However, our sensitivity analysis with respect to $N, s_0, \beta, p, \alpha, \delta$ and η (i.e. all the arguments in 15) confirmed that it was precisely crossing the value consistent with $BRN = 1$ and moving into the range $BRN < 1$ that pushes the excess explosive

eigenvalue into the stable region. This makes the model compatible with Blanchard-Kahn conditions. Hence, it is only the unrealistic situation of achieving the herd immunity that allows to solve the log-linear model.
Solving the New-Keynesian model extended with discrete-time versions of equations (9)-(13) (along with an ad-hoc equation of R flowing back to S) is therefore impossible using local approximation techniques, such as the standard Blanchard-Kahn framework. Several alternatives can be considered. As the global stability of the model is not doubtful (or even the stability under the quadratic approximation (14)), one can turn to accurate, but computationally far more challenging global methods. One can also specify the model in a regime-switching setup and solve with the method proposed by Farmer et al. (2008) – like in the case of active-passive monetary policy regimes, described by Davig and Leeper (2007), so that the Taylor principle is safeguarded in the long term and the solution exists. This requires specifying two stochastically switching regimes, in which the vector of parameters implies $BRN > 1$ and $BRN < 1$.
Yet another solution is to ignore the disadvantages of implied perfect foresight assumption and run a deterministic simulation, using an exogenous path of the number of infected people that is known in advance. This approach is explored in the next Section, and allows us to leave aside the considerations of violated Blanchard-Kahn conditions and of recurrent epidemics mechanism.

4 Deterministic simulation: indirect cost of influenza

To simulate the shock related to the occurrence of influenza epidemic, one should first map the data presented in Figure 2 into health-related labour utilisation shock H_t as described in Section 2. In general, $\bar{H} = 1$ refers to the steady state (no influenza) and values below indicate drops of working hours in a given quarter, e.g. $H_t = 0.99$ indicates a drop by 1%.
In constructing the exogenous variable H_t, we apply the following steps:

1. Start with the number of infected people, reported by NIZP-PZH on a quasi-weekly basis (i.e. always 4 times per month).
2. Reduce this to working-age population. NIZP-PZH reporting presents the age breakdown of the infected, and the group 15-64 is considered. On average, the working-age cases account for 49.7% cases over the sample period.
3. Reduce this to working-age individuals active in the labour market (and working) using Eurostat data on labour market activity from the Labour Force Survey (approximately 55% towards the end of the sample).
4. The number of cases (1), reduced to labour market relevant quantity (2-3), should be translated into a number of working hours. We do this in two ways.

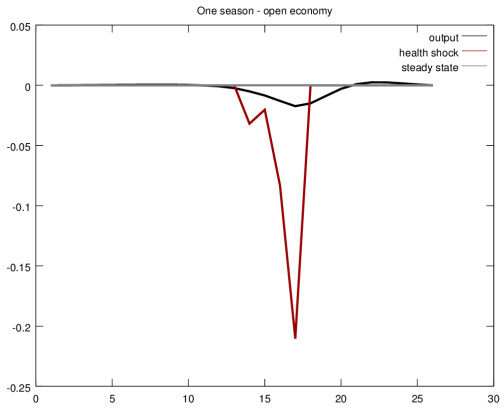
(a) First, we use the data from the statistical portal of the Social Insurance Institution (ZUS) regarding the number of days spent on sick leave due to influenza. In the breakdown of sick leaves according to ICD-10 classification, the positions J10 and J11 were considered. This is annual data, so it was interpolated into quarters proportionally to the number of infected people. We assume a working week to last 40 hours.

(b) Secondly, the difference between the number of sick, working population and the number of sick leaves in a quarter was attributed to presenteeism. In the case of influenza, the unit cost of presenteeism is estimated in the literature at 2.5 days of lost productivity per case (Nichol, 2001).

5. The number of lost working hours calculated in 4a+4b was divided by all hours worked (plus 4a and 4b), which yields the percentage loss of labour utilisation H_t in question.

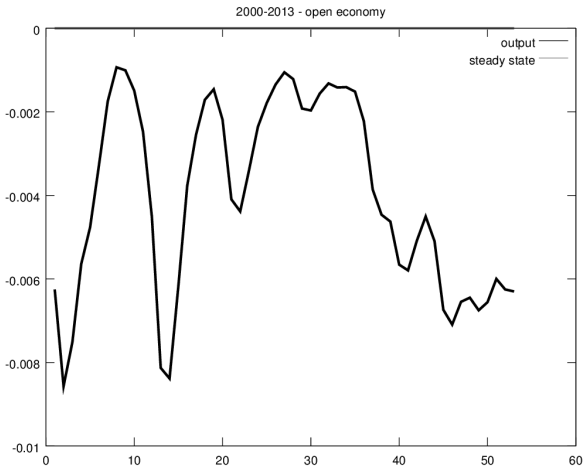
We consider 2 simulation scenarios: a single epidemic season (example 2012-2013), providing us with IRF-like qualitative insights, as well as the whole path between 2000 and 2013 (to draw preliminary quantitative conclusions).
Based on a single epidemic season (Figure 8), a few points can be made. First, the expectational effects prior to the outbreak of the epidemic do not seem to be significant. This may at least partly validate the use of deterministic simulation. Secondly, in contrast with negligible *ex ante* impact, the *ex post* impact (i.e. after the epidemic subsides) seems to account for approximately one-fourth of output losses. This stems mainly from applying the New Keynesian setup, with nominal price and wage rigidities, as well as wage indexation, inflation persistence and consumption habits. This may lead to a hypothesis for further exploration – that the indirect cost of an illness is a function of deep economic parameters, describing the social preferences and market structures.
Thirdly, and most importantly, the percentage loss in health-related labour utilisation is incomparably higher than the loss in output. This is also a direct consequence of using the New Keynesian, and hence a demand driven model. Note that here, it is market clearing condition (rather than production function) that determines the level of output, whereby the production function only affects the cost side of the economy. This makes the factors of production more scarce, increases their cost, and hence suppresses output. However, the extent of this suppression is not comparable with a simple analysis of the production function. From this perspective, one can say that a New Keynesian analysis of the indirect cost is closer – in its general idea – to the notion of friction cost approach than to the human capital approach. The latter would probably be better represented by a real business cycle model. Figure 9 presents the output loss related to the historical epidemics of influenza over the period 2000-2013. The output in Poland was, on average, lower than the steady state by -0.004%. Note that this estimate only contains absenteeism and presenteeism, that – according to available static, human-capital-based calculations – accounted for

Figure 8: Output in a single epidemic year – open economy, deterministic simulation, percentage deviations from steady state



ca. 1 bn PLN of annual indirect cost in the average year over the last decade (Ernst & Young, 2013). We also do not take account of the complications of influenza, incurring – according to Ernst & Young (2013) – approximately 30% of the costs. The obtained result – expressed here in real terms as % of GDP – is only comparable to the available estimates of the indirect cost incurred by pure influenza (J10 and J11) presenteeism and absenteeism. Once again it should be stressed that our result

Figure 9: Influenza-based output loss in Poland in 2000-2013: percentage deviations from steady state



applied a different methodological approach than the available ones (including Ernst & Young, 2013), by using a demand-oriented, and not supply-oriented model. In a sense, it can also be treated as a rough proxy for what the friction cost approach could suggest. The key, and in fact empirical, question regarding the validity of the former or the latter approach is the decision of enterprises on the micro level as regards the level of their output. Namely, they can either attempt to defend their level of production and pursue this goal more expensively (via additional hires at lower marginal productivity, overtime payments etc.) or forego some output, accepting the lower capacity. These two types of behaviour would validate the described, friction-cost-flavoured New Keynesian framework or the alternative human capital framework based on production function, respectively. There is probably no universal choice here, but the decision must be made specifically for various types of diseases.

5 Conclusions and further research

This paper attempts to apply a New Keynesian open economy model to simulate the economic consequences of influenza epidemic in Poland and measure the output loss (indirect cost) related to this disease. We introduce a negative health shock on the supply side of the model. On the household side, we demonstrate how leisure and health-related utility issues could additionally be taken into account, should the analysis be extended to account for broader welfare (or social) costs. We also demonstrate that such a negative health shock – implemented as a reduction in labour utilisation under unchanged labour cost – is not equivalent to negative labour supply shock. The latter is far more aggressive and persistent in the economy, as it directly hits the nominal wage behaviour. We argue that this representation of the shock is adequate when computing the costs of short-lived diseases like epidemics of influenza, and when assuming away its long-run consequences like permanent disability to work or a decease after severe complications.

As expectational effects may play a significant role in determining the economic cost of influenza, we attempt to endogenise the mechanism of epidemic in the model for the rational expectations solution algorithm to take account for the possibility of epidemic. This attempt has failed for the standard SIR model of epidemic and for the standard Blanchard-Kahn-like local solution methods (see Blanchard and Kahn, 1980). It turns out that a SIR block is only consistent with Blanchard-Kahn conditions under herd immunity of the population, i.e. when the basic reproduction number in the SIR model is below unity. This is why we turned to deterministic simulation which demonstrated a relatively low impact of expectational effects, and an output loss at 0.004% of the steady state level on average in the period 2000-2013. This does not account for complications, resulting in disability or deceases.

More importantly, the simulated indirect cost in the New Keynesian model has turned out to be substantially lower than the available estimates obtained using the human capital approach. The reason for this discrepancy is the demand-oriented construction

of the New Keynesian framework, which makes producers attempt to defend their previous level of output at increased real marginal costs. This defense obviously cannot be fully successful, and the obtained numbers reflect the size of this failure. However, unlike in the human capital approach, the supply side is not represented here by a production function in its pure form, as it appears only implicitly in the real marginal cost equation. In this sense, we treat the obtained results as a tentative, rough proxy for what the friction cost approach might indicate as the indirect cost of influenza in Poland.

This paper was meant as an initial attempt to introduce the notion of indirect influenza cost into a New Keynesian DSGE model, rather than as an ultimate economic framework for analysing the economic impact of influenza. There are at least a few missing points to investigate in further research. Firstly, the model should be extended by the public finance sector, including the social insurance and health insurance aspects, to investigate the redistribution effects related to missing contributions and additional transfers to households. This would also allow to provide a more nuanced picture of what would happen on the cost side of the producers. Secondly, a more complex and complete picture could be obtained after including the health sector and the direct costs of illness as a flow of resources from the non-health sectors to the health sector. Further attempts to endogenise the epidemic could be focused introducing a regime-switching framework that could potentially ensure compatibility with Blanchard-Kahn conditions. Finally, one could take a closer look at what the marginal loss of output with respect to a missing worker really is, e.g. by means of multi-agent modelling in an environment with various production functions in various firms on the micro level. A multi-agent model could also be an interesting polygone to analyse the propagation of the epidemic in a more nuanced way that in a standard SIR model.

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