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H1N1 Influenza in Australia and its Macroeconomic Effects

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Abstract

Early 2009 saw the emergence of an H1N1 influenza epidemic in North America that spread to eventually become a global pandemic. Previous work has suggested that pandemics can have large macroeconomic effects on highly affected regions; here we estimate what those effects might be for Australia. Our analysis applies the MONASH-Health model: a quarterly computable general equilibrium model of the Australian economy. We simulate the effects of two H1N1 epidemics; the relatively mild 2009 outbreak and also a more severe episode. The analysis supports the assertion that an H1N1 epidemic could have significant short-run macroeconomic effects.

JEL codes: C68, E37, I18

Keywords: computable general equilibrium, H1N1 influenza, pandemics

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1. Introduction

April 2009 saw the emergence of an H1N1 influenza (swine flu) epidemic in North America that spread to eventually become a global pandemic. By January 2010 H1N1 activity had peaked in most regions of the world but intense pandemic activity was still being observed in North Africa, Southern Asia, and parts of East and Southeast Europe. The pandemic had ended by August 2010, by which time there had been laboratory confirmed cases of swine flu in over 214 countries and, at least, 18,449 deaths (WHO 2010a, 2010b, 2010c).[1] In Australia, there had been 37,562 confirmed cases of swine flu and 191 deaths by the middle of summer (January) 2010, by which time national influenza activity was low (Department of Health and Ageing 2010).

A measure of the relative severity of the epidemic in Australia is given by a comparison of two indicators. Relative to other southern hemisphere countries, Australia's crude rate of confirmed cases per 100,000 population was very high at 166.7; this compares to 74.1 in Chile, 72.8 in New Zealand, and 20.7 in Argentina. Australia also compares unfavourably to northern hemisphere countries on this measure; 21.6 in the UK, 21.1 in Mexico, and 12.5 in the USA. Australia compares more favourably in terms of hospitalisations per 100,000 population at 21.4. Similar rates were observed in New Zealand (22.7), and Argentina (22.1), but much lower rates in Chile (9.1), Canada (4.4), the USA (3), and the UK (2.7).[2] Of the number hospitalisations, 13% were in intensive care units (ICUs) and this was much higher than expected (Bishop 2009). Overall, the above measures indicate that with respect to other countries in 2009, Australia's H1N1 epidemic can be regarded as relatively severe. Reported numbers of cases per capita were high, reflecting intensive case finding efforts in the initial phases of the pandemic response, and ready access to laboratory testing resources around the country.

In 2003 there was a near pandemic of SARS; analyses of this episode estimated very large temporary economic effects in highly affected Asian regions (Chou et al. 2004; Hai et al. 2004; Lee and McKibbin 2004). This suggests that the 2009 H1N1 pandemic might also have large temporary economic effects on highly affected regions. Here we estimate what those effects might be for Australia with a focus on the macroeconomic adjustments that would take place in response to the epidemic. Thus, we take an economy-wide approach to estimating the economic impacts of an H1N1 pandemic. Beutels et al. (2008), Lee and McKibbin (2004), and Smith et al. (2005) argue convincingly that economic analysis of public health emergencies of international concern (e.g., H1N1 influenza and SARS) should not take a partial equilibrium approach by focussing on only the health sector (or parts of the health sector) and forgone incomes resulting from disease-related morbidity and mortality, while ignoring effects in other parts of the economy (e.g., Sander et al. 2009). Illness and death due to public health emergencies raises perceptions of risk and leads to risk-modifying behaviour in an effort to reduce the risk of contracting illness, e.g., prophylactic absenteeism from work and public gatherings. Risk-modifying behaviours affect consumption and reduce labour productivity. Deaths due to illness reduce the availability of workers. Both of these effects will affect all parts of the economy to a greater or lesser extent. Further, Lee and McKibbin (2004), and Smith et al. (2005) show that the effects on the non-health sectors due to SARS and antimicrobial resistance are larger than the

[1] To quote WHO (2010b), "The reported number of fatal cases is an under representation of the actual numbers as many deaths are never tested or recognized as influenza related."

[2] These figures are as at September 2009.

effects on the health sector. Thus, an economy-wide approach is the ideal framework for evaluating the economic impacts of public health emergencies such as H1N1 influenza.

Our economy-wide analysis applies the MONASH-Health model of the Australian economy. MONASH-Health is a detailed, dynamic, computable general equilibrium (CGE) model of the Australian economy. The theoretical structure of MONASH-Health is similar to that of the MONASH model of Australia (Dixon and Rimmer 2002). MONASH-Health places special emphasis on the health sector to aid economic analysis of health sector issues. The health sector detail allows us to carefully target the increased demand on health services that an H1N1 epidemic would be expected to cause, whereas the general equilibrium nature of the model allows us to capture the indirect effects of the epidemic on the non-health sectors.

The MONASH-Health model is modified in a number of important ways to help capture the likely effects of a flu pandemic. Three separate tourism sectors are identified in the model: domestic, inbound and outbound. Previous experience with the SARS epidemic indicates that international tourism is strongly negatively affected by epidemics of this kind (Pine and McKercher 2004; Wilder-Smith 2006). Identifying international tourism separately in the model allows us to accurately target the likely negative effects on international tourism of a flu pandemic. In previous applications MONASH-Health produced annual results (Brown et al. 2009). Here the model is modified to produce quarterly results; quarterly behaviour is an uncommon characteristic of CGE models. This modification is important because pandemics tend to be of short, sharp duration. An annual model tends to smooth out short-term effects leading to potential underestimation of disruption. MONASH-Health has the real world feature of inertia in the labour market (sticky real wages) to which we add the complementary feature of non-instantaneous price responses in the physical capital market (excess capacity). This allows us to avoid the assumption of full capacity utilisation (common in CGE models) in the presence of a contraction (such as a pandemic) that leads to the prediction of a strong export upturn in the short-run. Dixon and Rimmer (2010) show that this is an unrealistic response to a demand-contracting shock.

Applying the MONASH-Health model, we simulate the economic effects of two H1N1 epidemics in Australia: the 2009 outbreak and a significantly more severe episode. There are a number of previous studies focussing on the economy-wide effects of pandemics: some of these studies use macroeconomic (i.e., single sector) models. These include Fan (2003), Jonung and Roeger (2006) and Keogh-Brown et al. (2010). These studies have the advantage of applying quarterly models that allow them to capture the short, sharp nature of pandemics; they have the disadvantage of a single-sector approach that ignores sectors that are particularly relevant to the study of the economic effects of epidemics (e.g., medical services, inbound and outbound tourism). Other studies apply a CGE (i.e., multi-sector) approach. These include Lee and McKibbin (2004), Chou et al. (2004) and McKibbin and Sidorenko (2006). While these studies apply models that have the advantage of identifying multiple sectors in the economy (albeit with a simple aggregated health sector), they are of annual periodicity and so they are unable to accurately capture the short, sharp nature of pandemics. Dixon et al. (2010) are unique in applying a model with the dual advantage of identifying multiple sectors and having quarterly periodicity in analysing the effects on the United States of a serious H1N1 epidemic. The model applied in this study has this dual advantage as well as a disaggregate health industry.

2. The nature of influenza pandemics

2.1 Overview

Detailed epidemiologic observations of influenza in human populations span more than 100 years, including four well-documented pandemics in the 20th century. The majority of influenza morbidity and mortality is due to seasonal strains that circulate each winter in temperate climates and over longer periods in the tropics (Nicholson et al. 2003). Less frequently, the emergence of antigenically novel viruses, often through cross-species transfer or reassortant events, can result in devastating outbreaks with large numbers of excess deaths in a single season (Doherty et al. 2006). The relative absence of immunity to such pandemic strains results in potential for widespread infection with higher attack rates (i.e., infection rates) across the age spectrum, and a greater burden of disease borne by otherwise healthy young adults than in inter-pandemic years (Ahmed et al. 2007). Following the first wave of infection, the new pandemic virus typically replaces previously circulating seasonal strains to produce annual epidemics in an increasingly immune population (Spicer and Lawrence 1984), with historical records describing variable patterns of subsequent disease. In 1918-19 multiple waves of infection occurred in a single year, some out of season, while in 1968 more severe second than first waves were observed in some populations (Fox and Kilbourne 1973).

Precise estimation of death rates due to influenza is made difficult by the potential for misclassification of cause of death. For example, this bias arises when deaths occur in the community prior to medical consultation, or result from some other diagnosed cause such as a cardiac event that might have been precipitated by underlying influenza (Warren-Gash et al. 2009). Having recovered from their infection, individuals are less likely to be reinfected with related H1N1 strains, and may retain some measure of broad cross-protection against unrelated seasonal influenza viruses (Steel et al. 2010).

2.2 How influenza pandemics affect human behaviour

Direct economic effects of illness resulting from influenza include increased healthcare expenditures by patients and funders (e.g., governments, insurers), and increased workloads for healthcare workers. Indirect effects include a smaller labour supply due to deaths, and increased absenteeism from work by sick workers and by workers wishing to reduce the risk of contracting illness in the workplace, i.e., prophylactic absenteeism.[3]

Prophylactic absenteeism is one example of voluntary risk-modifying behaviour in response to a pandemic. Other examples are reduced domestic and international travel, and reduced public gatherings at sporting and other events (Congressional Budget Office 2006). Non-voluntary risk-modifying behaviour may be imposed on workers with children by school closures intended to mitigate the spread of the virus (Beutels et al. 2008). Thus, some workers will be forced to take leave to care for young children. Workers who take paid leave from work, whether forced or voluntary, reduce their firm's labour productivity i.e., output per worker, unless other workers can fully replace output lost due to absenteeism. But this may be difficult during an influenza pandemic because the virus will be widespread and many workers may not present to the health system but will be less productive than would otherwise be the case.

[3] James and Sargent (2006) argue that there is little evidence of prophylactic absenteeism during past influenza pandemics. This stands in contrast to survey results from Sadique et al. (2007) where European respondents suggest that 34% of workers would take prophylactic absenteeism in the event of an influenza pandemic.

It is unclear what attitudes firms have towards absenteeism during pandemics, including whether they prepare for such events or whether it affects their hiring behaviour. A related question is whether firms utilise workers differently during pandemics, e.g., do they expect present workers to work harder or longer to compensate for output lost due to absent workers? And do pandemics directly impact investment behaviour by firms? These issues have not been given much attention by pandemic researchers up to now.

Fan (2003) asserts that a pandemic will reduce business investment due to increased uncertainty and risk, leading to excess capacity. Similarly, consumer confidence will decline due to uncertainty and fear, leading to reduced spending as people elect to be homebound to reduce the probability of infection – this is another example of risk-modifying behaviour. Reduced consumer confidence may particularly affect services involving face-to-face contact (e.g., tourism, transportation and retail spending). James and Sargent (2006) argue that evidence from past pandemics suggests that it is mainly discretionary spending (e.g., tourism and transportation) that is reduced.

Fan (2003) also asserts that an epidemic does not need to be of high morbidity and mortality in order to exert a large psychological impact on attitudes to risk. For instance, although the 2003 SARS epidemic was characterised by low morbidity and mortality it did have a large psychological impact on attitudes to risk. Fan (2003) argues that this paradox can be explained by:

- modern communication technology that transmits information almost instantaneously at low (or zero) cost; and
- the lack of adequate medical information on SARS.

James and Sargent (2006) evaluate this argument by examining evidence from the SARS epidemic and agree that people did experience increased fear of infection, e.g., 50 per cent of Taiwan respondents reported wearing a mask during the height of SARS (p. 22). Nevertheless, they argue that the evidence indicates that the only economic impact during the SARS epidemic was on air travel to affected locations and related impacts on accommodation. Keogh-Brown and Smith (2008) perform a retrospective analysis of the economic impact of the 2003 SARS epidemic and find that the economic effects were mainly but not exclusively centred on East Asian regions, and that the effects went beyond air travel and accommodation.

The above discussion serves to illustrate the unsettled nature of researchers' understanding of how influenza pandemics affect human behaviour.

3. The model

3.1 Overview

MONASH-Health is a detailed, dynamic, CGE model of the Australian economy that places special emphasis on the health sector to facilitate economic analysis of health sector issues. Its theoretical structure is similar to the MONASH model of Australia; MONASH is well documented in Dixon and Rimmer (2002). Here we provide a stylised description of MONASH-Health.

MONASH-Health represents the health sector with 18 separate treatment activities (Table 1); there are also 41 non-health sectors in the model. The detailed representation of the health

sector in MONASH-Health represents a new development for CGE models. Previous work in this area usually treats the health sector by subsuming it within a broader sector within either a multi-sector model (e.g., Keogh-Brown et al. 2009; McKibbin and Sidorenko 2006) or a single-sector model (e.g., Keogh-Brown and Smith 2010). The health treatment activities in MONASH-Health allows carefully targeted analysis of changes in (i) demand for health treatments caused by epidemics or health promotion activities, and (ii) supply of health treatments from potential changes in institutional arrangements and other health-sector reforms.

Table 1 Health treatment industries/commodities in MONASH-Health

1. Cardiovascular	10. Genitourinary
2. Nervous system	11. Endocrine, nutritional & metabolic
3. Musculoskeletal	12. Skin diseases
4. Injuries	13. Maternal conditions
5. Respiratory	14. Infectious and parasitic
6. Oral health	15. Diabetic mellitus
7. Mental disorders	16. Neonatal causes
8. Digestive system	17. Congenital anomalies
9. Neoplasms	18. Signs, symptoms, ill-defined conditions and other contact with the health system

The health care sector is represented in the CGE model as 18 health treatment industries based on the International Classification of Diseases–10th Revision as shown in Table 2, which presents the input-output structure for broadly aggregated industries and commodities. The health treatment industries are linked to other sectors of the economy via inter-industry demands for manufacturing (e.g., pharmaceuticals) and services (e.g., hospital and doctor services). Intermediate inputs (e.g., pharmaceuticals, doctor services) used by the health treatment industries are used in proportions that only vary if there are changes in treatment technology; the health treatment industries do not directly demand primary factors. The non-health industries (primary, manufacturing and services) use their own outputs as inputs as well as primary factors (land, labour and capital). Integrating the health treatment and traditional industries allows us to capture the indirect effects of changes in demand for, or supply of, the treatment industries simultaneously with the direct effects. This is the major advantage of using an economy-wide model with a health sector focus for analysing the economic impacts of health sector changes. Nevertheless, the output of the health treatment industries does not directly impact the performance of workers via changes in their health status.

Table 2 also presents the sales structure of MONASH-Health. We see that health treatments are purchased by households as consumption: these purchases are made at subsidised prices. Consumption expenditure is dominated by manufacturing and services. Physical capital investment is also dominated by services (mainly construction). Government spending is dominated by the provision of public administration, defence and education.

Table 2 Input-output and sales structure in MONASH-Health (2009 $A billion)

Inputs	Industries				Total[a]
	Health treatments	Primary	Manufacturing	Services	
Health treatments	0	0	1	7	9
Primary	0	19	65	16	101
Manufacturing	11	20	112	211	354
Services	59	17	58	595	729
Primary factors	0	72	98	747	916
Total[a]	70	127	335	1,575	2,107

Commodities	Consumption	Investment	Government	Exports	Imports	Total[a]
Health treatments	24	0	0	3	0	26
Primary	17	7	1	89	-23	90
Manufacturing	189	93	0	142	-233	190
Services	384	177	132	72	-62	703
Total[a]	614	277	132	305	-319	1,009

Source: MONASH-Health database.

a Totals may not sum due to rounding.

The non-health treatment sectors in MONASH-Health are treated in a fashion that is typical for detailed, open economy CGE models (e.g., Francois and Reinert 1997). There is a representative firm for each sector that uses a CES (constant elasticity of substitution) combination of primary factors and intermediate inputs. There is limited substitution between the primary factor composite and intermediate inputs, and also between individual primary factors; intermediate inputs are mostly used in fixed proportions. Firms' outputs can be sold to other firms, capital creators (for investment), the representative household, the government, or exported. All domestic agents can choose to buy domestically-produced or imported goods; this choice is also handled by a CES function. Ex-duty prices of imports are assumed to be fixed. Exports are assumed to respond to foreign currency prices, which are endogenous. Thus, the terms of trade are endogenous and the economy is treated as 'almost small'. Total household consumption is assumed to be a fixed proportion of household disposable income; total government consumption is assumed to be fixed.

MONASH-Health includes three types of dynamic mechanisms: capital accumulation; liability accumulation; and lagged adjustment processes. Capital accumulation is specified separately for each industry. An industry's capital stock at the start of year t+1 is its capital at the start of year t plus its investment during year t minus depreciation. Investment during year t is determined as a positive function of the expected rate of return on the industry's capital.[4] Liability accumulation is specified for the public sector and foreign accounts. Public sector liability at the start of year t+1 is public sector liability at the start of year t plus the public sector deficit incurred during year t. Net foreign liabilities at the start of year t+1 are net foreign liabilities at the start of year t plus the current account deficit in year t plus the effects of revaluations of assets and liabilities caused by changes in price levels and the exchange rate.

[4] In this application expected rates of return are only a function of current period variables, i.e., capital creators are assumed to have adaptive expectations. We feel this is appropriate for the application analysed here; an H1N1 epidemic is an event rather than a policy change. Further, it is a new event of unknown magnitude at the time it occurs. It is difficult to envisage how capital creators could accurately predict future variables affecting the rate of return given these characteristics of the epidemic.

Lagged adjustment processes are specified for the response of wage rates to gaps between the demand for and the supply of labour by occupation.[5] Thus, unemployment is endogenous.

In a MONASH-Health simulation of the effects of a pandemic or policy change, we run the model twice to create the baseline and policy runs. The baseline is intended to be a plausible forecast[6] while the policy run generates deviations away from the baseline caused by the shocks under consideration (e.g., an outbreak of H1N1 influenza). For the non-health treatment sectors, the baseline incorporates trends in industry technologies, household preferences and trade and demographic variables. These trends are estimated largely on the basis of results from historical runs in which the MONASH model is forced to track a piece of history (see Dixon and Rimmer 2002, p.38, for further details). For the health treatment sectors, the baseline incorporates forecasts that have been developed from Australian Institute of Health and Welfare data outlining expected future demand for each treatment. Most macroeconomic variables are exogenous in the baseline so that their paths can be set in accordance with forecasts made by expert macroeconomic forecasting groups such as the Australian Treasury and Access Economics. This requires endogenisation of various macroeconomic propensities, e.g., the average propensity to consume. These propensities must be allowed to adjust in the baseline run to accommodate the exogenous paths for the macroeconomic variables.

The policy run in MONASH-Health is normally conducted with a different closure from that used in the baseline. In the policy run, macroeconomic variables must be endogenous: we want to know how they are affected by the shocks under consideration. Correspondingly, macroeconomic propensities are exogenised and given the values they had in the baseline. More generally, all exogenous variables in the policy run have the values they had in the baseline with the exception of the variables of interest. Comparison of results from the policy and baseline runs then gives the effects of moving the variables of interest away from their baseline values. For the present study, the baseline and policy runs differ with regard to the values given to exogenous variables representing an outbreak of H1N1 influenza. We interpret the differences between the results in the baseline and the policy runs as the effects of the outbreak.

3.2 A linear equation system

The model is represented by equations specifying behavioural and definitional relationships. There are m such relationships involving a total of p variables and these can be compactly written in matrix form as

$$A\boldsymbol{v} = \mathbf{0}, \qquad (1)$$

where A is an $m \times p$ matrix of coefficients, $\boldsymbol{v}$ is a $p \times 1$ vector of percentage changes (or changes) in model variables and $\mathbf{0}$ is the $p \times 1$ null vector. Of the p variables, e are exogenous (e.g., measures of the H1N1 pandemic). The e variables can be used to shock the model to simulate changes in the $(p-e)$ endogenous variables. Many of the functions underlying (1) are highly nonlinear. Writing the equation system like (1) allows us to avoid finding the explicit forms for the nonlinear functions and we can therefore write percentage changes in the $(p-e)$ variables as linear functions of the percentage changes in the e variables. To do this, we rearrange (1) as

$$A_n \boldsymbol{n} + A_x \boldsymbol{x} = \mathbf{0}, \qquad (2)$$

[5] This last dynamic mechanism gives MONASH-Health New Keynesian behaviour with respect to the labour market.
[6] Thus, the model baseline is a non steady-state baseline.

where $\boldsymbol{n}$ and $\boldsymbol{x}$ are vectors of percentage changes in endogenous and exogenous variables. A_n and A_x are matrices formed by selecting columns of A corresponding to $\boldsymbol{n}$ and $\boldsymbol{x}$. If A_n is square and nonsingular, we can compute percentage changes in the endogenous variables as

$$\boldsymbol{n} = -A_n^{-1} A_x \boldsymbol{x}. \tag{3}$$

Computing solutions to an economic model using (3) and assuming the coefficients of the A matrices are constant is the method pioneered by Johansen (1960).

Equations (1) represent the percentage-change forms of the nonlinear functions underlying the model; these forms are derived by total differentiation. Thus, (1) is an approximation based on marginal changes in the independent variables. So (3) only provides an approximate solution to the endogenous variables $\boldsymbol{n}$; for marginal changes in $\boldsymbol{x}$ the approximation is accurate but for discrete changes in $\boldsymbol{x}$ the approximation will be inaccurate. The problem of accurately calculating $\boldsymbol{n}$ for large changes in $\boldsymbol{x}$ is equivalent to allowing the coefficients of the A matrices to be nonconstant. The problem is solved by breaking the change in $\boldsymbol{x}$ into i equal percentage changes. The multistep solution procedure requires that there are $(i-1)$ intermediate values of the underlying (levels) values of $\boldsymbol{n}$, i.e., $\boldsymbol{N}$. The intermediate values of $\boldsymbol{N}$ are obtained by successively updating the values of $\boldsymbol{N}$ after each of the i steps is applied. Once the values of $\boldsymbol{N}$ are updated for any given step, the coefficients of the $\boldsymbol{A}$ matrices in (3) are recomputed before (3) is solved again.[7]

3.3 Quarterly periodicity

MONASH-Health typically produces annual results (e.g., Brown et al. 2009). But for the work presented here the model has been modified so that it produces quarterly results. This modification is important because past influenza pandemics have had sharp effects over a short period (e.g., 3-6 months). An annual model tends to smooth out such effects leading to potential underestimation of disruption. For example, if an epidemic caused an 80% loss of inbound international tourism within a particular quarter, then the adjustment path of the tourism industry would be quite different from that in a situation in which international tourism declined by 20% for a year. Similarly, a 20% increase in a single quarter in demands for medical services related to infectious diseases would place more stress on the medical system than a 5% increase spread over a year. Fan (2003), Jonung and Roeger (2006) and Keogh-Brown et al. (2010) all apply models with quarterly periodicity to analyse economy-wide impacts of pandemics.

Annual CGE models like MONASH-Health are commonly solved in a recursive manner.[8] Dynamic-recursive models usually divide time into discrete intervals and economic variables are assumed to change at the end of each interval. Such models take the form

$$Y = G(X), \tag{4}$$

where Y and X are the levels of the endogenous and exogenous variables in a period. Computations are then carried out according to

$$\Delta Y = G'(X)\Delta X, \tag{5}$$

[7] The model is implemented and solved using the multistep algorithms available in the GEMPACK economic modelling software (Harrison and Pearson 1996).

[8] The exceptions are intertemporal models that compute results simultaneously for all time periods (e.g., McKibbin and Wilcoxen 1999).

where Δ refers to changes from one period to the next. If we have changes from one year to the next for the exogenous variables, i.e., ΔX , then to model quarterly changes we must divide these changes by four.

Besides dividing exogenous changes by four, there are three other changes required to move a discrete-time model from annual to quarterly periodicity:

a) equations must be added that handle quarterly accumulation of stock variables;
b) the base data for the initial values of lagged variables must be altered, e.g., if the data for a price variable in the base and lagged years are 1 and 0.96, then we should use 1 and 0.99 for the quarterly model;
c) parameter values in equations describing partial adjustment mechanisms must also be altered.

We describe changes (a) and (c) below.

3.3.1 Stock-flow relationships

Discrete time CGE models with annual periodicity usually have an equation linking beginning-of-period capital stocks and end-of-period capital stocks per period such as

$$KE_j^t = KB_j^t\left[1-D_j\right]+I_j^t; \qquad (6)$$

where KB_j^t and KE_j^t are the quantity of capital available for use in industry *j* at the beginning and end of year *t*, I_j^t is the quantity of new capital created (i.e., investment) for industry *j* during year *t*, and D_j is the rate of depreciation in industry *j*, treated as a parameter. With I_j^t and D_j representing annual values, KE_j^t in (6) will grow at an annual rate.

To define a quarterly rate of capital accumulation (signified here by superscript *q* rather than *t*) with no change in the values of any variable in (6), we add the following equation to the model,

$$KE_j^q = KB_j^q\left[1-D_j^q\right]+I_j^q. \qquad (7)$$

In deriving (7), we create quarterly values for depreciation, D_j^q, and investment, I_j^q, that ensure KE_j^q accumulates at a quarterly rate. To ensure that when D_j^q is applied four times over four quarters it gives the same depreciation rate as D_j applied once, we set $D_j^q = 1-\left(1-D_j\right)^{1/4}$.[9]

In calculating I_j^q, note that $I_j^t = VI_j^t \big/ PI_j^t$, where VI_j^t is the value of investment for industry *j* during year *t*, and PI_j^t is the asset price of capital for industry *j* during year *t*. So $I_j^q = VI_j^q \big/ PI_j^q$, where

$$VI_j^q = \frac{VI_j^t}{4}; \qquad (8)$$

[9] We thank Kevin Hanslow for making us aware of the correct formulation for D_j^q.

$$PI_j^q = \sum_{is} PI_{ijs}^q S_{ijs} . \qquad (9)$$

(9) defines PI_j^q as a weighted-sum of the price of good *i* used to produce capital used by industry *j* sourced from location *s* (i.e., domestic or foreign) in quarter *q*. This is similar to the definition of PI_j^t. In both cases the shares applied, S_{ijs}, are calculated from the base year data.

In the annual model, KB_j^t is set to reflect the rate of growth of the capital stock in the initial solution (i.e., the initial data). This rate of growth is function of the initial value of the capital stock and investment, and the depreciation rate. So if the initial data is for 2008, then our initial solution is for 2008 and the first year of our annual simulation will be a solution for 2009. In the quarterly model, we still use 2008 as our initial data but we apply quarterly values for depreciation rates and investment to determine the quarterly rate of growth of the capital stock in the initial solution. So our initial data period is reinterpreted as the last quarter of 2008 (2008:4) and the first period of our model simulation becomes 2009:1.

Note that (6) remains in the model and KE_j^t continues to be used in the equation that postulates a relationship between the rates of return on capital and capital growth rates. Thus, the relationship between rates of return on capital and capital growth rates (i.e., investment) is an annual one even though the periodicity of the model is now quarterly. This assumes that firms still make investment plans over a one year time horizon but only one-quarter of those plans come online in the current period. Thus, KE_j^t and I_j^t are never realised, they are only planning variables. Note also that the gestation lag for capital is a function of the periodicity in the discrete-time approach. If we assume that investment occurs smoothly through the model time period, annual periodicity assumes a gestation lag of about six months whereas quarterly periodicity assumes a gestation lag of about six weeks. It is not ideal that the gestation lag is a function of periodicity, but it is unclear how undesirable this property is.

Similar changes to those described above for the capital accumulation relationships are also made to the stock-flow relationships for debt, credit and equity.

3.3.2 Partial adjustment mechanisms

MONASH-Health contains an equation that controls the deviation of employment from the baseline whereby it is assumed that in policy simulations the deviation in the real wage from the basecase level increases at a rate that is proportional to the deviation in gap between employment and labour supply from its basecase level:

$$\left\{\frac{W^t}{W_b^t}-1\right\} = \left\{\frac{W^{t-1}}{W_b^{t-1}}-1\right\} + \beta\left\{\frac{E^t}{E_b^t}-\frac{LS^t}{LS_b^t}\right\} + U^t , \ \forall t . \qquad (10)$$

In (10), W^t and W_b^t are the real wage rates in year *t* in policy and baseline simulations, E^t and LS^t are employment and labour supply in year *t* in the policy simulation, E_b^t and LS_b^t are employment and labour supply in year *t* in the baseline simulations, U^t is a slack variable set exogenously at zero, and β is a positive parameter.

The relationship between real wage and employment deviations from baseline is controlled by β. If β is zero, the real wage stays at its baseline level regardless of labour

market conditions in the current period; if β is one, the real wage responds flexibly to clear non-zero excess demand for labour in the current period. β is usually calibrated so that the employment deviations of a shock to the economy are approximately zero after about five years, e.g., $\beta = 0.5$.[10] In a quarterly model we wish this relationship to continue to hold. So we divide the parameter by four so that the employment deviations of a shock to the economy are approximately zero after about twenty quarters, e.g., $\beta = 0.125$.

3.4 Capital idling

Earlier discussion in Section 2.2 has noted the importance of risk-modifying behaviour during pandemics that leads to reductions in demand (e.g., tourism and transportation). Previous analysis has found reductions in demand to be an important part of the negative shock to the economy from pandemics, e.g., Keogh-Brown et al. (2010). But these analyses have assumed full capacity utilisation despite the fact that variation in capacity utilisation is a prominent feature of recessions in Australia (Otto 1999) and the US (Board of Governors of the Federal Reserve System 2010). Moreover, Otto (1999) provides evidence that demand shocks explain nearly all of the variation in capacity utilisation for Australia. This suggests that modelling capacity utilisation may be important in analysing the effects of demand-contracting events. This view is supported by Dixon and Rimmer (2010) in their analysis of the current US recession using a dynamic CGE model.

Dixon and Rimmer (2010) show that assuming full capacity utilisation in each industry involves sharp reductions in rental rates on capital in response to reductions in demand. Lower rental rates on capital lead to a net capital outflow and a real exchange rate depreciation; this drives an export upturn in the short-run. This is an unrealistic response to a demand-contracting shock: for example, this is not a feature of the current US recession. Such unrealistic export responses can only be moderated by allowing for less-than-full capacity utilisation.

Variable capacity utilisation has been extensively analysed in various contexts within the real business cycle (RBC) literature (Nakajima 2005). In allowing for excess capacity here, we follow Dixon and Rimmer (2010). Their representation of idle capital is most closely related to the RBC model of Cooley et al. (1995) who represent production by a continuum of firms that each period decide whether to run a plant or not. That is, adjustment in capital used occurs along the extensive margin. The basic idea in Dixon and Rimmer (2010) is that capital rental rates are sluggishly adjusting mark-ups on variable costs (wage plus materials) that fall in response to excess capacity. Whereas the typical view of rental rates is that they represent market clearing prices for the use of existing capital stocks. We implement the former view in the policy run and this requires that we distinguish between capital in use $\left(KU_j^t\right)$ and capital in existence $\left(KE_j^t\right)$: excess capacity is where $KE_j^t > KU_j^t$. Thus, equations (6)–(7) in Section 3.3 are rewritten in terms of KE_j in place of K_j.

We allow for sticky rental rates and excess capacity in the policy run only, via

$$\left\{\frac{Q_j^t}{Qb_j^t} - 1\right\} = \left\{\frac{Q_j^{t-1}}{Qb_j^{t-1}} - 1\right\} + \alpha\left\{\frac{KU_j^t}{KE_j^t} - 1\right\} + S_j^t, \ \forall t; \qquad (11)$$

[10] This labour market assumption is consistent with conventional macroeconomic modelling in which the NAIRU is exogenous. It is also compatible with search models and efficiency-wage theory.

where Q_j^t and Qb_j^t are the rental rates for industry j in quarter t in policy and baseline simulations, S_j^t is a slack variable, and α is a positive parameter. S_j^t is used to implement (11) in the policy run only, and to turn off (11) once $KE_j^t = KU_j^t$. Note also that $Q_j^t = f\left(KU_j^t,...\right)$, $\forall t$, where f is a decreasing function.

In (11) the degree of rigidity in the rental price deviation $\left\{\frac{Q_j^t}{Qb_j^t} - 1\right\}$ is controlled by α, which determines how responsive the rental price deviation is to excess capacity per quarter: it is set at 0.05. This means that if we set all other terms to zero in (11), 10% excess capacity will cause a -0.5% rental price deviation from baseline. This gives rental prices a high degree of downward rigidity in the presence of excess capacity, which all else constant will work to remove excess capacity quickly once demand conditions begin to move towards baseline levels.

Two adjustments are also made to the investment function. First, expected rates of return on investment, $EROR_j^t$, will be lower under excess capacity than under full capacity utilisation by defining them as

$$EROR_j^t = \left\{\frac{KU_j^t}{KE_j^t}\right\} ROR_j^t - \left\{1 - \frac{KU_j^t}{KE_j^t}\right\} D_j, \ \forall t. \qquad (12)$$

That is, $EROR_j^t$ will be a weighted average of the rate of return on capital, ROR_j^t, and the negative of the depreciation rate, where the weights are the share of capital in existence that is in use and the share not in use. So we are assuming that capital not in use earns no rental and deteriorates at the depreciation rate.

Second, demands for additional capacity in quarter t (when demand for capital exceeds supply during recovery) are partially met by recommissioning idle capital from period t–1. The appropriate parameter values are set so that around 16.5% of idle capital is recommissioned per quarter to satisfy demands for additional capacity; this equivalent to two-thirds of idle capital being recommissioned per year.

3.5 Tourism demands

To facilitate capturing the effects on tourism of an influenza pandemic, we define three tourism sectors in the model: domestic, international inbound and international outbound. We apply data from Australian Bureau of Statistics (2009b) to determine the size and composition of the tourism sectors in the model data. As our base year data is for 2002, we apply data for 2001-02. Below we describe the theoretical changes to household's utility function in defining the new sectors.

MONASH-Health assumes households derive utility (U) from the consumption of i individual goods (X_i):

$$U = LES(X_i); \qquad (13)$$

where the function *LES* represents the well-known linear expenditure system. We modify (13) by adding an extra good representing tourism expenditure (X_{i+1});

$$U = LES(X_i, X_{i+1}). \quad (14)$$

The LES makes demands for X_i and X_{i+1} a function of total consumption (C), the number of households (Q) and the prices of $X_i(P_i)$ and $X_{i+1}(P_{i+1})$.

There are three tourism goods in the model: domestic, international inbound and international outbound. International inbound tourism is consumed by foreigners and so does not enter the domestic household's utility function, but it does form part of domestic firms' sales. Thus X_{i+1} only represents domestic $\left(X_{i+1}^d\right)$ and international outbound tourism $\left(X_{i+1}^i\right)$. These two tourism goods are somewhat substitutable. We handle this *a priori* behaviour by specifying

$$X_{i+1}^d = DDS\left(X_{i+1}, P_{i+1}, P_{i+1}^d\right), \quad (15)$$

$$X_{i+1}^i = DDS\left(X_{i+1}, P_{i+1}, P_{i+1}^i\right). \quad (16)$$

The function *DDS* in (15)–(16) represents Theil's (1980) differential demand system. This makes X_{i+1}^d and X_{i+1}^i a function of X_{i+1} and their relative prices. The system (15)–(16) is calibrated so that the expenditure elasticities for X_{i+1}^d and X_{i+1}^i are 0.9 and 1.3, and the price elasticities are -0.8 and -0.6.

4. Influenza scenarios

The earlier discussion of previous analyses of pandemics and their potential economic effects flagged a number of channels through which an economy could be affected by a serious outbreak of H1N1 influenza. Considering these channels, we decide on four types of economic shocks to impose on MONASH-Health to simulate an H1N1 pandemic:

(1) a surge in demand for hospital and other medical services;

(2) a temporary upsurge in sick leave and school closures requiring withdrawal of parents from the labour force;

(3) some deaths with a related permanent reduction in the labour force; and

(4) temporary reductions in inbound and outbound international tourism and business travel.

We have developed two quantitative scenarios covering factors (1) to (3), to represent the 'first wave' of infection due to a newly emerged pandemic influenza strain. The scenarios are both constructed from the classic Susceptible-Exposed-Infected-Removed (SEIR) model of infectious disease transmission (Kermack and McKendrick 1927; Anderson and May 1992) with allowance for unobserved (mild and non-presenting) transmission and pre-existing immunity in a portion of the population. The number of people in the population who are susceptible (*S*), exposed (*E*), infectious (*I*) or recovered (*R*) are described by a set of coupled non-linear first-order differential equations and associated initial conditions:

$$\frac{dS}{dt} = -\beta IS,$$

$$\frac{dE}{dt} = \beta IS - a\gamma E ,$$

$$\frac{dI}{dt} = a\gamma E - \nu I ,$$

$$\frac{dR}{dt} = \nu I ;$$

where β is the production rate of new exposed individuals, γ is the transition rate from the exposed to infectious class (and so $1/\gamma$ is the average duration spent in the exposed class), ν is the transition rate from infectious to recovered ($1/\nu$ is the average duration of infectiousness) and α is the proportion of all infections that present to health service providers (i.e., the proportion of infectious individuals that are *not* mild or non-presenting). The initial conditions are $S(t=0) = (1-z)N$, $E(t=0) = 0$, $I(t=0) = 10$ and $R(t=0) = zN$, where N is the population size and z is the proportion of the population immune to infection prior to the onset of the pandemic.

The infection rate, driving the S to E transition, is proportional to the product of S and I and so the model is non-linear. Given an initial reproduction number (the number of secondary cases arising from a primary case, given by β/ν) that is greater than 1, the model will naturally show an initial exponential increase in the proportion of the population that is infected. The epidemic peaks when the reproduction number is equal to 1 (i.e., when $S(t) = \nu/\beta$), and then declines due to exhaustion of the susceptible pool, returning to a steady state $(I = 0)$. The initial reproduction number is tuned to provide the desired total number of infections over the course of the epidemic. The time scale of the epidemic is set by adjusting the serial interval $1/\gamma + 1/\nu$, which is the average time between infection of one individual and subsequent infection of the next.

4.1 Scenario 1: the 2009 outbreak

The first scenario is calibrated, wherever possible, on available data for the 2009 H1N1 outbreak as of October 2010 (Department of Health and Ageing 2009). A number of serosurveys (Gilbert et al. 2010, McVernon et al. 2010) covering the Australian population indicate that approximately 11% of the Australian population were infected in the course of the 2009 Australian pandemic,[11] which is higher than the values used for planning purposes in the *Australian Health Management Plan for Pandemic Influenza* (Department of Health and Ageing 2008). Twelve per cent of the population were assumed to be protected prior to the outbreak, based on serological data (McVernon et al. 2010). The 2.2 million Australians infected with H1N1 experience symptoms over 2009:2–2009:4 (i.e., quarters 2–4 of 2009). The dynamics of the outbreak meant that 87% of all new infections occurred in 2009:3; the shocks described below reflect these dynamics. In formulating these shocks we have deliberately chosen conservative assumptions that give smaller economic disruptions. We feel this is appropriate given the unsettled nature of researchers' understanding of how influenza pandemics affect

[11] We include the following multiplicative factors to allow for the influenza-like illness presentation rate to differ from the true H1N1 rate: 5 for general practitioners and flu clinics, and 2 for non-ICU hospital admissions. These factors were chosen based on expert opinion and consultation with government during the early phases (May–June 2009) of the Australian outbreak (see Table 3).

human behaviour. Given the scenario parameters, we assume the following shocks to the economy.

(1) That out of the 2.17 million people who experience symptoms: 1.96 million seek no medical attention but spend $5 (2009 dollars) on pharmaceuticals; 0.21 million seek medical attention (by visiting a general practitioner or a flu clinic) but are not hospitalised, incurring expenses of $61 (2009 dollars); 4,305 are hospitalised and survive, incurring expenses of $3,564 (2009 dollars); 700 are hospitalised and spend time in an ICU, incurring expenses of $85,395 (2009 dollars), of which 506 survive and 194 die.[12] The presentation venues for the 2.17 million symptomatic individuals have been chosen to reflect the official Australian presentation counts. Table 3 shows the assumed proportional split of influenza-like illness (ILI) presentation to different venues, and the values are calibrated to reproduce the 2009 Australian experience. Altogether medical expenses are $100 million (2009 dollars) incurred over May–December 2009. This amounts to a 6.3% increase in demand for medical services relating to respiratory treatments over 2009:2–2009:4. In MONASH-Health, respiratory treatments are mainly comprised of four inputs: human pharmaceuticals, hospital and nursing services, doctor services, and ambulance services. Thus, it these services whose demand will be mostly affected by the increased demand for respiratory treatments. We assume that demand for respiratory treatments returns to normal in 2010:1.

(2) That workers miss a total of 1.44 million workdays over 2009:2–2009:4 on account of their own sickness including 0.4 million days while caring for children who are either sick or kept home by school closures. This assumes: 0.5 workdays are lost per working age person who experiences symptoms and seeks no medical attention; 2.4 workdays are lost per working age person who seeks medical attention (by visiting a general practitioner or a flu clinic); 13.9 workdays are lost per working age person who is hospitalised. Working parents caring for homebound children are assumed to miss a comparable number of workdays except in the case of hospitalisation where half as many days are missed.[13] Workdays lost by parents while caring for children are scaled for the share of families with all parents working (Australian Bureau of Statistics 2009a). The total loss in workdays translates to a reduction in labour productivity of 0.22% (= 1.44 million days out of the 646 million days available from 10.77 million workers supplying 60 days each quarter). We assume that labour productivity returns to normal in 2010:1. Note that our workday losses assume neither prophylactic absenteeism nor lower productivity by workers may not present to the health system but will be less productive than would otherwise be the case, both of which would increase the workday losses.

(3) That of the 194 persons who die, 78 are workers. This translates into a permanent reduction in the labour force of 0.0007% over 2009:2–2009:4.

[12] When the 2009 outbreak occurred the government announced a H1N1 vaccination program; our estimates do not include vaccination expenditure due to the lack of available data.

[13] Per capita workday losses (for workers and parents) were suggested by Molinari et al. (2007) for working age people (18-64 years) in their study of seasonal influenza; the estimates take account of workforce participation rates.

Table 3 Key parameter values for the simulated epidemics in scenarios 1 and 2

Parameter	Scenario 1	Scenario 2	Comment
Percentage of population infected with H1N1 and displaying symptoms	11	30	Scenario 1: parameter values are based on estimates from historical (20th century) influenza pandemics, published pandemic preparedness modelling studies and expert opinion.
Percentage of H1N1 cases that are mild	95	75	
Percentage that are moderate	4	15	
Percentage that are severe	1	10	Scenario 2: parameter values are based on the assumption that the proportion infected with H1N1 is increased threefold relative to scenario 1 1 and by 50% relative to the primary scenario considered in the AHMPPI[a].
Non-presenting, GP, flu clinic split for mild cases (%)	99, 0.5, 0.5	99, 0.5, 0.5	
Non-presenting, GP, flu clinic split for moderate cases (%)	90, 5, 5	90, 5, 5	
Proportion of severe cases allocated to GP, flu clinic, hospital (non-ICU), ICU (%)	37.5, 37.5, 21.5, 3.5	32, 33, 30, 5	Scenario 1: splits to hospital and ICU set to reflect recorded presentations. Scenario 2: splits are skewed to reflect more hospitalisations.
Proportion of ICU admissions resulting in death	28	56 (a further 20% of hospitalised cases die)	Scenario 1: as given in the national data set. Scenario 2: 0.26% of the Australian population dies, a value consistent with the case-fatality-rate in the Western world during the 1918-19 influenza pandemic.
Total ILI cases per true H1N1 case at GP, flu clinic, hospital and ICU (multiplicative factor)	5, 5, 2, 1	5, 5, 2, 1	Based on early findings of a surge in ILI (non-H1N1) presentations to these venues, presumably due heightened public awareness and concern.

[a] *Australian Health Management Plan for Pandemic Influenza* (Department of Health and Ageing 2008).

(4) We assume that during the three peak quarters of infection and cost that inbound and outbound tourism are negatively affected. Here we follow previous estimates of the effects of SARS and the Iraq war on Australian tourism for 2003 by Dwyer et al. (2006). Given that these estimates are for both the effects of SARS and the Iraq war, we conservatively assume half the effects estimated by Dwyer et al. (2006), i.e., during the first two peak quarters of infection (2009:2–2009:3) that inbound tourism is negatively affected by 9.2% and 2.2%. For outbound tourism (including business travel), we think it reasonable to posit that some potential Australian travellers would be dissuaded from international travel by fears of becoming symptomatic on vacation. Thus, we assume that the outbound tourism effect is half that of the inbound effect. Tourism numbers stay at their recessed levels in 2009:4 and then recover smoothly to their baseline levels over 2010:1–2010:2. Australians who cancel their outbound tourism redirect their

purchases to other goods. This is an optimistic assumption; a pessimistic assumption is that consumers save their money until the pandemic ceases.

Note that part of the shocks for scenario 1, in terms of increased demand for medical services and absenteeism, are not that dissimilar to the expected shocks that would occur every year due to influenza and ILIs. So the true impact on demand for medical services and absenteeism may be somewhat less than we have predicted.

4.2 Scenario 2: a severe outbreak

The second scenario is based on a severe H1N1 outbreak assumed to occur during the same period as scenario 1, i.e., 2009:2–2009:4 and similar to the more severe scenarios considered in modelling work that informed the *Australian Health Management Plan for Pandemic Influenza* (AMHPPI). The proportion infected with H1N1 is increased to 30% of the population or 6 million individuals, a 50% increase over the baseline case assumed in the AMHPPI and a three-fold increase over that observed in 2009. To account for non-H1N1 ILIs, the total number of symptomatic infections is 8.1 million. Given the scenario parameters, we assume the following shocks to the economy.

(1) Out of the 8.1 million people who experience symptoms: 5.3 million seek no medical attention but spend $5 (2009 dollars) on pharmaceuticals; 2.6 million seek medical attention (by visiting a general practitioner or a flu clinic) but are not hospitalised, incurring expenses of $61 (2009 dollars); 180,000 are hospitalised and survive, incurring expenses of $3,564 (2009 dollars); 64,000 are hospitalised and spend time in an ICU, incurring expenses of $85,395 (2009 dollars), of which 11,000 survive and 53,000 die (see Table 3 for the proportional breakdown of presentations). Altogether medical expenses are $2.4 billion (2009 dollars) incurred over May–December 2009. This amounts to a 154% increase in demand for medical services relating to respiratory treatments during the period 2009:2–2009:4. We assume that demand for respiratory treatments returns to normal in 2010:1.

(2) That workers miss a total of 7.9 million workdays over 2009:2–2009:4 on account of their own sickness and a further 3.8 million days while caring for children who are either sick or kept home by school closures. The total loss in workdays translates to a reduction in labour productivity of 1.8%. We assume that labour productivity returns to normal in 2010:1.

(3) That out of the 52,000 persons who die, 21,000 are workers. This translates into a permanent reduction in the labour force of 0.19% over 2009:2–2009:4.

(4) That inbound tourism falls by 39% over 2009:2–2009:4 and then recovers smoothly to its basecase level over the next four quarters. In setting the shocks for inbound tourism we considered the experience in Asia during the SARS epidemic of 2003. This episode suggests that regions suffering a widespread influenza infection could incur reductions in inbound tourism in the range 20%–70% during the peak infection period (Pine and McKercher 2004; Wilder-Smith 2006). For our hypothetical severe H1N1 epidemic, we adopt a number towards the lower end of this range (39%). As in scenario 1, we assume that the outbound tourism (and business travel) effect is half that of the inbound effect over 2009:2–2009:4. Outbound tourism then recovers smoothly to its basecase level over the next four quarters. Australians who cancel their outbound tourism redirect their purchases to other goods.

Although both scenarios simulate a global H1N1 pandemic, we are unable to explicitly apply pandemic shocks to countries other than Australia in our model. This limitation may overstate the extent to which Australia's international trade in goods and assets is affected by the

pandemic and the size of the impacts on economic activity. We cannot overcome this limitation within the present framework but we do flag the importance of this limitation in the discussion of results below.

5. Results

5.1 Scenario 1

The main effects of the 2009 outbreak occur in 2009 and peak in 2009:4, by which time GDP and employment are 0.9% and 0.7% below baseline (Table 4); the size of the deviations over 2009:2–2009:4 reflect the dynamics of the pandemics in terms of new infection rates per quarter. The decline in GDP relative to employment reflects the loss in labour productivity and the stronger fall in capital in use relative to employment. On average through 2009, the epidemic reduces GDP and employment by 0.5% and 0.4%. Through 2010, GDP is a little lower even though employment is unchanged. This reflects the slow return to full capacity after the pandemic ends; thus capital in use is 0.2% below baseline through 2010.

Table 4 Scenario 1 effects (percentage deviation from baseline)

Variable	2009				2010				
	Q2	Q3	Q4	Average	Q1	Q2	Q3	Q4	Average
GDP	-0.3	-0.8	-0.9	-0.5	-0.4	0.0	0.1	0.1	-0.1
Employment	-0.3	-0.6	-0.7	-0.4	-0.4	0.1	0.2	0.1	0.0
Capital in use	-0.3	-0.7	-0.9	-0.5	-0.4	-0.1	-0.1	-0.1	-0.2
Capital in existence	0.0	0.0	0.0	0.0	0.0	-0.1	-0.1	-0.1	-0.1
Investment	-0.2	-1.2	-2.1	-0.9	-1.9	-0.6	0.1	0.1	-0.5
Consumption	-0.3	-0.6	-0.8	-0.4	-0.4	-0.1	0.0	0.0	-0.1
Exports	-1.0	-1.5	-1.1	-0.9	0.4	0.5	0.2	0.2	0.3
Imports	-0.7	-1.3	-1.5	-0.9	-0.8	-0.1	0.1	0.1	-0.2

Source: MONASH-Health database.

The epidemic reduces investment by 0.9% through 2009. In 2009:2 investment falls below baseline (-0.2%) because demand-contracting (tourism) and cost-increasing (labour productivity) shocks reduce the rental value of capital. This damps expected rates of return and thereby reduces investment. In 2009:2 the epidemic causes excess capacity to appear in some industries (Figure 1), particularly those related to tourism and construction. Excess capacity in 2009:2 has a strongly negative effect on investment in 2009:3. Weak investment in 2009:3 causes further excess capacity to appear, explaining weak investment in 2009:4. In 2010:1, much of the pick up in demand for capital associated with the recovery in labour productivity and the start of the recovery in tourism is satisfied by working down the excess capacity that appeared in 2009:2–2009:4. With excess capacity peaking in 2009:4, investment starts to move back towards baseline in 2010:1. This is because excess capacity in 2010:1 is declining as capital in existence adjusts down and capital in use adjusts up. By 2010:2, excess capacity is eliminated.

Through 2011, Figure 1 shows average deviations for GDP and employment of 0.0% and 0.1%. The epidemic-related reduction in employment in 2009 causes real wage rates to be lower than they otherwise would have been. This allows the Australian economy to arrive in 2010 with enhanced international competitiveness so that when tourism recovers and the other epidemic-

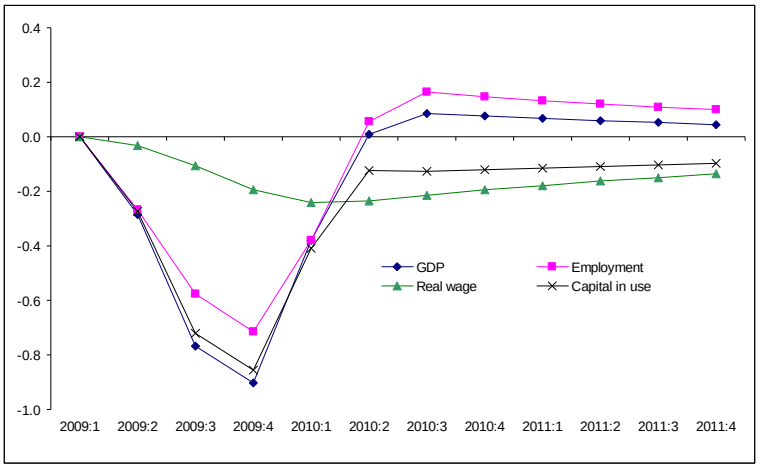
related shocks disappear, employment and output move above their baseline values. The improvement in Australia's international competitiveness is likely to be tempered by a global H1N1 pandemic that affects Australia's trading partners and competitors, i.e., their real wage rates are also likely to move below baseline, but likely with different dynamics. This would make Australia's exports and domestically-produced goods less competitive than suggested by our results here. It is difficult to account for these trading partner and competitor effects in a national model like the one we are applying here.

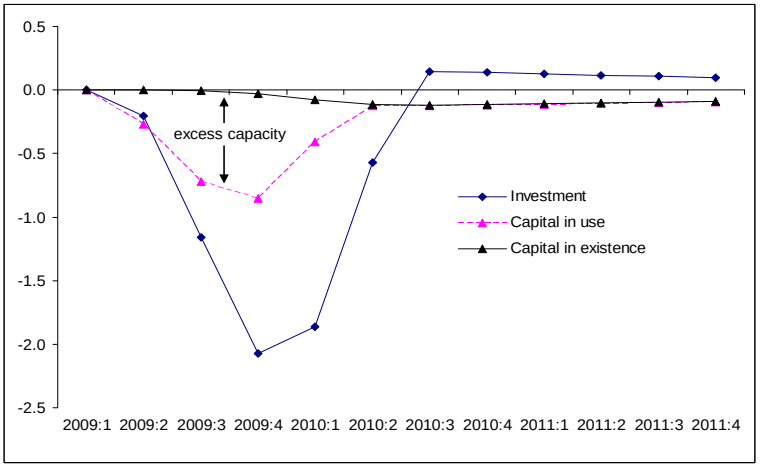
Figure 1 indicates the relative importance of the different shocks in determining the overall employment effects. We see that H1N1 deaths have an almost imperceptible effect on aggregate employment due to their small number. Increased expenditure on medical services and away from other items of household expenditure has slightly larger employment effects: employment is a little higher in the short run. This is because production of medical services is considerably more labour intensive than production of most other items of household expenditure. Extra medical expenditures in 2009 have a small negative effect on aggregate employment in 2010. This is a reflection of the wage mechanism mentioned earlier: extra employment in 2009 associated with medical expenditures weakens the competitive advantage that the Australian economy experiences in 2010. We see that the main drivers of lower employment are lost workdays and the fall in international tourism, with each effect contributing roughly equally to the overall employment loss of 0.8%.

Figure 2 compares our results for scenario 1 with the typical CGE assumption of full capacity utilisation (FCU); the differences in GDP effects are significant. Dropping the assumption of variable capacity utilisation (VCU) reduces the peak GDP effect from -0.9% to -0.28%. As discussed in Section 3.4, assuming FCU involves sharp reductions in rental rates on capital in response to reductions in demand. Lower rental rates on capital lead to a net capital outflow and a real exchange rate depreciation; this drives an export upturn in the short-run. We see this mechanism at play in Figure 2 where under FCU the real exchange rate[14] depreciates initially and stays below baseline through 2009 and 2010, compared to no initial depreciation with VCU. Thus, exports fall less with FCU. Although exports decline sharply by 2009:3 with VCU, reflecting the 8% reduction in inbound tourism, exports begin to recover by 2009:4 even though inbound tourism does not fully recover until 2010:2. The recovery of exports is assisted by the real exchange rate depreciation that begins in 2009:4 that is associated with the peak effect on investment.

[14] The real exchange rate is measured by movements in the Australian price level compared with price levels in trading partners expressed in a common currency. Negative movements in the real exchange rate indicate improvements in the international competitiveness of the Australian economy.

Figure 1 Scenario 1 effects on selected aggregates
(percentage deviations from baseline)





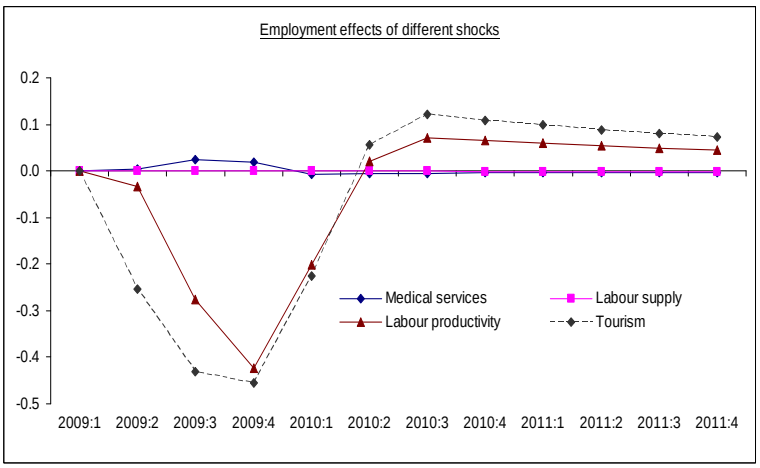
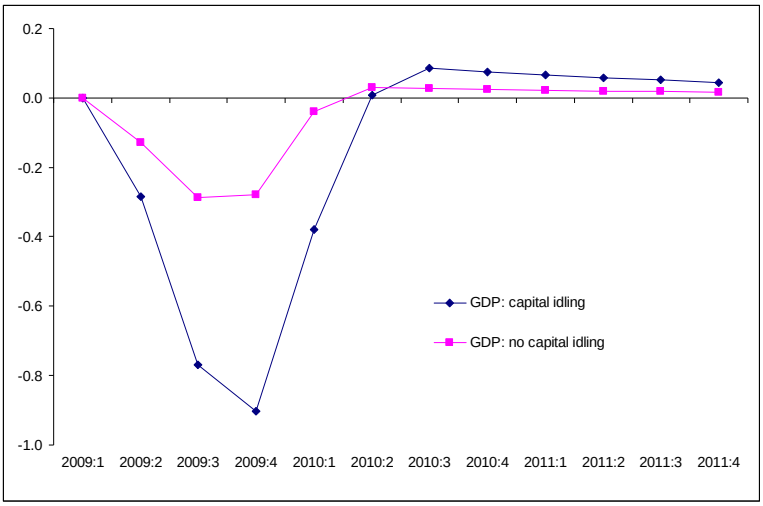
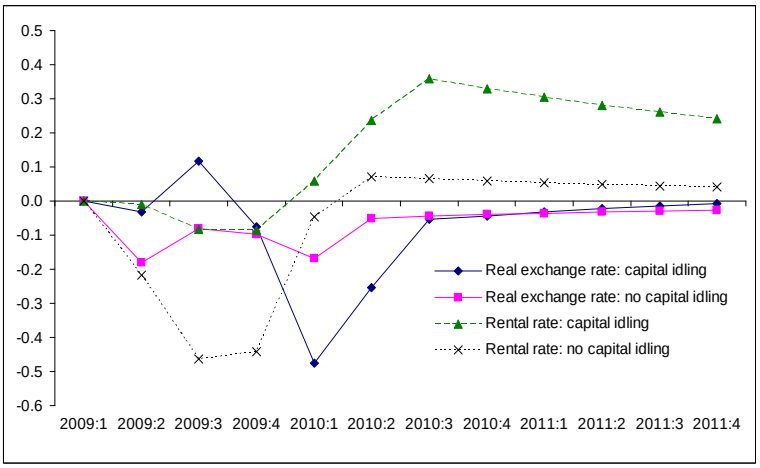
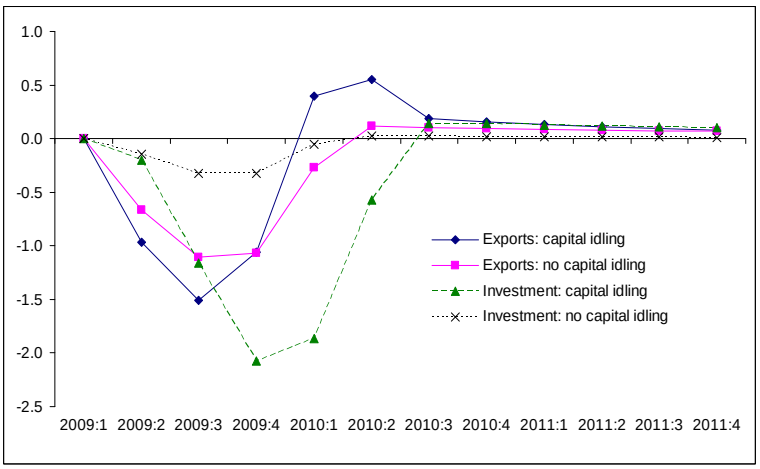


Figure 2 Scenario 1 effects with variable and full capacity utilisation
(percentage deviations from baseline)







5.2 Scenario 2

The severe outbreak has similar dynamics to the 2009 outbreak, and so its peak effect also occurs in 2009:4. Importantly, however, the effects are much larger with GDP and employment 6.2% and 4.1% below baseline in 2009:4 (Table 5). These effects are between six and seven times larger than the peak effects of the 2009 outbreak. This is a combination of two major differences between the two outbreaks: (i) the clinical attack rate is three times higher in the severe outbreak; (ii) the proportion of infected cases that are moderate or worse is five times higher in the severe outbreak. On average through 2009, the severe outbreak reduces GDP and employment by 3.6% and 2.2%. Through 2010, the economy recovers strongly but is still recessed; GDP and employment are 0.7% and 0.4% below baseline. As observed in the 2009 outbreak, the recovery is impeded by a slow return to full capacity after the pandemic ends. The slow return to full capacity is impeded here by the slow recovery in tourism numbers through 2010.

Table 5 Scenario 2 effects (percentage deviation from baseline)

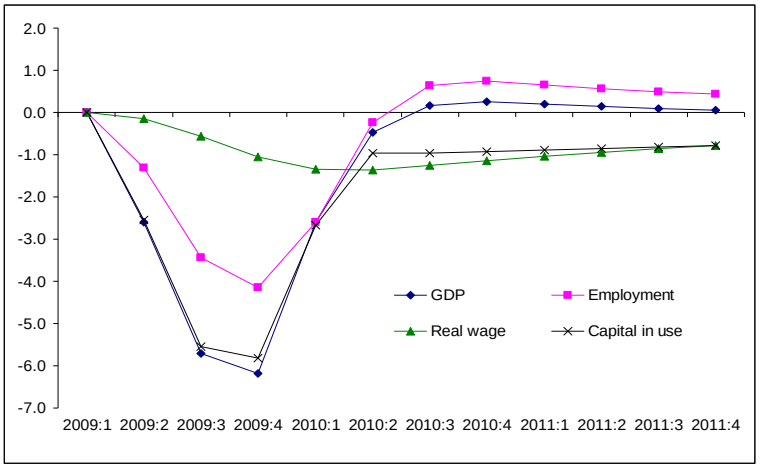
Variable	2009				2010				
	Q2	Q3	Q4	Average	Q1	Q2	Q3	Q4	Average
GDP	-2.6	-5.7	-6.2	-3.6	-2.6	-0.5	0.2	0.2	-0.7
Employment	-1.3	-3.4	-4.1	-2.2	-2.6	-0.2	0.6	0.7	-0.4
Capital in use	-2.5	-5.5	-5.8	-3.5	-2.7	-1.0	-1.0	-0.9	-1.4
Capital in existence	0.0	0.0	-0.3	-0.1	-0.6	-0.8	-0.9	-0.9	-0.8
Investment	-1.9	-8.8	-14.6	-6.3	-12.2	-4.2	0.3	0.6	-3.9
Consumption	-1.8	-4.4	-5.0	-2.8	-2.8	-0.7	0.0	0.1	-0.9
Exports	-6.2	-9.2	-5.8	-5.3	2.4	1.5	0.1	0.5	1.1
Imports	-3.0	-6.8	-8.1	-4.5	-5.1	-1.9	-0.3	0.4	-1.7

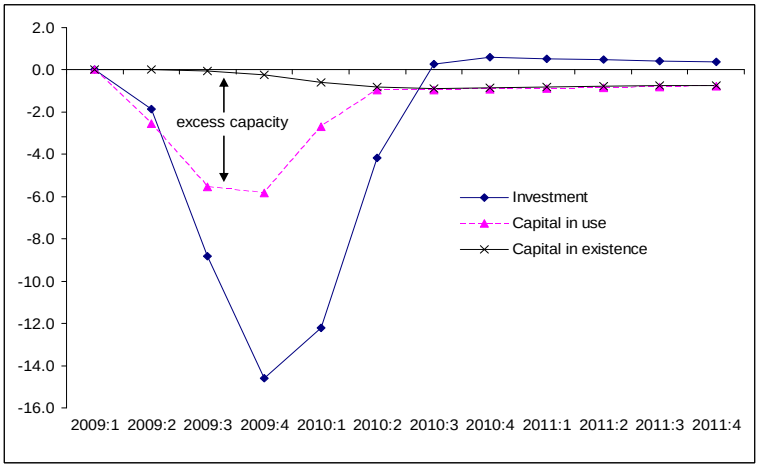
Source: MONASH-Health database.

An important contributor to the larger effects in the severe outbreak is the much greater peak effect on investment: -14.6% versus -2.1% in 2009:4. With a much larger fall in investment, a much larger level of excess capacity emerges in 2009 (Figure 3). Like investment, excess capacity peaks in 2009:4. From 2010:1, investment starts to move back towards baseline reflecting the beginning of the pickup in capital in use once the pandemic ends. At the same time, capital in existence continues to adjust down. Excess capacity is eliminated by 2010:3 and so investment moves above baseline.

Comparing the relative importance of the different shocks in determining the overall employment effects (Figure 3), we see that H1N1 deaths have almost no effect on employment in the short run but have a small negative effect in the long run. Short-run results in MONASH-Health are mainly demand driven and the deaths have little effect on aggregate demand. In the longer run, employment is determined mainly by labour supply: demand for labour adjusts to changes in supply via wage movements. Beyond 2010, H1N1-related deaths in 2009 reduce employment by reducing labour supply. Increased expenditure on medical services and away from other items of household expenditure has similar effects in the severe outbreak to the 2009 outbreak: a positive effect on employment in the short run; a small negative effect on aggregate employment in 2010.

Figure 3 Scenario 2 effects on selected aggregates
(percentage deviations from baseline)





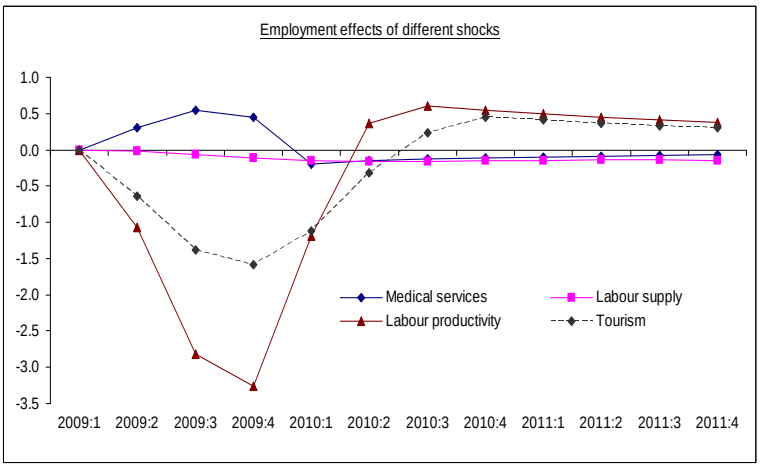
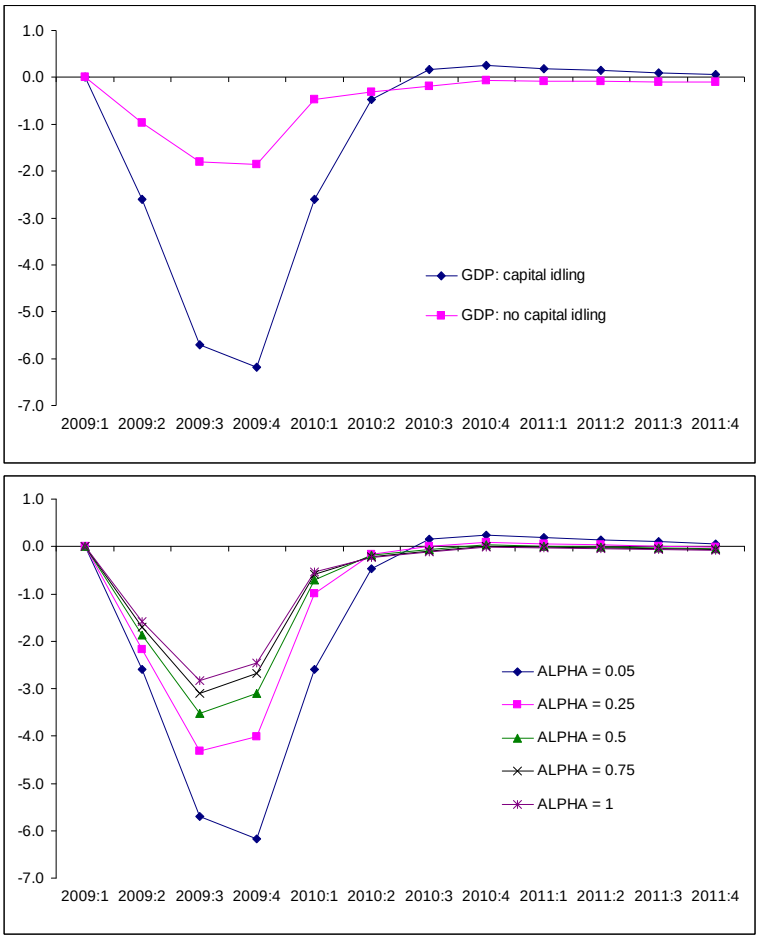


Figure 4 Scenario 2 effects with variable and full capacity utilisation
(percentage deviations from baseline)



Similar to scenario 1, the main drivers of lower employment are lost workdays (labour productivity) and the fall in international tourism, but now lost workdays are much more employment-reducing than lower tourism; 3.3% versus 1.6% in 2009:4. This reflects the assumption that the risk-modifying behaviour leading to lower tourism is not assumed to increase in direct proportion to infection rates as we move from the 2009 outbreak to the severe outbreak. It is not obvious whether this is an appropriate assumption. Nevertheless, as the simulated effects are approximately linear in the shocks, we know that larger tourism effects would lead to larger adverse employment effects.

Figure 4 shows, like in scenario 1, the assumption of VCU or FCU leads to very different GDP deviations from baseline. Assuming VCU gives a peak GDP effect of -6.2% in 2009:4; assuming FCU gives a peak GDP effect of -1.9%. The mechanism driving this difference is that already outlined earlier. The relative differences in peak GDP effects between the VCU and FCU simulations for scenario 2 are much smaller than those observed for scenario 1. This suggests that the degree of rental price stickiness may be important in determining the depth of the trough in economic activity when a pandemic hits (see the parameter α in equation (11)). We test this assumption by varying α from its current value of 0.05 through to 1 (Figure 4). We see that increasing the value of α reduces the size of the GDP effect but by decreasing amounts.

6. Discussion

Our results show that the possible GDP effects of a mild H1N1 pandemic (scenario 1) are in the range -0.5% to 0% in the peak year, depending on the degree of capacity utilisation assumed. For a severe H1N1 pandemic, our results range from -3.6% to -1.2%. The results are not directly comparable to most previous studies as, as far as we are aware, there are no studies that have estimated the economic effects of the 2009 H1N1 pandemic for Australia or other countries. There are a number of studies that have analysed past influenza pandemics and hypothetical future influenza pandemics. Most recently, Dixon et al. (2010) estimate the economic effects of a serious H1N1 epidemic for the US; their work showed a GDP effect of -1.6% in the peak year. Keogh-Brown et al. (2010) estimate the effects on the UK of the 1957 or 1968 influenza pandemics could affect GDP by -0.58% in the peak year, and a more extreme scenario could increase this to -4.5% or -6%. The estimates for both of these studies are strongly driven by the lost workdays and reduced consumption by households; this is also true for our analysis. Jonung and Roeger (2006) conduct a similar study to Keogh-Brown et al. (2010) but estimate the effects of mild and severe influenza pandemics on the European economy. Their estimates of GDP effects in the peak year range from -1.1% to -4%. McKibbin and Sidorenko (2006) estimate the global effects of a broad range of pandemic influenzas. For Australia they estimate GDP effects of -0.8% to -10.6%.

Our peak GDP effects are within the range of estimates of previous studies cited above. The lower bound of our estimates is smaller than the lowest estimate of the previous studies, e.g., 0% viz. -0.58%. Moreover, the upper bound of our estimates is only one-third as large as the highest estimate of previous studies, e.g., -3.6% viz. -10.6%. There are differences across all these studies including ours. In terms of the analytical framework, differences relate to periodicity, sectoral detail, and capacity utilisation. In terms of the scenarios modelled, differences relate to infectiousness (infection rates), virulence (death rates) and timing. Further differences relate to the nature of the shocks imposed for a given scenario, e.g., the degree of risk-modifying behaviour undertaken in response to the pandemic. This last set of differences across studies is legitimate as researchers have well-grounded reasons for assuming different degrees of risk-modifying behaviour and the response by authorities. Our approach to estimating pandemic responses is conservative and we have adopted responses toward the middle of the range of previous studies due to uncertain nature of the response by people to pandemics. This approach is reflected in the fact that our estimates of economic disruption are low compared to previous studies.

7. Concluding remarks

We apply a quarterly CGE model to simulate the economic effects on the Australian economy of two H1N1 epidemics; the relatively mild 2009 outbreak and a more severe episode infecting about 8 million Australians. The use of a model with quarterly periodicity rather than the usual annual periodicity allows the analysis to capture the short-run nature of influenza pandemics. Such an event would have its initial economic effects concentrated over no more than one or two quarters.

Our analysis demonstrates that an H1N1 pandemic could have significant macroeconomic effects. It is likely that it would reduce household demands for international travel and demands by international tourists for hotels, travel and other services within Australia. It is also likely that industries would face increased costs via absenteeism. Both the demand decreases and cost

increases associated with an epidemic of the size assumed here could be expected to cause a sharp reduction in investment with resulting adverse effects on employment and GDP. The simulations show reductions in GDP and employment in the peak quarter of the assumed epidemic as: 0.9% and 0.7% for the 2009 epidemic; and 6.2% and 4.1% for a severe epidemic. The size of these effects is highly dependent on the degree of capacity utilisation assumed. The analysis also demonstrates that the sharp downturn in economic activity would be short-lived with employment and GDP returning to business-as-usual growth rates within two to four quarters, depending on the severity of the epidemic.

It should be noted that our analysis does not take into account a number of responses that are difficult to quantify but are likely as the severity of the epidemic increases. These are: the reaching of capacity constraints for hospitals in general and ICUs in particular; the change in risk-modifying behaviour as deaths become a much larger proportion of population; and the ameliorating effects of policy responses such as vaccination, prophylactic anti-viral medications and their costs.

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