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Deaths and the marginal changes in health care costs

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Abstract

Changes in the absolute number of deaths (all-cause mortality) act via their impact on end-of-life care to affect marginal changes in NHS medical admissions, bed occupancy and costs. These effects are poorly understood. A very large and unexpected increase in deaths in 2015 offers the opportunity to explore the reasons for this and previous examples of periods of increased deaths. While the event in 2015 was large it is simply a further example of a long time series of similar events. An influenza outbreak in early 2015 did make an additional contribution, however deaths had already begun to increase around mid-2014. Interaction between the agent causing the earlier increase in deaths and influenza appear highly likely. The resulting complex short-term trends in death imply that health and social care costs will show far higher volatility than that implied by simple demographic change.

Key Words: End-of-life, medical admissions, all-cause mortality, health care costs, dementia, influenza

Introduction

In a bid to contain rising health care costs NHS England has mandated that health and social care organisations across England work together to implement a series of Sustainability & Transformation Plans (STPs) (Kings Fund 2016). The hidden assumption behind STPs is that changing the way in which health and social care is delivered will deliver sustainable reductions in health and social care costs, i.e. will not only reduce costs but will reduce the slope of the alarming rise in acute emergency admissions seen in recent years (Jones 2016i,j).

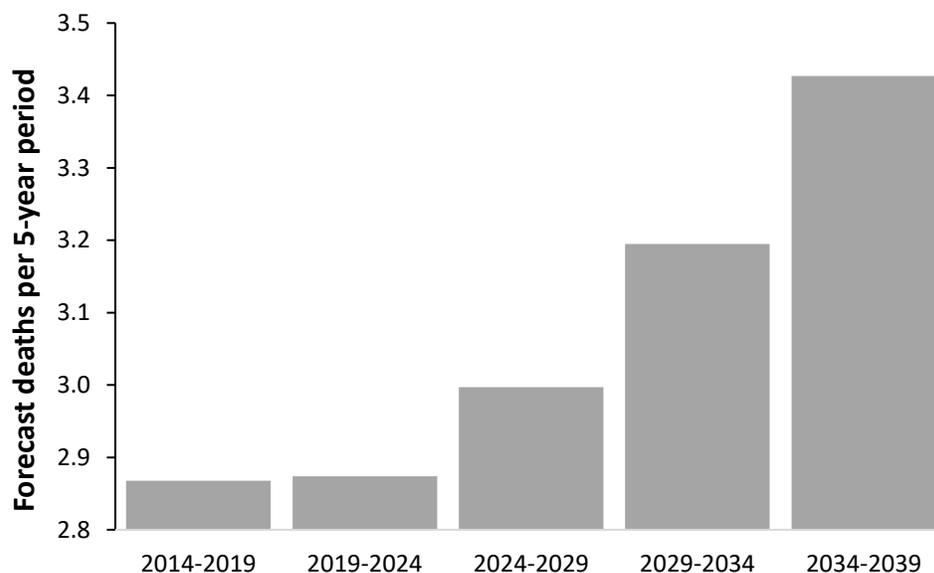
A somewhat poorly-recognised aspect of health care costs lies in the fact that marginal changes in death (via end-of-life care) have a major impact on the NHS leading to marginal changes in medical admissions, bed occupancy and costs (Jones 2012, 2015d-g, 2016c,f). Hence it is the absolute number of deaths in a particular location which drives the marginal change in costs and not the age standardized mortality rate *per se* (Karamanidis et al 2007, Payne et al 2007, Forget et al 2008, Rosenwax et al 2011). This appears to contradict the widely held assumption that it is population demography (the ageing population) which drives costs, however, the

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reality is that both probably contribute to rising costs. The fact that the last year of life (irrespective of age at death) is the most expensive leads to a consideration of both long-term trends in death, and more short-term trends which may contribute to unexpected surges in costs. Interestingly, the NHS funding formula does not contain any recognition for the role of deaths in local costs.

Figure 1 shows that the Office for National Statistics (2015) expects that the number of deaths per annum will markedly rise beyond 2020. Hence the end-of-life component of health and social care costs is set to markedly escalate in the near future. Having surveyed the expected long-term trends, the short term trends and associated volatility in the end-of-life component of costs can now be explored.

Figure 1: ONS forecast trend in deaths per five-year period in the UK (millions)



Unexpected Surges in Deaths

In this respect, deaths in 2015 displayed the highest increase over the previous year (+5.6%) since 1967 to 1968 (+6.3%) (Office for National Statistics 2016). Age-standardized mortality rates likewise showed a large increase, and females were affected more so than males. The increase was partly due to a large influenza event in January 2015 and the rest of the increase is unexplained. An 'unexpected' large increase in deaths for those suffering from Alzheimer's and dementia also seemed to be a characteristic of this event (ONS 2016, Jones 2015e, 2016i).

To unravel what is happening we need to be aware of several facts.

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1. Influenza vaccination is generally effective in reducing both primary care consultations for influenza-like-illness and for hospital admission, however, the effectiveness diminishes with age, and reduces fairly rapidly after vaccination (Cowling et al 2016).
2. The jury is out as to whether influenza vaccination makes a positive effect against mortality, as opposed to morbidity (Doshi 2008).
3. If there is antigenic shift between vaccine formulation/preparation and vaccination then vaccine effectiveness can be seriously reduced or even go negative.
4. The vaccine administered in late 2014 showed negative effects in China and Canada (Qin et al 2016), and in Canada those suffering the greatest negative effects were the recipients of regular vaccination in the previous winters (Skowronski et al 2016).

Based on the above there will undoubtedly be claim and counter-claim over the extent of the contribution of vaccination to the large January spike in deaths in 2015. However, the associated increase in end-of-life associated admissions will have led to an unwelcome increase in costs (Jones 2015f, 2016c).

The next issue of relevance is research into outbreaks of a presumed novel infectious agent leading to periods of higher deaths, medical admissions and health care staff sickness absence in the UK, and elsewhere in the world (Jones 2015a-g, 2016e,f). Key features are:

1. Relatively slow spatial spread and complex time effects, leading to the outbreaks hiding themselves within the complex spatiotemporal patterns so generated (see reviews Jones 2015b,d, 2016a,d)
2. A potential interaction between this agent and the devastating World War I Spanish flu pandemic (Jones 2016b).
3. In- and Out-of-hospital deaths are both affected (Jones 2015c, 2016g)

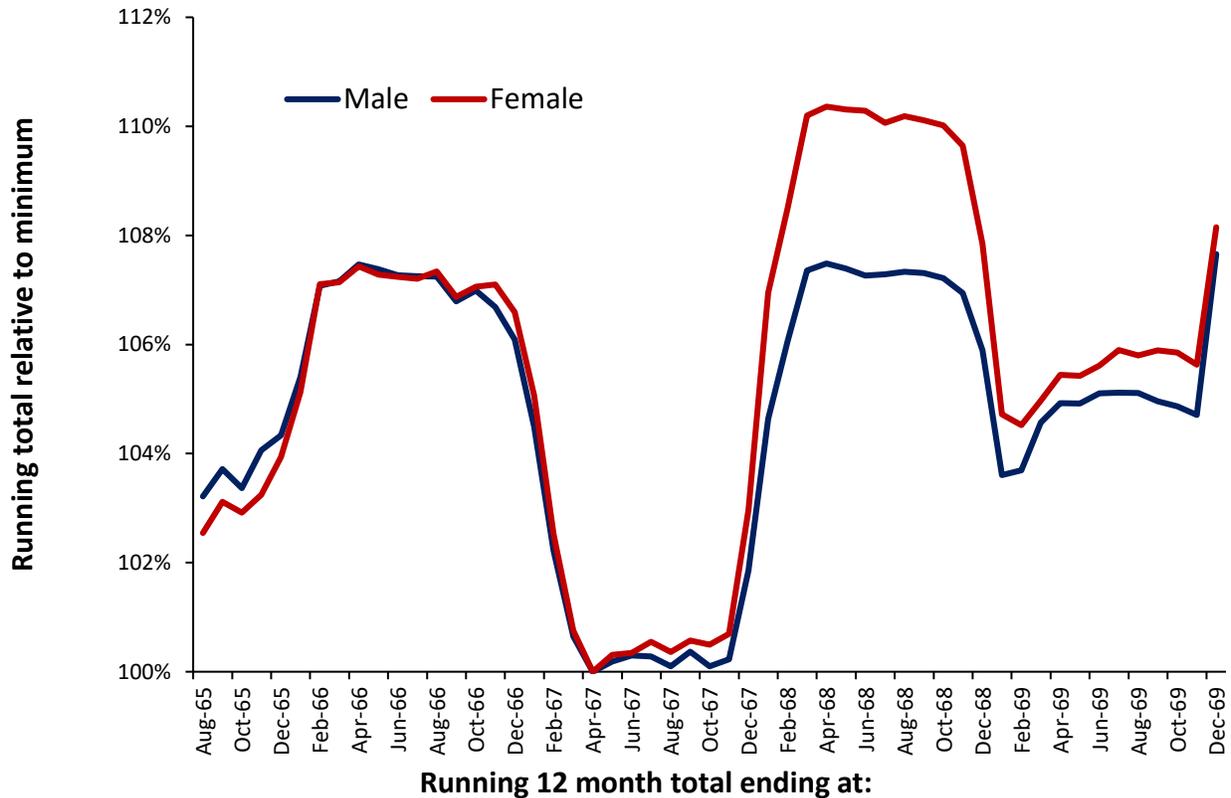
Analysis of the trends

With the above in mind, we can now examine the detail of the trends using running 12 month, 52 week and 365 day totals. A running annual total is exceedingly useful for detecting the onset and magnitude of hidden step-like increases in deaths (especially where there is background seasonal behaviour), however, it has the disadvantage of transforming the trend such that:

1. A spike event such as a seasonal influenza outbreak is transformed into a 'table top' or 'plateau' as the spike enters the running total, moves backward in the running total and then finally leaves the running total at a point 12 month on from the start of the spike event.
2. A genuine step-increase in deaths, however, shows up as an inverted 'V', as additional month of increased deaths are added to the running total in the upward slope of the inverted 'V', while at cessation, is diluted out of the running total in the downward slope of the inverted 'V'.

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Figure 2: Running 12 month total trends surrounding the 1967 to 1968 large increase (relative to the minimum 12 month total deaths in April 1967). Data from Office for National Statistics (ONS).

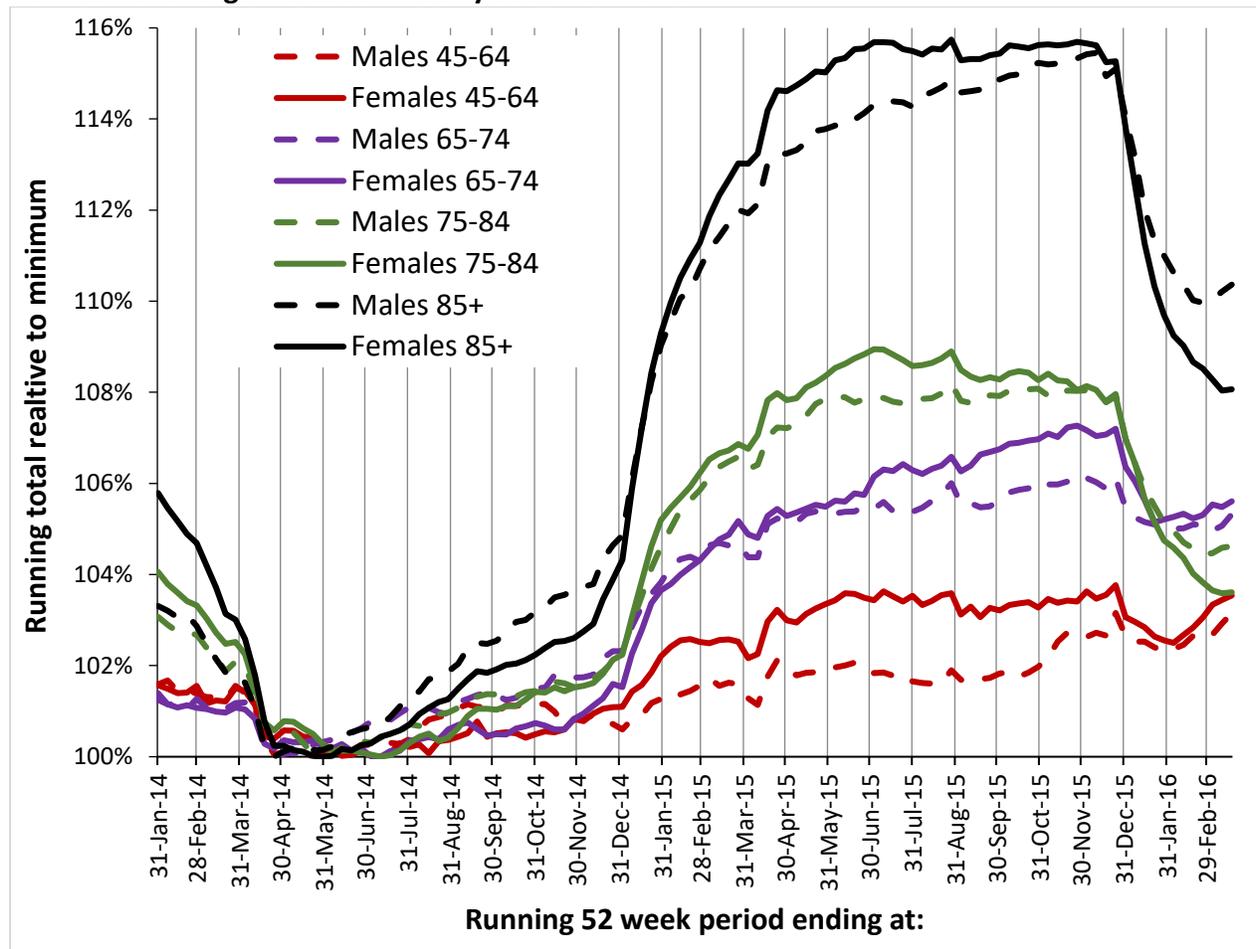


Footnote: Data is from the ONS. The trend has been linearized using a second order polynomial to remove the slight upward trend during the period 1960 to 1975.

Firstly, Figure 2 examines the events surrounding the large 1967 to 1968 event. A previous influenza outbreak (centred on Jan-66) leads to a table top feature in 1966. Deaths do however return to the baseline position during 1967. Males and females are equally affected. Another influenza event follows which is spread across Dec-67 to Feb-68) and leads to the large difference between 1968 and 1967. However, after the event deaths remain stubbornly high through to the next influenza event in Dec-69. There is the hint of two step-like events commencing Apr-67 (relatively small) and Mar-69 (larger). Females are however disproportionately affected in the assumed 67/68 influenza event.

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Figure 3: Running 52 week total deaths in England & Wales, by age band and gender, for the 2014 to 2015 large increase. Weekly data from ONS.



Next the more recent 2014 to 2015 large increase in Figure 3 showing increasing effect with age and a degree of female specificity (as above). A step-like increase in deaths clearly initiates in early Jun-14. As above, deaths remain stubbornly high after the influenza event has worked its way out of the running total.

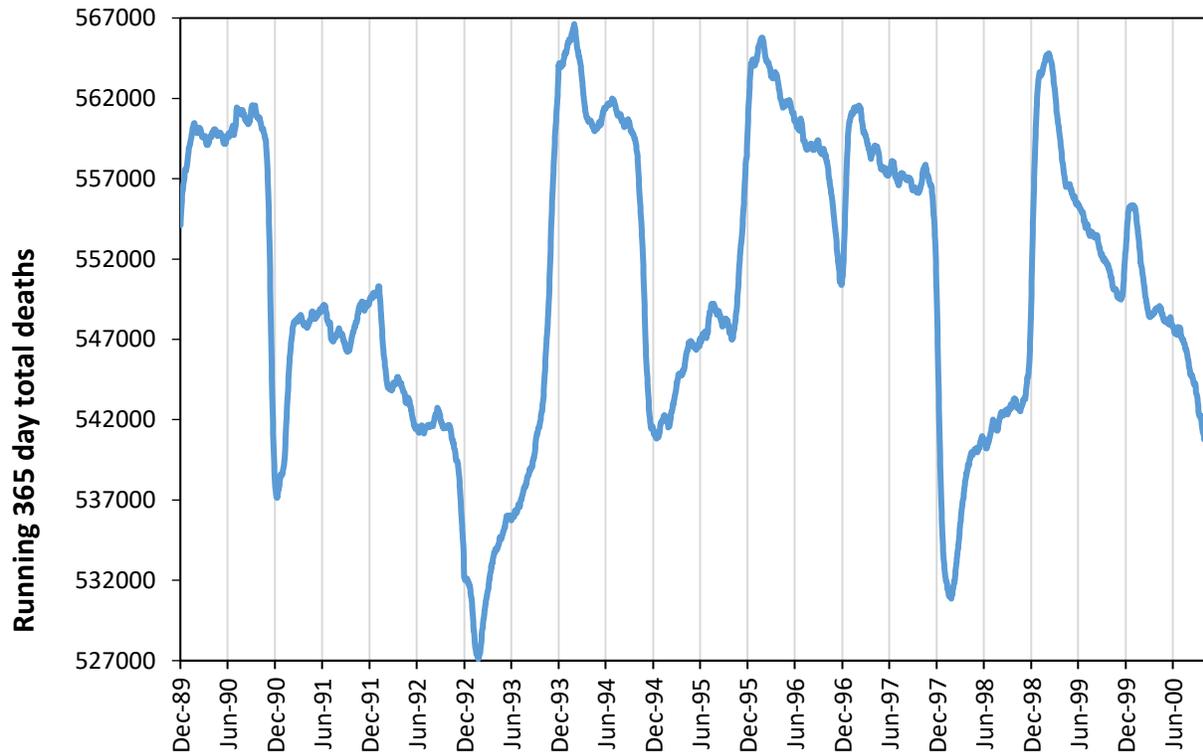
Finally, a daily time series from Jan-89 to Oct-00 is used to construct a running 365-day total in Figure 4. A classic influenza event in Dec-89/Jan-90 leads to a simple table top feature in the running total during 1990. Then follows three influenza events overlaid on three step-change events commencing after the 22nd February 1993, 12th January 1995 and 16th February 1998. In all three cases deaths remain high after the influenza spike has left the running total. A classic influenza event occurs at the start of 1997 but deaths return to baseline. Of particular interest is the parallel effect of the 1993, 1995 and 1998 outbreaks on medical admissions in England (reviewed in Jones 2015f).

The common themes in the three charts regarding how deaths change after different types of event are reasonably obvious.

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1. An influenza event in isolation generates a spike in deaths followed by a return to the baseline trend
2. An influenza event which is preceded by the presumed infectious outbreak generates a more semi-permanent response

Figure 4: Running 365 day total deaths in England and Wales (1989 to 2000)



Running 365 day period ending at:

Footnote: Daily deaths from Jan-89 to Nov-00 were kindly provided by the ONS. Data for the 29th of February has been removed from 1992, 1996 and 2000 to maintain a consistent 365 day total. Total deaths trend downward over this period, and so a second order polynomial trend has been used to linearize the data.

A common pathogen as an enabling force?

At this point the known roles of the common immune modulating herpes virus, cytomegalovirus (CMV), in manipulating influenza resistance and the effectiveness of influenza vaccination need to be highlighted. The ability of CMV to modulate immunity to other agents including influenza has been known for many years (reviewed in Jones 2016a). Indeed CMV is central in the expression of what is known as the 'infectious burden', where multiple pathogens act to exacerbate disease progress or symptom severity (reviewed in Jones 2016a). CMV has been proposed to be a likely cause of the step-like increases in death which appear to potentiate the effects of influenza, as observed above (Jones 2015d, 2016a,b).

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Increased susceptibility in neurological conditions

In their analysis of the large increase in deaths in 2015 the Office for National Statistics (2016) noted a large increase in deaths among those suffering from Alzheimer's and dementia. In England, those suffering from Alzheimer's and dementia are known to account for around 30% of excess winter mortality, range 12 to 43% depending on the year (Liddell et al 2016). Hence, as a group they are clearly susceptible to 'winter' environmental and infectious stress in general, hence the wide range seen between years. Part of this susceptibility lies in the observation that those with neurological disorders are also characterised by inflammatory processes, leading to a higher risk of becoming bed ridden and the consequent effects thereof (Jones and Goldeck 2014). Also that the presumed infectious agent behind the step-like increases in death appears to act by immune manipulation leading to increased susceptibility to infection and inflammation. The neurologic disorders group then become a prime target during these outbreaks and to the combined (possibly synergistic effects) of one of these outbreaks plus an influenza outbreak. This has been discussed elsewhere (Jones & Goldeck 2014, Jones 2015e, 2016b,i), but curiously receives no mention by the ONS (2016) or Public Health England (2016).

Given the central role of CMV in the expression of pathogen burden this virus will almost certainly be involved in one way or another, and indeed there is an increasing body of literature pointing to CMV's multiple direct and indirect roles in the debilitation associated with neurological conditions (Jones 2016i).

Conclusions

In conclusion, common themes have emerged in this analysis relating to an influenza epidemic occurring shortly after an outbreak of the agent leading to step-increases in death. Deaths appear to be potentiated, females are affected more than males, and deaths remain stubbornly high after the effect of influenza has waned – which only occurs during a dual plus influenza event.

All major studies on influenza vaccine effectiveness conducted to date have ignored the effects of CMV and/or this other agent (Osterholm et al 2012). We need to ask the question as to exactly how much antigenic drift *per se* contributes to the lower vaccine effectiveness observed in some years (Osterholm et al 2012), as opposed to the contribution from CMV and/or the other agent?

These combined events will lead to periods of higher than expected health care costs (Jones 2015f, 2016c), and the role of the absolute number of (local) deaths needs to be reflected in the NHS funding formula.

It also remains to be answered as to why UK government agencies stubbornly refuse to discuss any possibility that this is a recurring event, or that a second agent is involved, and why the NHS remains so poorly informed regarding the impact upon admissions, bed occupancy and costs.

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