FORUM ECONOMICS

The cost of a warming climate

A study finds that meeting climate-change mitigation targets will lead to a substantial reduction in economic damages. Here, economists present opposing views on the approach used by studies such as this one. SEE LETTER P.549

THE TOPIC IN BRIEF

- Climate change is already affecting the economy through hurricanes, droughts and floods.
- On page 549, Burke *et al.*[1] report that achieving global-warming targets set by the United Nations could save trillions of dollars in damages.
- The study's methodology follows previous literature[2,3] by examining the short-term effects of weather on the growth rate of gross domestic product (GDP) — the market value of all goods and services produced in a country in a specific time period.
- These data are then extrapolated into the future to assess the economic impacts of climate change.
- The validity of this approach has been intensely debated in the economics community.

Feeling the heat

WOLFRAM SCHLENKER

Estimates of the economic impacts of climate change are essential for the development of climate policies. Important concerns have been raised about studies such as that of Burke *et al.*, and more research needs to be carried out. However, I think that the authors of these studies are doing the best job possible by basing their estimates on a rigorous analysis and clearly stating their assumptions.

The extrapolation of the historical relationship between temperature and GDP into the future raises the question of whether technological advances might change the predicted trajectory. However, it is worth emphasizing that such extrapolation is based on an econometric model — an economic model based on an empirical analysis — that has been shown to be remarkably consistent between rich and poor countries, as well as between the earlier and later part of the sample period involved (see, for example, ref. 3). This makes it unlikely that adaptation measures are already available, because they have not been deployed in the past even though hot countries would have benefited from them.

GDP is a useful metric to assess the benefits of limiting global warming. It provides a measurement of human welfare under the assumption that the market prices of goods and services fully reflect the costs of their production and use[4]. In reality, this assumption is not always valid. For instance, fossil-fuel prices often do not include the costs associated with global warming and other environmental effects on society. However, by focusing only on GDP, huge economic impacts from limiting global warming are predicted. These estimates would be even bigger if the non-market benefits of reduced fossil-fuel use — for example, for human health and ecosystems — were considered.

The predicted impacts are larger than those obtained in earlier work[5]. The main reason is that strong effects of weather on the growth rate of GDP are found, whereas the earlier work stipulated, but did not empirically test, that weather affects only the level of GDP in a particular year. Heat and drought, for example, directly influence agricultural yields in a given year, but have limited impact in the following years. By contrast, a growth effect implies that a destructive weather event not only decreases GDP in a given year, but also lowers the value for all future years.

The main innovation of studies such as that of Burke and colleagues is to use an econometric model that can incorporate both level and growth effects, without favouring one type of effect over the other[2]. This is accomplished by including both the current temperature and the temperatures in previous periods in the analysis.

If weather affected GDP only in a given year, immediate impacts would be offset in the future — for example, a 1% decrease in GDP would be offset by a 1% increase in GDP the following year. On the contrary, the authors of these studies find that the impacts are not offset, but rather amplified. The one caveat is that when temperatures in previous periods are included in the analysis, the uncertainties in the projected economic damages increase substantially. Resolving this issue is a key direction for future research. ■

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Grand damage projections

MAXIMILIAN AUFFHAMMER

Translating the impacts of climate change on surface temperature, precipitation and sea level into economic damages is challenging. One approach is to use longitudinal data (repeated measurements of the same variables) to estimate damage functions — mathematical expressions that translate physical impacts into monetary damages[6]. These functions are associated with specific locations and sectors, such as agriculture or manufacturing. A major drawback is that coverage across locations and sectors is incomplete. Studies such as that of Burke *et al.* circumvent this problem by using GDP to aggregate economic impacts across sectors. Nevertheless, several issues must be considered before strong conclusions can be drawn from this work.

First, the authors of these studies argue that societal adaptation to climate change is accounted for statistically. However, what is incorporated stems from a historical cross-country comparison of the temperature sensitivity of GDP. In reality, future adaptation will probably involve innovative technologies with lower costs than those that are currently used. Such technologies might include, for example, air conditioners powered by carbon-free electricity that are more energy efficient than present-day devices. Adaptation could therefore result in lower economic damages than are predicted.

Second, on a larger scale, climate change will lead to a planetary redistribution of economic activity, which will result in a redistribution of international trade flows. Such an effect is impossible to quantify credibly and could have a large impact on the projected damages.

Third, GDP includes only goods and services that are transacted in markets and therefore have measurable prices. It does not capture the effects of climate change on valuable non-market sectors, such as ecosystem services and biodiversity.

Finally, allowing climate change to influence both the level and the growth rate of GDP is shown to lead to a wide distribution of projected impacts. As a result, neither small (or zero) effects nor massive effects can be ruled out. Attempts to distinguish between these two

possibilities using simple statistical models at the global level have been inconclusive.

There are good reasons for thinking that some effects of climate change might be cumulative. For instance, climate and weather will affect the level, and potentially the growth rate and efficiency, of capital and labour. Furthermore, climate might induce technological change through both adaptation and mitigation measures. Pinning down these macroeconomic processes to resolve just how large the effects of climate will be on the long-term growth of GDP needs to be a high priority for future work. ■

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NEURODEGENERATION

Sabotage by the brain's supporting cells

Several neurodegenerative disorders are linked to the build-up of abnormal α-synuclein protein in distinct cell types. It emerges that differing intracellular factors dictate the properties of this protein in each cell type. SEE LETTER P.558

LARY C. WALKER

One of the many mysteries surrounding neurodegenerative diseases is how they can manifest in such a variety of ways. Disorders such as Alzheimer's disease, Parkinson's disease and amyotrophic lateral sclerosis (also known as motor neuron disease) are each defined by a core set of nervous-system abnormalities, but every affected person's brain responds slightly differently. Moreover, although each of these disorders is associated with the abnormal accumulation of a different protein in or around cells, some protein aggregates can give rise to more than one neurodegenerative disease. How can this happen? On page 558, Peng *et al.*[1] present persuasive evidence that different types of cell accumulate structurally distinct forms of one protein, α-synuclein. By shaping the 3D architecture of the corrupted protein, the cell type helps to determine the nature of the resulting disease.

Most normal proteins fold into characteristic conformations that are strongly governed by the protein's amino-acid sequence. But in age-related neurodegenerative conditions, certain proteins misfold, and induce other proteins of the same type also to misfold and to stick to one another. In this way, the abnormal molecular structure propagates by means of a crystallization-like process called seeded protein aggregation[2].

One such protein is α-synuclein. Under normal circumstances, α-synuclein is located mainly in nerve terminals. But in some cases, the protein forms intraneuronal aggregates called Lewy bodies and Lewy neurites — for instance in Parkinson's disease and a condition known as dementia with Lewy bodies, which are collectively referred to as Lewy body diseases (LBDs). In a more-aggressive brain disorder called multiple system atrophy (MSA), misfolded α-synuclein accumulates mostly in neuron-supporting cells called oligodendrocytes[3], in clumps known as glial cytoplasmic inclusions.

Why α-synuclein aggregates are mainly found in different cell types in MSA and LBDs has been uncertain. It cannot be attributed to differences in amino-acid sequence, because α-synuclein is not typically mutated in the common form of either condition[4,5]. However, previous work[6] has shown that aberrant α-synuclein in LBDs is structurally and functionally different from that in MSA. These variant molecular states are known as protein strains[7]. When injected into the brains of susceptible mice, the MSA strain causes a fatal disease similar to human MSA. By contrast, injecting the LBD strain fails to induce major signs of disease in this model[8].

Peng *et al.* set out to investigate the causes behind this difference in α-synuclein potency. The authors first confirmed that protein aggregates in the oligodendrocytes of people with MSA are conformationally distinct from those in neurons from people who have LBDs. In MSA, a few neurons do harbour α-synuclein aggregates, but the researchers found that these aggregates display the LBD conformation — thus, the two strains can occupy the same brain, albeit in different cell types. Next, the team exposed cultured cells to each strain, and found that MSA-derived α-synuclein is approximately 1,000 times more potent at inducing aggregation than is the LBD-derived protein.

The authors then injected the two types of aggregated α-synuclein (called seeds) into the brains of wild-type mice. This *in vivo* experiment confirmed that MSA-derived seeds are much more effective than seeds derived from LBDs at seeding aggregation. However, the seeds instigated aggregation only in neurons, not in oligodendrocytes.

Why might this be the case? Oligodendrocytes normally produce little, if any, α-synuclein[9]. The authors therefore genetically engineered mice to express α-synuclein only in oligodendrocytes. They found that α-synuclein aggregation could be induced in oligodendrocytes in these mice using seeds from either the MSA or the LBD strain — but again, the MSA strain was much the more potent. Importantly, the aggregates that emerged were always the MSA strain, regardless of the type of seed injected. Finally, when Peng *et al.* exposed synthetic, unaggregated

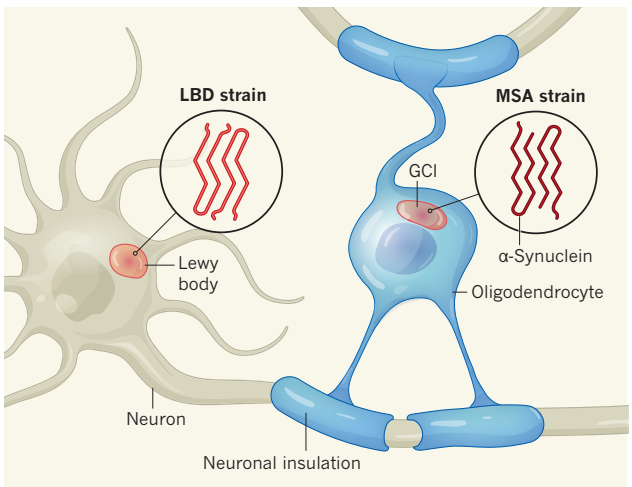

Figure 1 | Distinct strains of α-synuclein protein. In Parkinson's disease and dementia with Lewy bodies (collectively referred to as Lewy body diseases; LBDs), a misfolded form of α-synuclein called the LBD strain aggregates mainly in neurons to form anomalous structures called Lewy bodies and Lewy neurites (not shown). But in a disease known as multiple system atrophy (MSA), a different strain of misfolded α-synuclein forms aggregates called glial cytoplasmic inclusions (GCIs) in oligodendrocytes — non-neuronal cells that normally produce the fatty insulation for neuronal projections. Peng *et al.*[1] show that differences in the intracellular environments of the two cell types are responsible for the formation of the two strains.