NEWS & VIEWS

CLIMATE CHANGE

Too much of a bad thing

Gavin Schmidt and David Archer

There are various — and confusing — targets to limit global warming due to emissions of greenhouse gases. Estimates based on the total slug of carbon emitted are possibly the most robust, and are worrisome.

It is one thing to agree on a goal of international policy, quite another to achieve it. The 192 signatories of the 1992 United Nations Framework Convention on Climate Change (including the United States) have committed themselves to reducing the emissions of carbon dioxide and other greenhouse gases to avoid "dangerous interference in the climate system". But policy-makers around the world are still trying to figure out how, specifically, to do that.

The European Union has adopted a goal of keeping temperatures below 2 °C above pre-industrial levels. Others argue for a stabilization of atmospheric CO_2 concentrations at 350 parts per million (p.p.m.), or 450 p.p.m., or higher. In the United States, the administration of President Barack Obama has proposed reducing emissions by 80% by the year 2050. These schemes are all intended to be solutions to the same problem. But relating and comparing one to another is not straightforward. For instance, solutions to what level of atmospheric CO_2 is required to avoid a 2 °C temperature rise, and what emission pathways might achieve that goal, are still unclear. Papers elsewhere in this issue by Meinshausen *et al.*[1] and Allen *et al.*[2] explore the uncertain relationships between carbon emissions and climate response, with the aim of better estimating how much additional CO_2 might indeed be too much.

Meinshausen and colleagues[1] (page 1158) take a comprehensive probabilistic approach, combining the uncertainties in climate sensitivity and carbon-cycle feedbacks, and integrating the two over a large range of potential emission pathways. Their target is to avoid a peak global mean warming from the pre-industrial level of more than 2 °C (equivalent to a further rise of about 1.2 °C from today). We must note here that there is nothing special about 2 °C that would make warming of less than this magnitude 'safe'. It is more analogous to a speed limit on a road, and is a guide to the scale of the problem. With 2 °C of global warming (more over land and at the high latitudes), Earth would probably be warmer than it had been in millions of years — a huge change.

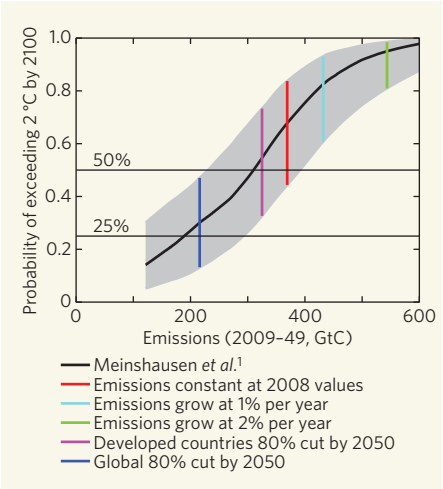

Figure 1 | The 2 °C lottery. The black line shows the probability of the peak global mean temperature exceeding 2 °C above pre-industrial levels before the year 2100 as a function of the integrated emissions from 2009 to 2049. The graph is adapted from the base case of Meinshausen *et al.*[1], including uncertainty ranges. Also shown are the cumulative emissions under various scenarios. Red, emissions constant at 2008 values until 2050. Light blue, growth in emissions continues at 1% per year until 2050 and then falls rapidly. Green, growth in emissions continues at 2% per year until 2050 and then falls rapidly. Purple, an 80% cut in emissions by 2050 (linearly applied, starting in 2010) from developed countries only, while developing-country emissions continue to grow at 1% per year. Dark blue, an 80% cut in emissions by 2050 from all countries.

Meinshausen *et al.* find that the maximum temperature that Earth will experience to the year 2100 depends most reliably on the total amount of CO_2 emitted to the year 2050, rather than on the final stabilized CO_2 concentration. Their base-case estimate is that the total emissions from today (2009) to 2050 need to stay below 190 GtC (equivalent to 700 $GtCO_2$; 1 GtC = 10^{12} kg of carbon) for us to have a good chance (75%) of staying below 2 °C (Fig. 1). The probability drops below 50% if we emit more than 310 GtC in that time. This is significantly less than the amount of carbon contained in proven reserves of gas, oil and coal, let alone reserves of non-traditional fossil-fuel sources such as tar shales, oil sands or methane hydrates. Last year, we probably emitted more than 9 GtC, and this has been increasing at around 1–3% a year. At that rate, we will reach 190 GtC in under 20 years.

Allen and colleagues[2] (page 1163) take a slightly different tack, using a combined climate and carbon-cycle model, and varying uncertain parameters, to produce a series of simulations that attempt to span the range of projections that are consistent with already observed changes. They agree with Meinshausen *et al.* that it's the total slug of carbon that matters most, and define a term they call the cumulative warming commitment (CWC) as the peak temperature change expected as a function of the total anthropogenic carbon.

Comparing the bottom-line results from the two studies is tricky because of the use of different units, different base periods and different experimental design[3]. However, given that humans have already emitted roughly 520 GtC to the end of 2008, Allen and colleagues' best-estimate CWC — 2 °C per 1,000 GtC emitted from 1750 to 2500 (compared with 2000–2050 in Meinshausen *et al.*) — implies that another 480 GtC would put us over 2 °C with more than 50% likelihood. This is broadly consistent with the 310-GtC estimate from Meinshausen *et al.* over a much shorter time frame. For comparison, two scenarios with cuts of 80% in emissions by 2050, in developed countries and globally, give an additional 325 GtC and 216 GtC, respectively (Fig. 1).

A lot rides on the questions addressed in these papers, and they are unlikely to be the last words written on the topic. There is certainly room for further debate on the definition of 'dangerous'; the maximum global temperature is a good place to start, but ice sheets and sea level, for example, probably depend on the integrated climate impact rather than on peak warming[4–6]. Also, these studies[1,2] use the traditional, short-term 'Charney' climate sensitivity, which includes some fast feedbacks such as variations in atmospheric water vapour and clouds, but not the slower feedbacks such as changes in vegetation or ice sheets, or feedbacks in atmospheric aerosols. The true

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sensitivity of the Earth system may well be higher[7], implying that any temperature-based target will become progressively harder to maintain as slower feedbacks kick in.

Finally, both studies[1,2] make different assumptions about how non-CO_2 factors (anthropogenic methane, ozone, black carbon, sulphates and so on) will change. These effects can't be shoehorned into the same cumulative-emissions metric as CO_2 because their effects over time are much more closely tied to contemporaneous emission levels. However, they remain a tempting additional policy target that might usefully limit near-term temperature rises[8].

The bottom line? Dangerous change, even loosely defined, is going to be hard to avoid. Unless emissions begin to decline very soon, severe disruption to the climate system will entail expensive adaptation measures and may eventually require cleaning up the mess by actively removing CO_2 from the atmosphere. Like an oil spill or groundwater contamination, it will probably be cheaper in the long run to avoid making the mess in the first place. ■

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See also Editorial, page 1077, and online at www.nature.com/climatecrunch

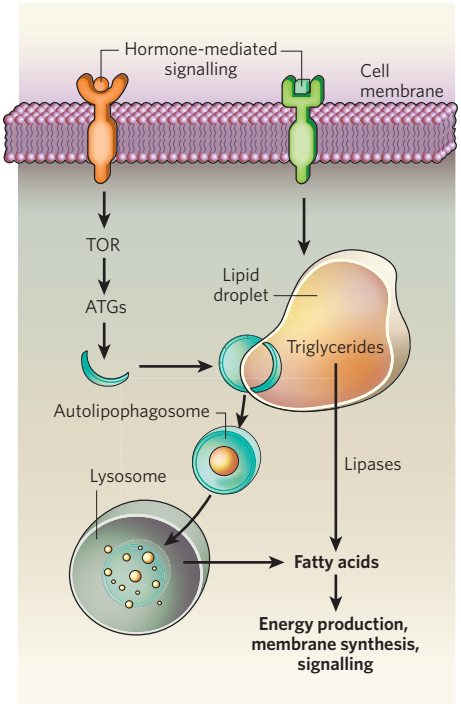

Figure 1 | Lipolysis and autophagy. Activation of intracellular signalling pathways mediated by the hormones glucagon and catecholamines can trigger the hydrolysis of triglycerides to fatty acids in lipid droplets by the process of lipolysis, which is mediated by lipase enzymes. Singh and co-authors[2] demonstrate that, during fasting, one such pathway in the liver involves TOR kinase and ATG proteins, and results in fatty-acid generation by an alternative mechanism. In a process they call macrolipophagy, portions of lipid droplets, or even whole droplets, become trapped inside the double-membrane-bound autolipophagosome vesicles and are transported to lysosomes, where they are degraded to fatty acids. Both lipolysis and macrolipophagy are inhibited by the hormone insulin.

CELL BIOLOGY

Another way to get rid of fat

Rudolf Zechner and Frank Madeo

When starved, cells resort to breaking down their assets — proteins, lipids and even whole organelles. An investigation of lipid metabolism indicates that one process — autophagy — targets all three cellular components.

Fatty acids are essential to all organisms — as substrates for energy production, as precursors of membrane lipids and as signalling molecules that control various cellular processes, including gene expression. They are stored as triglycerides in highly dynamic organelles called lipid droplets[1] and, when necessary, are re-released by the process of lipolysis. Established models of lipid storage and breakdown have undergone substantial revision in recent years. The latest adjustment is offered by Singh *et al.*[2] (page 1131 of this issue): their spectacular findings suggest that autophagy, the pathway by which excess or damaged organelles and proteins are degraded[3], also mediates fat mobilization and breakdown in liver cells (hepatocytes).

A rapid flux of fatty acids into and out of lipid droplets occurs through the deposition and degradation of triglycerides in adipose tissue and in other tissues (liver, heart, muscle and testis) and cells (macrophages) that require abundant fatty acids. Hydrolytic enzymes called lipases mediate lipid breakdown (catabolism), and for 40 years it was believed that hormone-sensitive lipase (HSL) acted alone in the catabolism of triglycerides. But the discovery of other essential lipases, such as ATGL, and regulatory proteins, including perilipin and CGI-58, indicated that the lipolytic pathway is much more complex than that[4].

In fact, key aspects of lipid turnover remain unclear. One mystery is the rapid turnover of triglycerides and cholesteryl esters (another component of lipid droplets) in hepatocytes, despite the cells' low concentrations of HSL and ATGL. By presenting compelling evidence for an autophagic mechanism mediating fasting-induced lipolysis in both mouse liver and culture-grown hepatocytes, Singh *et al.* resolve some of these issues.

In autophagy, cytoplasmic components and cellular organelles destined for degradation become trapped in double-membrane-bound vesicles called autophagosomes, and are then broken down in lysosomes with which the autophagosomes fuse[3]. This sequestration and lysosomal breakdown of autophagosomal contents is generally referred to as macroautophagy.

In a functional analogy to macroautophagy, Singh *et al.*[2] show that, under fasting conditions, the cytoplasmic protein LC3 and several other autophagy-related proteins are recruited to lipid droplets, where they form a double membrane that encloses droplet parts. These lipid-containing vesicles, termed autolipophagosomes, subsequently fuse with lysosomes, and their contents are degraded (Fig. 1).

The authors also show that the efficiency of this process of 'macrolipophagy' varies with the nutritional status of the mice. Feeding the animals a high-fat diet for an extended period (16 weeks) impairs autophagy-mediated breakdown of lipid stores in the liver, inducing a vicious circle in which — as the authors propose — increased fat ingestion may be associated with decreased fat removal and excessive lipid deposition in the liver. A similar response in obese people would explain how they might develop fatty-liver disease. Additionally, autophagy diminishes with age, possibly explaining the age-related deposition of fat in tissues where it does not belong. Singh and colleagues' results therefore provoke speculation that inducing autophagy through drug-mediated inhibition of TOR kinase, its master regulator protein, might ameliorate diet- or age-induced fatty-liver disease in humans. Intriguingly, resveratrol — a potent inducer of autophagy found naturally in red wine[5] — prolongs the lifespan of mice fed a high-calorie diet[6].

It remains to be seen how extensively macrolipophagy contributes to lipid homeostasis under physiological conditions, and whether it

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