CARBON CYCLE

Microbes weaken soil carbon sink

The rate at which carbon dioxide is lost from soil has risen faster than the rate at which it is used by land plants, because soil microbes have become more active — possibly weakening the land surface's ability to act as a carbon sink. SEE LETTER P.80

KIONA OGLE

The terrestrial land surface has a crucial role in the global carbon cycle, providing feedbacks to changes in atmospheric levels of carbon dioxide and associated climate change[1]. Increases in atmospheric CO_2 concentrations and in soil and air temperatures worldwide over the past several decades have been paralleled by an increase in the metabolism of organisms at the land surface — as demonstrated by enhanced rates of CO_2 uptake, mainly by plants through photosynthesis, and of CO_2 loss from plants and soil microorganisms, mostly owing to respiratory processes[2–6]. On page 80, Bond-Lamberty *et al.*[7] report that the rate of increase of CO_2 loss is outpacing that of CO_2 uptake by plants. The authors attribute the imbalance in these rates of increase to enhanced activity of microbes that obtain nutrition by decomposing or mineralizing organic matter in soil. If the observed trend continues, then respiration by microbes could contribute substantially to global warming by releasing CO_2 from organic matter that has previously been stored in soil for decades to millennia.

A variety of processes underlie the exchange of CO_2 between the land surface and the atmosphere. Bond-Lamberty *et al.* focused on soil respiration, which is arguably one of the largest fluxes of CO_2. The authors analysed previously published soil-respiration data[8] from many sites around the world that covered a broad range of ecosystems, including cropland, temperate forest and desert. They used these data to estimate the annual rates of soil respiration at various sites and to evaluate trends between 1990 and 2014.

Bond-Lamberty and colleagues then compared trends in soil respiration (CO_2 loss) with those of plant productivity (CO_2 uptake) that were derived from different data sources, including satellites. They found that the ratio of the rate of soil respiration to that of plant productivity has, in general, increased over the period covered by their data set. The ratio rarely exceeded 1, except at certain sites in particular years, which indicates that specific situations can lead to more CO_2 being lost from soil than is taken up by plants.

The findings beg the question of whether the average global ratio could become greater than 1 in the future, and, if so, when? Such an event would mark the tipping point at which the land surface stops operating mainly as a sink that helps to remove atmospheric CO_2 that is derived from fossil-fuel emissions[9,10], and starts acting as a source of CO_2 — exacerbating rising CO_2 levels and accelerating the pace of climate change[11] (Fig. 1).

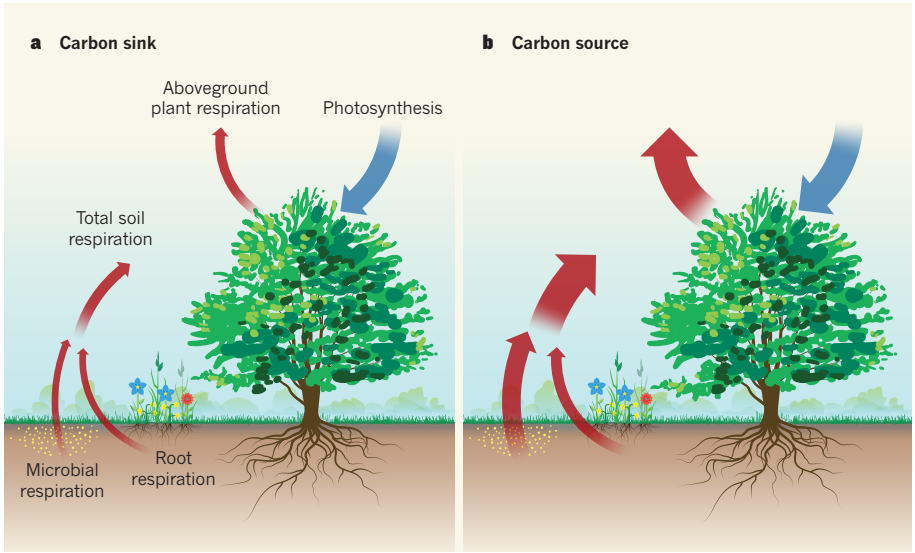

Figure 1 | Tipping the balance of carbon fluxes at Earth's land surface. **a**, The main sources of carbon dioxide from the terrestrial biosphere are respiration that is associated with the decomposition of organic matter in soil by microbes, and respiration of plants (both aboveground and belowground). Plants also absorb CO_2 through photosynthesis. At present, the total amount of CO_2 that is absorbed by plants exceeds the amount that is produced by respiration, and so the land surface acts as a carbon sink. Arrow widths roughly correspond to the sizes of the CO_2 fluxes. **b**, Bond-Lamberty *et al.*[7] report that, over the past few decades, the rate at which CO_2 is produced by soil microbes has increased faster than that at which CO_2 is used by plants. This raises the possibility that the rate of respiration will reach a tipping point at which it overtakes the rate of CO_2 uptake by plants. Under such a scenario, the land surface would act as a source of atmospheric CO_2. The time at which such a tipping point would be reached is unclear.

The authors next focused on studies in their data set that broke down total soil respiration into respiration dominated by decomposition by microbes and that associated with plant roots. Analysis of the microbial-dominated respiration rates led them to conclude that the disproportionately faster increase in the rate of total soil respiration is due to the enhanced activity of soil microbes. However, to understand whether accelerated rates of soil respiration will cause the land surface to become a source of CO_2, the temporal trends in respiratory losses associated with aboveground plant biomass must also be considered — the total loss of biologically derived CO_2 from the terrestrial biosphere is the sum of the soil and non-soil losses.

As Bond-Lamberty *et al.* acknowledge, previously published long-term data[12,13] recorded by eddy-covariance towers, which continuously monitor CO_2 concentrations and fluxes at specific sites across a range of ecosystems, suggest that the rate of increase of plant productivity has been faster than that of the total aboveground and belowground respiratory CO_2 losses. Further data and analyses are required to explain why those findings apparently contradict the authors' results.

If Bond-Lamberty and colleagues' findings are correct, which mechanisms could explain the markedly enhanced stimulation of the activity of soil microbes relative to plant productivity and plant respiration? Studies in the past few years have shown that the ability of plants to downregulate respiration in response to long-term increases in temperature[14] is

much greater than that of short-lived soil microbes[4,15–17]. The authors suggest that the increased microbial activity observed in their study probably reflects the stimulatory effects of elevated temperatures associated with climate change.

There are, however, potential issues when drawing global inferences from the data analysed by Bond-Lamberty and co-workers. Most of the data came from spot measurements of soil-respiration rates that were obtained by many different researchers, who used a variety of methods to work out the contributions of soil microbes. This diversity of methods might have led those researchers to come to contrasting conclusions about the relative importance of soil microbes in their studies. Moreover, Bond-Lamberty *et al.* used simplifying assumptions to translate hourly or daily snapshots of respiration rates into annual fluxes of CO_2, but did not take into account the uncertainty in these calculations. The soil-respiration data set is also limited in its temporal coverage of individual sites: repeated observations were available for only a handful of sites, yet recurrent observations are necessary to prevent temporal trends from being obscured by factors that vary between sites.

The authors acknowledge and account for some of these limitations in their statistical analyses, but clearly there is room for a more rigorous investigation. This would require researchers to gather continuous time series of soil respiration and its component fluxes, and demands the use of precise methods for quantifying uncertainty and for extrapolating local measurements to determine trends in larger regions. Despite the limitations, Bond-Lamberty and colleagues' work is valuable because it aids our understanding of soil's long-term potential for sequestering carbon — as well as how this sequestration might be threatened by accelerated rates of organic-matter decomposition by soil microbes. Their findings will be crucial for developing and testing models of the global carbon budget, of which soil carbon is a central component.

Fluxes of CO_2 across whole ecosystems are often measured using eddy-covariance towers. By contrast, continuous measurements of soil respiration and decomposition by microbes are not broadly available for sites worldwide or do not cover multi-year periods. The establishment of long-term observational projects such as the US National Ecological Observatory Network (NEON), which monitors fluxes of soil CO_2 among other ecological measures, will create opportunities for the systematic evaluation of temporal trends and the underlying causes of changes in the rates at which CO_2 is lost from soil. Such data will be paramount for developing regional and global models of the carbon cycle, as well as for assessing climate change and the strategies by which it might be mitigated[18]. ■

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AUTOIMMUNITY

Peptide secretion triggers diabetes

An autoimmune attack on cells that make the hormone insulin causes type 1 diabetes. A mouse study reveals that pancreatic-cell release of insulin peptide fragments into the bloodstream triggers this harmful process. SEE LETTER P.107

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Diabetes arises from problems in the regulation of blood glucose, which is controlled by releasing the hormone insulin. The amount of insulin made is abnormally low in type 1 diabetes owing to the autoimmune-mediated destruction of insulin-producing β-cells in the pancreas. Wan *et al.*[1] reveal on page 107 how this immune attack is triggered.

Type 1 diabetes was a lethal condition with a life expectancy of just months until the discovery of insulin in 1921 enabled clinical management by insulin injection[2]. Although this therapy greatly extends life expectancy, a deeper understanding of the disease is needed to develop treatments that delay or prevent disease onset.

Studies of non-obese diabetic (NOD) mice, which spontaneously develop the disease, have provided insights into the mechanisms that cause the condition. Such work has revealed that T cells of the immune system have a key role in destroying insulin-producing pancreatic β-cells. T-cell immunosurveillance is aided by antigen-presenting cells, which present peptide fragments called antigens on their surface bound to major histocompatibility complex (MHC) class I and II molecules. T cells typically encounter antigen-presenting cells in the lymph nodes, and their T-cell receptor samples the antigens presented on MHC molecules. T cells that respond to self-antigens are normally eliminated as they mature in the thymus gland, but imperfections in this process can lead to autoimmunity. Although many proteins present in pancreatic β-cells could potentially provide the autoimmune trigger for type 1 diabetes, insulin is the culprit in NOD mice[2].

In type 1 diabetes in humans and NOD mice, the presentation of a peptide consisting of amino acids 12 to 20 of insulin's B chain (B:12–20) by MHC class II molecules can activate CD4 T cells that recognize this peptide[3]. In NOD mice, a class II MHC molecule called I-A^{g7} presents B:12–20 to CD4 T cells and activates them. These cells then initiate a process that activates CD8 T cells, which are specific for other β-cell peptides[4]. Activated CD8 cells kill β-cells, leading to diabetes when the T cells eliminate enough β-cells. The risk of a person developing type 1 diabetes is often linked to MHC class II genes, which are among the mostly highly variable human genes. The gene encoding the version of MHC class II called HLA-DQ8 is tightly associated with the disease[5]. Remarkably, HLA-DQ8 has a highly similar peptide-binding specificity to that of I-A^{g7} (ref. 6).

"Why isn't there selection against the generation of secreted insulin peptides that trigger autoimmunity in humans?"

Wan and colleagues confirmed the pathological potential of T cells that are specific for I-A^{g7}–B:12–20 complexes by introducing such T cells into NOD mice under conditions that generate enough CD4 T cells to cause type 1 diabetes. As a control, the authors did a similar transfer into NOD mice that have a