Microbial change in warming soils

Long-term reorganization of microbial communities leads to pulses in carbon release

By **Daniel B. Metcalfe**

Soils are the largest single terrestrial source of carbon dioxide (CO_2), but these emissions are highly sensitive to a range of factors associated with climate change and human land use (*1*). Researchers have long sought to better understand the underlying drivers of soil CO_2 emissions, but the duration of experiments is all too often constrained by project deadlines and personnel contracts, hampering our ability to understand and predict the many gradual but important processes that occur in soils (*2*). On page 101 of this issue, Melillo *et al.* (*3*) report on an intriguing 26-year record of soil respiration responses to warming in a temperate forest. The results from this unusually long time series highlight both the potential pitfalls of drawing hasty conclusions from short-term studies and the importance of long-term experiments in ecosystem and climate science.

Shifts in the terrestrial carbon store, of which soils constitute around 70%, are one of the most important but least understood drivers of variation in atmospheric CO_2 levels (*4*, *5*). Soil carbon is sensitive to a wide range of factors, of which temperature is one of the most important from a climate change perspective (*6*). Generally, warm conditions promote microbial activity. Global warming would therefore be expected to increase microbial breakdown of organic carbon and subsequent release as CO_2, but demonstrating this conclusively has proved difficult.

Several approaches have been used to examine patterns in, and regulators of, soil carbon cycling. Multiple controlled experiments have repeatedly shown increasing soil respiration under warmer conditions (*6*, *7*). A comprehensive global synthesis of soil respiration measurements found an increase associated with rising air temperatures since the 1960s, but the exact mechanisms linking temperature and soil carbon cycling remain unclear (*8*). Furthermore, it is not well known how much soil carbon is readily available for microbial breakdown versus how much is locked up in recalcitrant material and to what extent this could shift with climate change (*6*, *9*).

Melillo *et al.* make important advances in addressing these gaps with one of the most detailed pictures yet of the responses of soil microbes and carbon cycling over more than two decades of experimental warming. Their results will be invaluable in ongoing efforts to better predict and mitigate carbon losses from soils that face rapid warming associated with climate change.

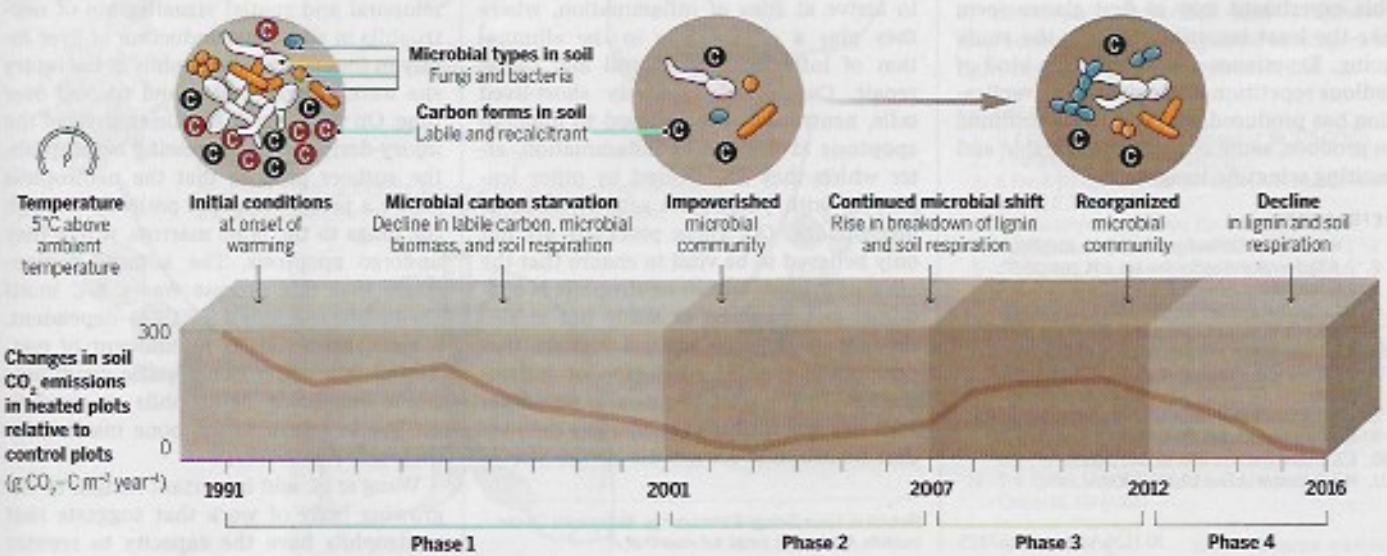
The authors compare soil respiration in forest plots heated with buried cables with soil respiration in untouched plots. The insights derived from these measurements are amplified by repeating these measurements for so long. The authors also draw from a wide range of shorter-term published experiments from the same site to elucidate the mechanisms that control observed shifts in respiration between plots and over time. Over the 26-year period of the experiment, they observed three multiyear phases of soil respiration (see the figure). In the first phase, soil respiration steadily decreased under warmed conditions. This was followed by a second phase where warming induced little change in respiration, and then a third phase of steadily rising respiration. At the time of publication, the experiment is entering a fourth phase where soil respiration is again gradually declining.

The authors conclude that decades-long warming stimulates phases of minimal warming-induced respiration, corresponding to widespread soil microbial commu-

Department of Physical Geography and Ecosystem Science, Lund University, Sölvegatan 12, SE 223 62, Lund, Sweden, and Department of Ecology and Environmental Science, Umeå University, Linnaeus väg 6, SE 901 87, Umeå, Sweden. Email: dbmetcalfe@googlemail.com

Long-term reorganization of soil microbial communities under warming

In a 26-year warming experiment, carbon dioxide emissions from heated plots changed with time as microbial communities reorganized.



nity reorganization as microbes struggle to adjust to the environmental conditions (see the figure). These phases are followed by surges in breakdown of previously inaccessible soil carbon pools as the reorganized microbial community takes advantage of the warmer conditions. If these findings hold more widely across major terrestrial ecosystems, then a much greater portion of the global soil carbon store could potentially be vulnerable to decomposition and release as CO_2 under global warming than previously thought.

Melillo *et al.* make a persuasive argument for the importance of considering the complexity and dynamism of the underground world to understand climate change. Further work would benefit from widening this focus to encompass the complexity above ground and the intricate linkages between above- and belowground domains. Plants regulate the amount and type of carbon inputs to soil over a range of time scales and via various distinct pathways, and these inputs are sensitive to various environmental factors, including temperature (*1*, *10*). These plant-soil interactions represent a key frontier for development in the global vegetation models that are used to predict feedbacks between terrestrial ecosystems and climate change (*10*, *11*).

Myriad linkages between plant and soil processes are included in current global vegetation models, but many of these interactions have not been rigorously evaluated against data from field experiments (*11*). With complementary aboveground data, the warming experiment described by Melillo *et al.* could add further scientific value as a real-world test bed for the scientific ideas and hypotheses about plant-soil interactions coded in the model algorithms (*11*). However, perhaps the most critical future activity in the case of this experiment may at first glance seem like the least inspiring: to keep the study going. Experience shows that this kind of tedious repetition and painstaking replication has produced, and will likely continue to produce, some of our most valuable and exciting scientific insights. ■

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IMMUNOLOGY

Neutrophils take a round-

Imaging sheds light on neutrophil dynamics in sterile inflammation

By **Hannah Garner** *and* **Karin E. de Visser**

Inflammation is a body's response to harmful stimuli that aims to eliminate the trigger that caused the initial injury and enable tissue repair. Inflammation can be either pathogen-associated or sterile. Sterile inflammation can be triggered by acute conditions, such as trauma, toxin exposure, and ischemia-reperfusion injury, but it is also an important component of life-threatening chronic inflammatory conditions, such as atherosclerosis, cancer, and asbestosis (*1*). Regardless of etiology, the mechanisms resolving inflammatory responses constitute highly coordinated and active processes that are vital for restoring tissue homeostasis. Although the cellular and molecular signals that drive the initiation of sterile inflammation are well studied (*1*), we have a relatively poor understanding of the mechanisms through which sterile inflammation is resolved, thus limiting our ability to therapeutically tackle harmful inflammation. On page 111 of this issue, Wang *et al.* (*2*) shed light on a provocative aspect of resolution by key orchestrators of the inflammatory process: neutrophils.

> ***"...reverse migration of neutrophils is a physiological aspect of resolution of inflammation."***

Neutrophils are the first immune cells to arrive at sites of inflammation, where they play a critical role in the elimination of inflammatory stimuli and tissue repair. Considered relatively short-lived cells, neutrophils are believed to undergo apoptosis at the site of inflammation, after which they are cleared by other leukocytes with phagocytic activity, such as macrophages (*3*). These processes are not only believed to be vital to ensure that the noxious armory within neutrophils is contained and disposed of safely but is also thought to provide critical signals that promote successful resolution of inflammation. In the past 10 years, a paradigm shift has emerged that challenges the idea that neutrophils always die at the site of inflammation. Several group
onstrated, with human cells
zebrafish (*6*, *7*), and mouse
that some neutrophils have t
actively leave the site of infla
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Wang *et al.* bring new ins
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site were photoactivated and
time. On the basis of kinetic a
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the authors propose that the
follow a preprogrammed path
the lungs to the bone marrow
undergo apoptosis. The auth
strate that this process was
chemokine receptor 4 (CXCR4)
a mechanism that is reminis
trophil fate under homeostati
where senescent neutrophils
CXCR4 to return to the bone
their safe removal (*11*).

Wang *et al.* add important i
growing body of work that s
neutrophils have the capacit

Division of Tumor Biology & Immunology, Netherlands Cancer Institute, Amsterdam. Email: k.d.visser@nki.nl