

Glucose as substrate and signal in priming: Results from experiments with non-metabolizable glucose analogues

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Priming of soil organic matter remains the subject of intense research, but a mechanistic explanation of the phenomenon remains to be demonstrated. This is largely due to the multiple effects of easily available carbon on the soil microbial community, and the challenge of separating these influences from one another.

Several glucose analogues can be taken up by microbial glucose transporters and have similar regulatory effects on metabolism. These substances are, however, not easily catabolized by the common glycolytic pathway, limiting their energy value. Therefore, they can be used to distinguish between the action of glucose as a metabolic signal, and its influence as an energy source.

We incubated an agricultural Haplic Luvisol under controlled conditions for 24 days after addition of: 1) glucose, 2) 3-O-methyl-glucose, 3) α -methylglucoside or 4) 2-deoxyglucose, at three concentration levels, along with a control treatment of water addition. CO₂ efflux from soil was monitored by trapping evolved CO₂ in NaOH and back-titration with HCl.

On the first day after amendment, CO₂ efflux from soil increased strongly for glucose and much less for the analogues, relative to the control. Only glucose caused a peak in efflux within the first two days. Peak mineralization of 2-deoxyglucose and α -methylglucoside was delayed until the third day, while CO₂ from 3-O-methyl-glucose increased gradually, with a peak delayed by approximately a week. For glucose, the immediate increase in respiration was strongly dependent on the amount of glucose added, but this was not the case for the analogues, indicating that the catabolic potential for these substances was saturated. This is consistent with only a small part of the microbial community being capable of utilizing these carbon sources.

In a subsequent experiment, ¹⁴C-labelled glucose or ¹⁴C-labelled 3-O-methyl-glucose were added to the same soil, enabling quantification of the priming effect. For 3-O-methyl-glucose, priming was observed before the peak of amendment-derived CO₂ efflux, indicating that proposed short-term mechanisms involving apparent priming should not be mechanistically dependent on use of an external energy source. Instead, microorganisms respond to glucose through a regulated metabolic activation that can draw on their internal reserves.

Stronger priming occurred during the delayed CO₂ peak of 3-O-methyl-glucose. The correspondence of maximum catabolism and priming supports a role for carbon and energy supply in the mechanisms of 'real' priming, which take place over the longer term.

These results demonstrate the potential of glucose analogues for disentangling energy-driven from activation-driven mechanisms of priming.