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Influence of low 2-methyl-4-chloro-phenoxyacetic acid (MCPA) concentrations on degradation dynamics

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Pesticide degradation studies in soil are often performed at higher concentrations than can be measured in soils after conventional agriculture use. Therefore, biodegradation rates determined for higher concentrations cannot be extrapolated to lower concentrations, which may explain residual concentration of pesticides even a long time after application. The purpose of the present study was i) to compare the half-life time of MCPA throughout a mechanistic model based on the ^{14}C-respiration rate at low and high concentrations, and ii) to clarify if energy limitation and concentration thresholds control the expression of functional genes involved in pesticide degradation, iii) to assess if the absence of functional gene expression is a possible mechanisms that might explain the build-up of persistent pesticides and metabolite pools in soils.

To test our assumption, we set up an incubation experiment for 4 weeks. 50 g topsoil samples of a Luvisol from an agriculture field, were weighed into microcosms. Subsequently, the soil moisture was adjusted to a volumetric water content of 25 % with ^{14}C-ring labelled MCPA solutions ranging from low (30-500 μg kg^{-1} soil) to high concentrations (1000-20000 μg kg^{-1} soil). In the first degradation step, 2-methyl-4-chloro-phenoxyacetic acid (MCPA) is converted to 4-chloro-2-methylphenol. The responsible α-ketoglutarate-dependent dioxygenase is encoded by the *tfdA* gene. To quantify the abundance of putative MCPA degraders and their degradation potential, *tfdA* gene (DNA) and transcript (mRNA) abundances were estimated for each concentration through quantitative real-time PCR. Mineralization dynamics for each treatment were analyzed, following the release of ^{14}C-CO_2 during microbial respiration. In addition, we quantified the assimilation of MCPA-derived C by soil microorganism through ^{14}C in microbial biomass C at three distinct phases: lag, exponential increase and saturation phase, depending on cumulated ^{14}C mineralization. The remaining model compound in the soil were analyzed using a combination of liquid chromatography (LC) and mass spectrometry (MS/MS).

First results showed that *tfdA* gene expression depended on the added MCPA concentrations. With increasing MCPA concentration the cDNA abundance increased up to a level of $2{\cdot}10^4$ copies g^{-1} dry weight for the highest concentration. Low MCPA concentrations (<1000 μg kg^{-1} soil) did not induce any detectable expression of *tfdA* genes. We suggest that the mechanisms of MCPA degradation depended on the initial MCPA concentration: Whereas co-metabolisms might be the dominant pathway under low MCPA concentrations, metabolic mineralization occurred at high concentrations which were visible from cumulative ^{14}C-CO_2respiration curves in sigmoidal forms.