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A speculative claim of mass mortalities of honeybee colonies caused by fipronil in France is not supported by published field data

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Holder et al. (1) speculate that fipronil (a phenylpyrazole insecticide), rather than imidacloprid (a neonicotinoid), caused mass mortalities of honeybees (*Apis mellifera*) in France. The paper provides laboratory experimental data contributing to the knowledge of the toxicity of fipronil and its sulfone metabolite to honeybees. However, its claim that "fipronil is a credible cause of the mass mortalities of honey bees that were associated with agricultural sunflower in France during the 1990s" is not supported by published data, which are not cited in ref. 1. Seed treatment was the main use of fipronil in Europe, and thus this Letter focuses on this.

To estimate the impact on honeybee mortality rates in colonies in the field, Holder et al. (1) use a simplistic model combining laboratory toxicity results and exposure data. For exposure, they use "environmentally realistic residue concentrations of 5 ppb" for 2 neonicotinoids and fipronil. Nevertheless, for fipronil, the paper provides no references supporting 5 ppb as a realistic concentration and states that "there is a lack of data to prove the historical levels and prevalence of its residues in nectar and pollen." However, residues of fipronil and metabolites in pollen and nectar of sunflowers in Europe (France included) are publicly available: In sunflowers, no residues of fipronil or fipronil sulfone at the limit of quantification of 1 or 0.5 ppb were found in field-collected samples (2, 3).

The inaccurate assumption on fipronil residues appears to be caused by the failure to conduct and report a comprehensive literature review and to differentiate between the mobility of fipronil and neonicotinoids in plants. Holder et al. (1) cluster fipronil and neonicotinoids, treating them equally as "these systemic insecticides," overlooking information showing that the properties of fipronil are very different, resulting in a lower mobility of fipronil in plants (4). This is the main reason why fipronil and its main metabolites were rarely detected after seed treatments in Europe in beerelevant matrices, i.e., pollen and nectar/honey, confirming that the likelihood for exposure of honeybees is low, both in frequency and in amounts (2, 3, 5–7).

Holder et al. (1) report a honeybee field incident in Switzerland attributed to an accidental contamination with fipronil of a fungicidal sprayed formulation, unrelated to seed treatments. However, they neither present any evidence on honeybee mortalities caused by fipronil seed treatments nor include information from monitoring studies in Europe evaluating the impacts of fipronil use under field conditions (3, 5, 6).

The risk from a pesticide (fipronil in this case) is determined by the combination of toxicity and exposure. Holder et al. (1) provide their own toxicity data from laboratory tests, but the assumptions about field exposure are unsupported and not in line with publicly available data. Holder et al. (1) fail to cite publications directly relevant to fipronil seed treatment uses, particularly in sunflowers. Because of such shortcomings, the main claim and title of the paper are highly speculative and not adequately supported by reliable data.



¹ P. J. Holder, A. Jones, C. R. Tyler, J. E. Cresswell, Fipronil pesticide as a suspect in historical mass mortalities of honey bees. Proc. Natl. Acad. Sci. U.S.A. 115, 13033–13038 (2018).

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⁴ S. R. Mortensen, J. D. Holmsen, L. Weltje, Fipronil should not be categorized as a "systemic insecticide": A reply to Gibbons et al. (2015). Environ. Sci. Pollut. Res. Int. 22, 17253–17254 (2015).

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Conflict of interest statement: J.P. and C.S. are employed by BASF, a chemical company that produces and sells fipronil products.

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