**Supplemental info**



**Figure S1.** Dry mass of Palmer amaranth (*A. palmeri*) in response to different proportions of glufosinate (100% = 50 g ha-1) and saflufenacil (100% = 2.5 g ha-1) at 21 d after treatment. Means with the same letter do not differ by Tukey test (p<0.05).

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**Figure S2.** Visual injury (%), reactive oxygen species (ROS, hydrogen peroxide and superoxide), and protoporphyrin (ng g-1) levels provided by glufosinate and saflufenacil isolated or in tank mix on *Amaranthus palmeri* at 24 HAT.

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**Figure S3.** Visual control over time with (●) glufosinate (300 g ha-1), (■) glufosinate + saflufenacil (300 + 1 g ha-1), (▲) glufosinate + pyraflufen (300 + 0.17 g ha-1), (▼) glufosinate + lactofen (300 + 5 g ha-1), and (⯁) glufosinate + paraquat (300 + 11) on *Amaranthus tuberculatus* (Waterhemp), *A. palmeri* (Palmer amaranth), *Bassia scoparia* (kochia), *Lolium rigidum* (ryegrass), Sorghum halepense (Johnsongrass), and *Echinochloa colona* (barnyardgrass).



**Figure S4.** Inhibition of glutamine synthetase (GS) by glufosinate leads to proline and arginine accumulation, and increased levels of protoporphyrin in the presence of protoporphyrinogen oxidase (PPO) inhibitors. An unknown compound with the same mass as aminolevulinic acid (131.13) accumulates in Palmer amaranth (*A. palmeri*) at 24 h after treatment with glufosinate (560 g ha-1). This supports the hypothesis that glufosinate enhances PPO-inhibitors by a transient accumulation of glutamate, which is diverted into the chlorophyll pathway and leads to increased levels of protoporphyrin.