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# SYMBIOTIC BACTERIA MANIPULATION OF THE HOST INNATE IMMUNE SYSTEM IN THE SOCIAL AMOEBA *D. DISCOIDEUM*

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Innate immunity is a non-specific immune response that targets agents of harm such as bacteria and acts as the first line of defense for many eukaryotes. The innate immune mechanism has been studied in depth in many organisms, especially humans, and now in the social amoeba *Dictyostelium discoideum*. The innate immune cells in *D. discoideum*, called sentinel cells, phagocytize harmful material inside multicellular aggregates of *D. discoideum* during the social stage. These spent sentinel cells are left behind in trails as the multicellular slug migrates and can be visualized using fluorescent confocal microscopy.

Some *D. discoideum* clones, known as farmers, have a symbiotic relationship with *Burkholderia* spp. These clones carry food bacteria and other kinds of bacteria, including *Burkholderia*, through the social cycle. We have previously reported that farmers have fewer sentinel cells than non-farmers and thus their innate immunity could be impaired. We also reported that farmers exposed to a toxic environment had a higher number of total spore counts compared to non-farmers challenged with toxin. Both farmers and non-farmers showed same level of spore viability regardless of toxin treatment. This equal spore viability result supports the decreased fitness in toxin-challenged non-farmers as being real. However, further research needs to be conducted to test if the differences in sentinel cell number are due to the different phenotypes of the farmers and non-farmers or due to the presence of *Burkholderia*.

Here, we found that the presence of *Burkholderia* leads to a decrease in sentinel cell number in *D. discoideum*. We cured *Burkholderia* from farmer clones and noted an increase in sentinel cell number. Conversely, we also infected non-farmer clones with *Burkholderia* and observed a decrease in sentinel cell number. These data suggest that *Burkholderia* spp. may be manipulating the innate immunity of their host *D. discoideum* to reduce the possibility of clearance during the multicellular stage. Further study of this simple system could lead to insights in how bacteria are able to interact and manipulate the innate immunity of their eukaryotic hosts.