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M. ulcerans polyketide macrolide toxin, mycolactone, alters freshwater biofilm metabolic profiles: Implications for Buruli ulcer disease

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Buruli ulcer (BU) disease is a cutaneous tropical infection caused by *Mycobacterium ulcerans* (MU). BU typically presents as necrotic ulcers beyond the site of original bacterial colonization. This pathology is due to a unique photosensitive polyketide macrolide toxin, mycolactone, which is produced at great genetic and metabolic cost to MU. While BU incidence is correlated with anthropogenic change to aquatic habitats, the niche and mode(s) of transmission of MU remain unknown. I hypothesized that mycolactone is integral to the pathogen niche in freshwater biofilms, where MU has been found in previous studies. I tested a novel weapons role of mycolactone on aquatic biofilms using metabolic profiles to assess whether mycolactone alters community function, potentially increasing MU fitness. Experimental biofilms were grown on glass cover slips using local environmental freshwater. Following treatment with varying concentrations of mycolactone or appropriate controls, biofilms were incubated (in light or dark condition) and then transferred to Biolog EcoPlate assays for community metabolic analysis of 31 carbon substrates. The data indicate that mycolactone does not play a significant role in alteration of biofilm metabolic requirements in light environments but does significantly decrease the use of available D-cellobiose ($P<0.05$) and glycogen ($P<0.01$) in dark environments. This suggests that mycolactone may act to eliminate species that metabolize these substrates in low-light biofilms, which suggests that mycolactone may increase the fitness of the slow-growing mycobacterium in dark, freshwater biofilms. Low-light biofilms have previously been correlated with anthropogenic change to indigenous aquatic habitats due to increased runoff and water turbidity. These data also impact the understanding of transmission, as it seems that biofilms serve as a reservoir, but may still require a vector for effective transmission to humans. Finally, the history of BU provides a cautionary tale for other emerging diseases, as human intervention in the environment has favored the emergence of BU through the creation of new pathogen niches.

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