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N-acyl-L-homoserine lactones (AHLs) are co-regulatory ligands required for control of the expression of genes encoding virulence traits in many Gram-negative bacterial species. Recent studies have indicated that AHLs modulate the cellular concentrations of LuxR-type regulatory proteins by binding and fortifying these proteins against proteolytic degradation (Zhu & Winans, 2001). Halogenated furanones produced by the macroalga Delisea pulchra inhibit AHL-dependent gene expression. This study assayed for an in vivo interaction between a tritiated halogenated furanone and the LuxR protein of Vibrio fischeri overproduced in Escherichia coli. Whilst a stable interaction between the algal metabolite and the bacterial protein was not found, it was noted by Western analysis that the half-life of the protein is reduced up to 100-fold in the presence of halogenated furanones. This suggests that halogenated furanones modulate LuxR activity but act to destabilize, rather than protect, the AHL-dependent transcriptional activator. The furanone-dependent reduction in the cellular concentration of the LuxR protein was associated with a reduction in expression of a plasmid encoded P-luxl-gfp(ASV) fusion suggesting that the reduction in LuxR concentration is the mechanism by which furanones control expression of AHL-dependent phenotypes. The mode of action by which halogenated furanones reduce cellular concentrations of the LuxR protein remains to be characterized.

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