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Substrate analogue targeting glutamate racemase (MurI) alters cellular morphogenesis and inhibits biofilm formation in Streptococcus mutans

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D-Glutamate (D-Glu) is an essential biosynthetic building block of the peptidoglycans that encapsulate the bacterial cell wall. Glutamate racemase (MurI) catalyses the reversible formation of D-glutamate from L-glutamate and, hence, the enzyme is a potential therapeutic target. The current study was designed to identify novel molecules that target glutamate racemase, thereby mitigating *S. mutans* cariogenic capacities, inhibiting biofilm formation and having the potential to prevent dental caries. High throughput screening of approximately 250 commercially available compounds against the recombinant *S. mutans* glutamate racemase resulted in the identification of a substrate-product analogue, D-glutamine, as a modest competitive inhibitor of glutamate racemase. *In vitro* assays, the addition of D-glutamine blocked the D-Glu metabolic way in S. mutans, leading to malformations in bacterial cell wall, inhibition of biofilm formation, and reductions in extracellular polysaccharide (EPS) synthesis without necessarily killing this bacterium directly. The exogenous addition of D-Glu could partially reverse the inhibitory effect of D-glutamine. In conclusion, these findings suggest that the substrate analogue of glutamate racemase represents a promising anti-cariogenic agent in that it suppresses virulence properties of *S. mutans* by affecting D-Glu metabolism.

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