

Metabolic support systems remove side products and protect cells against metabolite damage

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Promiscuous enzyme activities and spontaneous chemical reactions convert normal cellular metabolites into noncanonical or damaged metabolites. These damaged metabolites can be a useless drain on metabolism and may be inhibitory and/or reactive, sometimes leading to toxicity. Thus, mechanisms to prevent metabolite damage from occurring (i.e. preemption) or to convert damaged metabolites back to physiological forms (i.e. repair) are vital. Several features of the TCA cycle make it particularly prone to metabolite damage. Enzymes of the TCA cycle are highly abundant, and many are promiscuous. TCA cycle intermediates are similar in size and structure and generally abundant, making them potential unintended substrates for promiscuous enzymes. Further, some intermediates and cofactors of the TCA cycle are inherently reactive and undergo metabolite damage via spontaneous chemical reactions. My lab has uncovered several highly conserved metabolite damage repair and damage preemption enzymes associated with the TCA cycle. Our discoveries underscore that metabolic support systems are deeply woven into the fabric of life, ensuring that core pathways like the TCA cycle can operate reliably despite constant chemical and enzymatic challenges.

Research Interests:

Metabolism

Biochemistry

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