

Review



Cite this article: Scully TW, Jiao W, Mittelstädt G, Parker EJ. 2022 Structure, mechanism and inhibition of anthranilate phosphoribosyltransferase. *Phil. Trans. R. Soc. B* **378**: 20220039. <https://doi.org/10.1098/rstb.2022.0039>

Received: 30 June 2022
Accepted: 25 August 2022

One contribution of 15 to a theme issue
‘Reactivity and mechanism in chemical and
synthetic biology’.

Subject Areas:
structural biology, biochemistry

Keywords:
PRT, AnPRT, TrpD, tryptophan biosynthesis

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Structure, mechanism and inhibition of
anthranilate phosphoribosyltransferase

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Anthranilate phosphoribosyltransferase catalyses the second reaction in the biosynthesis of tryptophan from chorismate in microorganisms and plants. The enzyme is homodimeric with the active site located in the hinge region between two domains. A range of structures in complex with the substrates, substrate analogues and inhibitors have been determined, and these have provided insights into the catalytic mechanism of this enzyme. Substrate 5-phospho- α -ribose 1-diphosphate (PRPP) binds to the C-terminal domain and coordinates to Mg^{2+} , in a site completed by two flexible loops. Binding of the second substrate anthranilate is more complex, featuring multiple binding sites along an anthranilate channel. This multi-modal binding is consistent with the substrate inhibition observed at high concentrations of anthranilate. A series of structures predict a dissociative mechanism for the reaction, similar to the reaction mechanisms elucidated for other phosphoribosyltransferases. As this enzyme is essential for some pathogens, efforts have been made to develop inhibitors for this enzyme. To date, the best inhibitors exploit the multiple binding sites for anthranilate.

This article is part of the theme issue ‘Reactivity and mechanism in chemical and synthetic biology’.

1. Introduction

Anthranilate phosphoribosyltransferase (AnPRT) catalyses the second committed step in tryptophan biosynthesis, in which a phosphoribosyl group is transferred from 5-phospho- α -ribose 1-diphosphate (PRPP) to anthranilate, forming *N*-(5-phosphoribosyl)-anthranilate (PRA) [1,2]. Tryptophan biosynthesis operates in plants and microorganisms [3–5]. Its absence in humans means that the enzymes of tryptophan biosynthesis are an attractive target for developing new therapeutics to combat infectious diseases [6]. AnPRT from *Mycobacterium tuberculosis* (MtuAnPRT) has been validated as an antimicrobial drug target against *M. tuberculosis*, the pathogen responsible for tuberculosis [7–12]. Tuberculosis continues to cause several million deaths annually and the demand for new treatments for this disease will only increase as drug-resistant strains emerge [11–14].

The enzyme AnPRT belongs to a wider group of phosphoribosyltransferases (PRTs) that all catalyse the transfer of a phosphoribosyl moiety from PRPP to a nitrogen-based nucleophile, including purines, pyrimidines and their biosynthetic precursors [15]. PRTs have been divided into four types with characteristic structural properties shared among members in each type (figure 1).

Type I PRT is the largest group and comprises mainly enzymes involved in the nucleotide salvage pathways [4,20–25]. This type includes the enzymes glutamine PRPP amidotransferase, PRPP synthase, uracil PRT, xanthine/hypoxanthine PRT, orotate PRT, guanine PRT and adenine PRT. Type I PRTs share a common α/β fold decorated by a highly flexible and multifunctional loop ($\beta 2$ - $\beta 3$ loop) and a variable hood domain that play essential roles in catalysis and regulation of the corresponding PRT [15]. Type II PRTs include quinolinate and nicotinic acid PRTs of the nicotinamide pathway, and consist of an unusual open seven-stranded α/β

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Yi-Tong Ma



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