

Information about molecular changes in am mutants

Mutant	Peptide analysis	DNA sequence analysis	Enzyme properties
<u>am1</u>	Ser336 → Phe	TCC → TTC inferred	Enzymically inactive - failure to bind NADPH
<u>am2</u>	His142 → Gln	CAC → GAC inferred	Stabilised in inactive conformation, heat labile
<u>am2l</u>	Second-site ?	" + ?	Activated by warming
<u>am3</u>	Glu393 → Gly	GAG → GGG	Stabilised in inactive conformation, reduced negative charge
<u>am3a</u>	Second site ?	GAG → GGG + ?	Greatly increased Kms for NH ₄ . NAD, glutamate
<u>am3b</u>	Second site ?		Needs activation by high α-oxoglutarate + NADPH
<u>am3-13</u>	Second site ?		Needs activation by EDTA
<u>am4</u>	Gly112 → Asp (inferred)	GGC → GAC	Enzymically inactive
<u>am6</u>	Various double f/s sequences	Single bp deletion in Ser1 codon inferred	No enzyme frameshift
<u>am7</u>	Gly372 → Ser	GGT → TGT inferred	Enzymically inactive
<u>am8</u>	-	795-796 TA → GT	No enzyme - intron 2 not spliced or frameshift splice
<u>am9</u>	-	1671-1673 CAT → TA	No enzyme - frameshift
<u>am14</u>	Leu20 → His	CTPy → CAPy inferred	Osmotically repairable - weak 4-ary structure
<u>am14R1</u>	His20 → Tyr	CAPy → TAPy inferred	Both improved stability but further improved by high osmotic pressure
<u>am14R5</u>	Second site ?	?	
<u>am15</u>	Various double f/s sequences	Deletion of C in AAC Asn56 codon	No enzyme - frameshift
<u>am17</u>	Suppressible to Tyr313	Gln313 CAG → TAG (inferred)	No enzyme
<u>am17RN35</u>	Gln313 → Tyr	TAG → TAPy inferred	Both high Km glutamate
<u>am17RU4</u>	Gln313 → Leu	TAG → TTG inferred	