Leishmania is the causative agent of the leishmaniasis disease which affects 12 million people worldwide. The clinical forms of the disease vary from skin to the visceral, which can lead to death. Unlike mammals, Leishmania is unable to synthesize purines de novo, and nucleoside diphosphate kinases (NDK) are involved in the salvage pathway in which free purines are converted to nucleosides and subsequently to nucleotides. NDKs are required for intracellular levels maintenance of NTP/NDP catalysing the transference of $\gamma$-phosphoril group from a NTP to a NDP using a ping-pong mechanism. The lethal point mutation Killer of prune (Kpn) described in Drosophila was made in the coding sequence of the LmNDK producing the P95S mutant protein. E. coli BL21 (DE3) was transformed with the pET28NDK-P95S construction and the P95S protein was purified from soluble extract by affinity chromatography. The mutant protein showed a similar catalytic activity to wild-type recombinant LmNDK, however differences in the structure in solution were observed. Far UV circular dichroism showed a decreased capacity to maintain the secondary structure in GdnHCl denaturation. The intrinsic tryptophan fluorescence of the P95S mutant presented more sensibility to pH alterations and both proteins showed a signal transition at 0.8M of GdnHCl. The mutation P95S allowed a greater ANS binding to protein structure in the presence of guanidine compared to wild type. These results suggest that the mutation could affect the folding and stability of the protein structure without affecting its catalytic activity.

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