Cation diffusion facilitator (CDF) is a ubiquitous family of divalent transition metal cations transporters. Severe diseases – such as type-II diabetes and Alzheimer's disease – that are caused by dysfunctional human CDF proteins, emphasize the importance of these proteins for normal cell function. Most CDF proteins contain two domains, the assumable-regulatory C-terminal cytoplasmic domain (CTD) and the transmembrane domain, where the cations are transported through. Although CDF proteins have been extensively studied over the past two decades, the exact mechanism of their CTD remained elusive. In our study, we use the CDF protein MamM from magnetotactic bacteria as a model protein to investigate CDF proteins’ CTD role and mechanism. Using variety of biophysical and structural techniques along with \textit{in vivo} studies, we found that structural loss of this domain specifically, decreases the function of the protein dramatically. In addition, we characterized the conformational changes that occur during metal binding and the kinetics of this domain, which together elucidate the metal binding mechanism of MamM CTD. Lastly, we show that each metal binds differently to the CTD in terms of structure and affinity and that mutations in specific residues can alter these binding properties, which indicates that the CTD has a role in metal selectivity.