ABSTRACT

CGGBP1 is a CGG-triplet binding protein which has been established as an important factor involved in a variety of cellular processes which make it an indispensable factor in cytoprotection and normal cellular homeostasis. Many interacting partners of CGGBP1 have been reported in literature previously suggesting its role as an important signalling molecule. For protein to participate in a signalling cascade, it may undergo post-translational modification at specific sites in its polypeptide chain which facilitates a conformational change thus making it either functionally active or inactive. Cell cycle is a complex process involving a plethora of protein working in coordinated manner for the progression to occur without error. CGGBP1 undergoes phosphorylation, a post-translational modification at different sites by different kinases which render it specific functions. These functions mediate cell cycle progression and other events that are a part of the division process. CGGBP1 is an essential protein in cancer cell cycle as well as normal cell cycle. To assess growth-induced regulatory roles of CGGBP1, time dependent serum stimulation in cells with normal and depleted levels of CGGBP1 was done and analysed.

The PTMs of other canonical cell cycle markers have been well studied. As CGGBP1 is involved in many cellular functions, has many interacting partners, and undergoes PTMs at specific sites, so it is pertinent to ask how other predicted binding sites on CGGBP1 are important for its functionality. Assessment of these sites with phosphodeficient and phosphomimetic mutations can be a good approach as seen in literature. Since the PTMs on CGGBP1 are predominantly phosphorylation, these mutations will tell us how relevant these sites with regard to its function are.

Casein kinase 1 is an important protein involved in many vital cellular functions among which is DNA damage and repair. DNA damage and response is a highly regulated event as any mistake during replication or repair can facilitate the subsequent daughter cells to harbour mistakes which will continue to pass on to newer generations. In this study the functional relevance of serine residues at predicted Casein Kinase 1 binding site has been elucidated.