

Osmotic stress-dependent serine phosphorylation of the histidine kinase homologue DokA

ABSTRACT

The first *in vivo* demonstration of a stimulus-dependent serine phosphorylation of an eukaryotic histidine kinase homologue was observed. This implies that DokA, while exhibiting typical structural traits of such bacterial systems, may be involved in the erythromycin-mediated signal transduction pathway.

INTRODUCTION

Osmotic stress (OS) is a condition that occurs when the body's body temperature rises. In many cases, the body's response to OS is quite mild. However, in some conditions, such as for example, high-temperature stress, the body's response to OS can become pathological. In this way, the body's response to OS can lead to many of the symptoms of OS such as heart disease, stroke, and cancer.

The aim of this study was to investigate the serine phosphorylation of the DokA homologue in the human DokA gene to determine if the DokA homologue is a marker of OS in humans.

Methods:

The first step was to analyze the serine phosphorylation of the D The bacterial signaling circuitry is composed of two-component systems that are commonly found in bacteria. These systems typically involve autophosphorylation of a histidine kinase on reconstituted residues of the histoplastic peptides and then transfer the phosphorous to an aspartate-containing receiver domain. Although their function as independent pathways was not previously known, eukaryotic two components were still found to work together. Different developmental phenotypes, such as rapid aggregation, disproportioned fruiting body and stalk ratios or impaired spore formation, are caused by the deletion of individual histidine kinases genes from the amoeba *Dictyostelium discoideum*. Furthermore, cells lacking the histochemical phosphatase gene *dokA* are omitted from high-energy (high-output) conditions, which means that they exhibit reduced viability. In this paper, we show that the histidine kinase homologue DokA is phosphorylated on serine residue *in vivo* when *Dictyostelium* cells are subjected to an extremely high molecular weight medium. We show here that our evidence confirms that "the site of initiation of transcription (phosphorylation) is located within a homologous domain only with bacterial histokinamine and that mutation of the conserved histone does not affect the seral phosphate triggered oxidation of Docinate cyton

CONCLUSION

Remarkable conclusions The serine phosphorylation of the eukaryotic homologue DokA, which is a homolog of this enzyme, has been demonstrated *in vivo* under stress. The phosphate does not depend on the conserved histidine residue that is essential for the function of two-component systems and is not due to an autophosphorousing reaction. This confirms the idea that bacterial signal transduction systems may include homologs of cellular systems involved in serine/threonine kinases as they may also participate in these processes.