# Honda Xl 25

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#### with summary in persian

multisite phosphorylation generates ultrasensitivity in the regulation of cdc25c by cdk1 abstract the mitotic phosphatase cdc25c opposes the inhibitory activity of the mitotic kinases weel and mytl as part of a double feedback loop system that gates entry into mitosis the substrate for both cdc25c and weel is the kinase cdk1 which activates cdc25c and inhibits weel via hyperphosphorylation of both proteins n terminal regulatory regions this work characterizes the structure or lack thereof of the n terminal regulatory domain of cdc25c through biochemical assays and nmr analysis furthermore while previous evidence has shown that the response of

weel phosphorylation is ultrasensitive with respect to cdk1 activity this work demonstrates that cdc25c is similarly hyperphosphorylated in an ultrasensitive fashion in xenopus extract the ultrasensitive hyperphosphorylation of cdc25c does not depend on competition from other cdk1 substrates and is unchanged in a diluted extract system the response function of cdc25c in a simplified in vitro system remains ultrasensitive although to a lesser extent this mild ultrasensitive response becomes michaelian as phosphosites are mutated indicating that some fraction of cdc25c s ultrasensitivity is the result of multisite phosphorylation by cdk1 however a significant remaining fraction of the response cannot be explained by intrinsic ultrasensitivity and appears to be due to extract specific factors

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