Rabbit Anti-Emi1 Polyclonal Antibody
100ul : Quantity
24243 : Code
-20°C : Storage

CERTIFICATE OF ANALYSIS

Antigen Species	: N terminal peptide
Reactivity	: Expected to cross-react with Human (100% identity with immunogen) due to sequence homology. Not yet tested in other species.
Conjugate	: Unconjugated
Applications	: Western Blot
Host Species	: Rabbit
Type	: Polyclonal antibody
Description	: Rabbit polyclonal to Emi1
Specific Information	: Anti-Emi1 polyclonal antibody. Emi1 is an early mitotic inhibitor that regulates mitosis by inhibiting the anaphase promoting complex/cyclosome (APC). Emi1 is a conserved F box protein containing a zinc-binding region essential for APC inhibition.

"Cells that where mock-transfected or transfected withb-TrCP1/2 siRNA were synchronised by double thymidine treatment, released from G41/S arrest in the presence of nocodazole and analysed over time for Cdc25A. As they progressed through S phase, cells treated withb-TrCP1/2 siRNA showed a substantial accumulation of Cdc25A as compared to mock-transfected cells, as well as a failure to degrade the APC inhibitor Emi1 (also known as Fbx5), a target ofb-TrCP in early mitosis(refs 1 and 2).To analyse the kinetics of Cdc25A expression at mitotic exit andin G1 phase, mock-transfected or cells transfected withb-TrCP1/2siRNA were synchronized by nocodazole treatment, released fromthe mitotic block and analysed over time for Cdc25A. Cells transfected withb-TrCP1/2 siRNA had substantially more Cdc25A at mitosis than did mock-transfected cells, but similar tocontrol cells they proceeded normally into G1 phase and degraded both Cdc25A and cyclin B1, al be it with slower kinetics. This behaviour is probably caused by an increase in Emi1 in cellstrans fected with b-TrCP siRNA resulting in an indirect up-regulation of Cdc25A through inhibition of Cdh1 at the exit of mitosis21. They directly compared the amounts of Cdc25A in mock-transfected and cells transfected withb-TrCP1/2 and Emi1 siRNA at different time points.

Emi1 did not affect the expression of Cdc25A protein. they also observed that the Cdc25AKEN2mutant, which is not degraded byAPC/CCdh1, also accumulated in cells transfected with b-TrCP1/2siRNA. Together, these data indicate that b-TrCP-mediated degradation of Cdc25A may occur throughout S and G2, and that this event is independent of the release of the Emi1-mediated inhibition of Cdh1."

references:

1. Guardavaccaro D, Kudo Y, Boulaire J, Barchi M, Busino L, Donzelli M, Margottin-Goguet F, Jackson PK, Yamasaki L, Pagano M.

   Control of meiotic and mitotic progression by the F box protein beta-Trcp1 in vivo.
   Dev Cell. 2003 Jun;4(6):799-812.

2. Margottin-Goguet F, Hsu JY, Loktev A, Hsieh HM, Reimann JD, Jackson PK.

   Prophase destruction of Emi1 by the SCF(betaTrCP/Slimb) ubiquitin ligase activates the anaphase promoting complex to allow progression beyond prometaphase.
   Dev Cell. 2003 Jun;4(6):813-26.

3. Busino L, Donzelli M, Chiesa M, Guardavaccaro D, Ganoth D, Dorrello NV, Hershko A, Pagano M, Draetta GF.

   Degradation of Cdc25A by beta-TrCP during S phase and in response to DNA damage.
   Nature. 2003 Nov 6;426(6962):87-91.