Curriculum Vitae

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Sex: male
Date of Birth: 20 Dec. 1960
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Education

1979-1985	Nagoya City University Medical School Awarded the degree of MD
1985-1989	Nagoya City University Medical School Awarded the degree of PhD in Biochemistry

Research and Professional Experience:

April, 2002-Present	Professor and Chair at Department of Biochemistry and Cell Biology, Graduate School of Medical Sciences, Nagoya City University
Sept., 2000-March, 2002	Professor and Chair at Department of Biochemistry, Nagoya City University Medical School
July, 1998-Aug., 2000	Associate Professor at Department of Biochemistry, Nagoya City University Medical School
April, 1996- June, 1998	Chief, lab. of Drug Development at Depertment of Geriatiric Research, National Institute for Longevity Sciences
Feb., 1992- Mar., 1996	Lecturer at Depertment of Biochemistry, Jichi Medical School
Mar.,1993- Feb., 1995	Research Associate, Huffington Center on Aging and Division of Molecular Virology, Baylor College of Medicine, U.S.A.
April, 1989- Jan., 1992	Research Assistant Professor at Depertment of Biochemistry, Jichi Medical School

Chk1, an essential key player that regulates S/M phase transition

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Chk1 regulates both DNA damage and replication checkpoints in mammals. Although Chk1 is crucial for embryonic cell survival, its function in normal cell cycle remains largely unknown. With the use of mouse embryonic stem cells conditionally deficient in Chk1, we now show that this kinase is indispensable for the timing of both replication origin firing and mitotic initiation. Chk1 deficiency thus resulted in premature onset of mitosis and in an increased kinase activity of cyclin B1–Cdc2; the extent of Cdc2 phosphorylation on tyrosine-15 was reduced by Chk1 depletion as a result of increased nuclear Cdc25 activity and decreased Wee1 activity. Loss of Chk1 led to abnormal timing of DNA replication during S phase progression through increased Cdc7-dependent phosphorylations of MCM2 on Ser^{26} and MCM4 on Ser^{6}. Activation of Chk1-dependent checkpoint decreased Cdc7-dependent phosphorylation of MCM4. Our results thus suggest that Chk1 regulates Cdc7 and Cdc2 to establish proper timing of the replication program and mitotic initiation during the embryonic cell cycle.