Journal of Down Syndrome & Chromosome Abnormalities

Zhao and Liang, J Down Syndr Chr Abnorm 2016, 2:1
DOI: 10.4172/2472-1115.1000106

Review Article Open Access

The Role of Casein Kinase 1 in Meiosis

Yue-Fang Zhao and Cheng-Guang Liang*

The Key Laboratory of National Education Ministry for Mammalian Reproductive Biology and Biotechnology, The Research Center for Laboratory Animal Science, College of Life Science, Inner Mongolia University, Hohhot, Inner Mongolia, People's Republic of China

Abstract

The casein kinase 1 (CK1) belongs to the serine/threonine protein kinases. CK1 members, which commonly exist in all eukaryotes, are involved in the regulation of many cellular processes linked to cell cycle progression, spindle-dynamics, and chromosome segregation. Additionally, CK1 regulate key signaling pathways such as Wnt (Wingless/Int-1), Hh (Hedgehog), and Hippo, known to be critically involved in tumor progression. Considering the importance of CK1 for accurate cell division and regulation of tumor suppressor functions, it is not surprising scientific effort has enormously increased. In mammals, CK1 regulate the transition from interphase to metaphase in mitosis. In budding yeast and fission yeast, CK1 phosphorylate Rec8 subunits of cohesin complex and regulate chromosome segregation in meiosis. During meiosis, two rounds of chromosome segregation after a single round of DNA replication produce haploid gametes from diploid precursors. Any mistake in chromosome segregation may result in aneuploidy, which in meiosis are one of the main causes of infertility, abortion and many genetic diseases in humans. This review summarizes the expression and biological function of CK1 family members, especially their role in meiosis.

Keywords: Casein kinase 1; Meiosis; Chromosome; Cell cycle

Introduction

Meiosis, which is characterized by two rounds of chromosome segregation but only one round of DNA replication, is the foundation of propagation and inheritance in sexual reproduction. Errors in chromosome separation result in aneuploidy, associated with oocyte maturation failure and consequences for aneuploidy or dysplasia in the embryo [1]. A variety of protein kinases regulate chromosome alignment and separation through phosphorylation and dephosphorylation pathways to achieve high quality-matured oocytes, and the ability to support subsequent fertilization and embryo development [2].

The casein kinase 1 (CK1) is a family of serine/threonine protein kinases, commonly existing in all eukaryotes [3]. This abundant enzymes CK1, function as monomeric (34-55 k Da) and constitutively active enzymes [4]. Until now, seven family members of CK1 were identified, which are encoded by different genes, including alpha, beta 1, gamma 1, gamma 2, gamma 3, delta, and epsilon (α, β, γ1, γ2, γ3, δ, ε) [5]. All CK1 members are quite homologous (50% identical or greater) and highly conserved within their kinase domains, but differ significantly in the length and primary structure of their C-terminal and N-terminal non-catalytic domain which is an extended tail in the case of CK1δ/ε as opposed to CK1α which has a limited C-terminal domain, but CK1γ isoforms on the other hand vary in a longer N-terminal head [6]. Although CK1 isoforms and associated splice variants are ubiquitously expressed, their activity is greatly regulated via their expression levels [7], post-translational modifications by various mechanisms including subcellular stimuli [8], subcellular compartmentalization [9], proteolytic cleavage of the C-terminus, auto- and de-phosphorylation of the C-terminal regulatory domain [10].

Substrates and Biological Functions of CK1-isoforms

A large number of substrates have been identified that are phosphorylated by CK1 isoforms *in vitro* or *in vivo*. CK1 phosphorylates the cytoskeletal proteins myosin, ankyrin, troponin, spectrin, and protein 4.1; neural filaments; neural cellular adhesion molecules; RNA polymerases I and II; translation initiation factors 4B, 4E, and 5; tRNA synthetases; simian virus 40 large T antigen; the insulin receptor; the regulatory subunit (phosphatase inhibitor 2) of protein phosphatase 1; the erythrocyte anion transporter; and metabolic enzymes, including glycogen synthase [9,11]. Some of these substrates undergo defined functional changes when phosphorylated by CKI. The CK1 family not only have numerous substrates but also regulates diverse cellular processes including a plethora of pathways responsible for differentiation [12], proliferation, cell cycle progression [13], DNA replication and damage response [14], chromosome segregation [15], mRNA metabolism [16], cytoskeleton maintenance, membrane trafficking [17], nucleo-cytoplasmic shuttling of transcription factors [18], circadian rhythms [19], Wnt signaling pathways [20], apoptosis [21], translation initiation [22], and cell migration [23,24]. Therefore CK1 deregulation has been linked to neurodegenerative diseases like Alzheimer's, sleeping disorders and proliferative diseases such as cancer [25].

Expression and Subcellular Localization of CK1

The CK1 has been found in the nucleus, cytoplasm and membrane fractions of eukaryotic cells [4]. The subcellular localization of the CK1 isoforms is mainly regulated by binding to intracellular structures or protein complexes and thereby plays a role in substrate-recognition and CK1 activity [26,27]. The CK1 isoforms was found to play an important role in cancer progression in different types of tumors [28]. The localization of CKlα in the mitosis was researched by immunofluorescent studies using mouse fibroblasts and CHO cells [9]. In interphase cells, CK1α was localized to vesicular cytosolic structures. When cells enter prophase, the localization of CK1α increases around the centrosome, and there is uniform colocalization of CK1α with the microtubule aster. As cells progress into mitosis, the CK1α becomes localized to mitotic spindle fibers and there is no longer detectable astral microtubule localization. Moreover, in mitosis,

***Corresponding author:** Cheng-Guang Liang, The Research Center for Laboratory Animal Science, Inner Mongolia University, No. 24 Zhao Jun Road, Hohhot, Inner Mongolia 010070, People's Republic of China, Tel: 86-471-3679862; E-mail: liangchengguang@gmail.com

Received February 02, 2016; **Accepted** February 25, 2016; **Published** March 02, 2016

Citation: Davis TN, Camarata S, Camarata M (2016) Cross Modal Generalization of Receptive and Expressive Vocabulary in Children with Down Syndrome. J Down Syndr Chr Abnorm 2: 105. doi:10.4172/2472-1115.1000106

CK1α localizes to kinetochore fibers. As cells progress into anaphase, CK1α localization appears restricted to regions of microtubules between the centrosome and the chromosomes. The CK1α localization on microtubules decreases as cells progress to telophase. In telophase, CK1α is localized to the centrosome, but there is no localization to midbody microtubules [29]. Taken together, these results suggest that CK1α relocalizes from vesicular structures to the centrosome and aster microtubules in prophase and with kinetochore microtubules and possibly other interpolar microtubules during mitosis [30]. CK1δ was localized to the centrosomes, co-localized with γ-tubulin and associated with the mitotic spindle in many types of cells [31]. CK1ε is most like CK1δ with greater than 98% identity within the protein kinase domain, and also reported to be localized to the centrosomes in several types of cells [32].

During mouse oocyte meiosis maturation and early embryo development, Ck1α mRNA was all expressed from GV to the late 1-cell stage (GV, GVBD, MI, AI-TI, MII, AII-TII, pronuclear and late 1-cell stage), achieved the highest level in M phase, including MI, MII and metaphase of the 1-cell stage when chromosomes are highly congressed. The CK1α protein expression profile was consistent with the profile of mRNA levels except for GV [33]. The protein expression level of CK1δ and CK1ε were same as CK1α, relatively low at GV stages and then gradually increased at MI and MII stages [34].

The subcellular localization of CK1α in oocyte meiosis had some contradictory results (Figure 1). The first research showed that CK1α localized to the plasma membrane in the mouse GV oocyte and in the spindle area in the MII stage [35]. Subsequent our results showed that CK1α was colocalized with condensed and congressed chromosomes in mouse oocyte meiosis and mitosis of early embryo development, but was not detected in the spindle area. Recently, Qi et al. investigated subcellular localization of endogenous CK1 using immune fluorescent analysis with CK1δ and CK1ε specific antibodies and reported that CK1δ was distributed in both cytoplasm and GV, with slightly more concentrating in the GV, and there were usually one or two point signals adjacent to or around the GV at GV stage. At MI and MII stages, when chromosomes were aligned at the equatorial plates, CK1δ was localized to the two sides of aligned chromosomes. The localization pattern of CK1ε was similar to that of CK1δ. They also detected the localization of exogenous CK1α and found that exogenous CK1α was mainly localized to the spindle poles in mouse oocytes at both MI and MII stages. The different localizations of CK1α observed may be due to the different specificities of the CK1α antibodies that were used, as polyclonal antibodies could give non-specific signals.

The Role of CK1 Isoforms in Chromosomes Segregation

The CK1 isoforms represent central components to regulate the

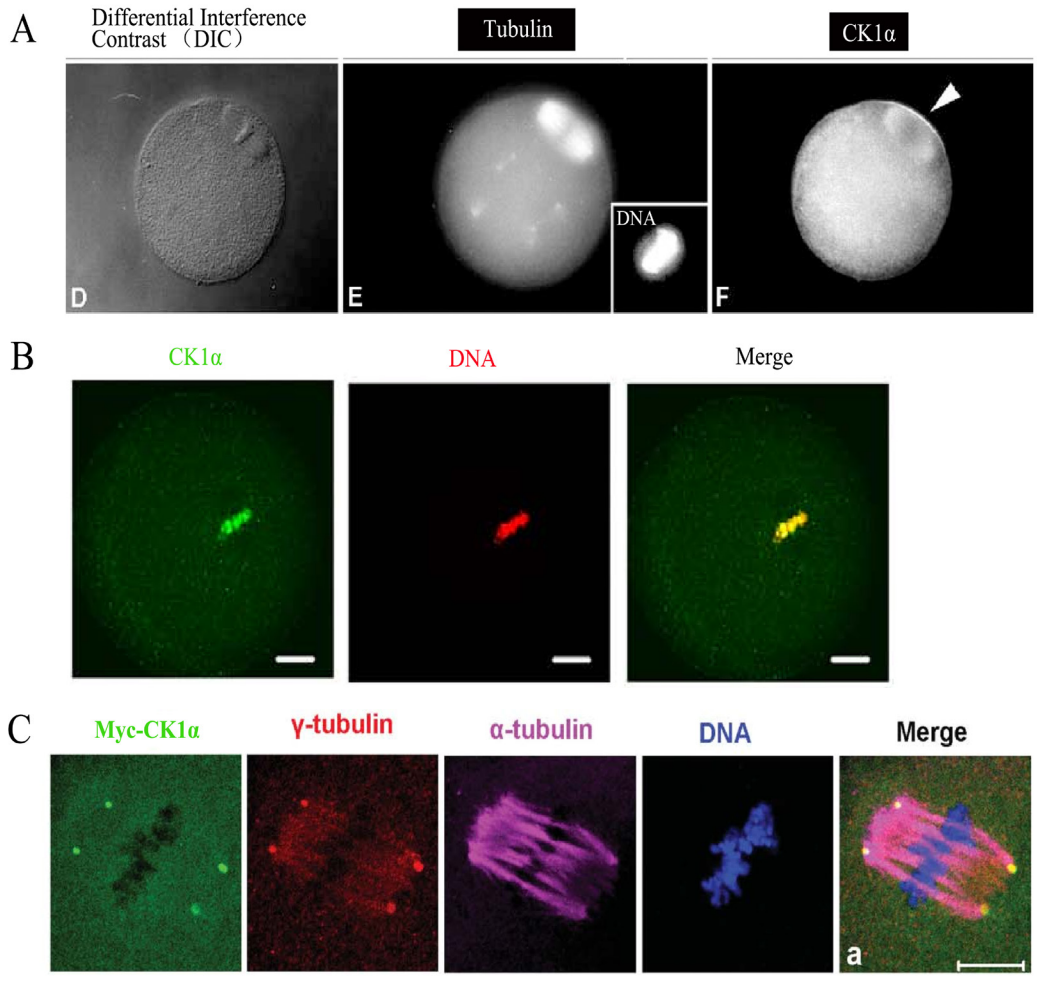

Figure 1: Subcellular localization of CK1α in mouse MII oocyte. (A) CK1α was associated with the spindle. The data comes from Gross SD, 1997. (B) CK1α was colocalized with condensed and congressed chromosomes. The data comes from Wang L, 2013. (C) CK1α was concentrated at spindle poles and co-localized with γ-tubulin. The data comes from Qi ST, 2015.

cell cycle progression, spindle-dynamics, and chromosome segregation [5]. CK1α is located at the centrosome, microtubule asters, and the kinetochore in mitosis. Depletion of CK1α using an RNAi based approach in Drosophila cultured cells, resulted in perturbation of interphase nuclear morphology. The results also showed that CK1α functioned to prevent centromere dispersal, inhibited chromosome compaction, and promoted chromosome pairing. Therefore, CK1α is an important modulator of interphase genome organization, regulating homologous chromosome pairing, centromere clustering, and chromosome compaction in Drosophila cultured cells and *in vivo* [36]. Furthermore, CK1α affected these processes by attenuating interphase condensin II activity as CK1α function was required for protein turn-over of the Cap-H2 condensin II subunit. CK1α was a negative regulator of interphase condensin II activity by promoting Cap-H2 destruction and limiting chromatin-bound Cap-H2 levels [37].

In addition, CK1δ especially associates with the spindle apparatus during mitosis and directly modulates MT by phosphorylation of α-, β-, and γ-tubulin, thereby exerting stress-induced functions at the spindle apparatus and the centrosome [38]. Recently, knockdown of CK1δ by siRNA was reported to inhibit microtubule nucleation at the Golgi apparatus [39]. Furthermore, homologs of CK1, such as casein kinase 1-like 6 (CKL6), associate with cortical MT in vivo and phosphorylate tubulin in vitro [40]. CK1δ regulates microtubule- and spindle-dynamics in response to genotoxic stress in order to maintain genomic stability by site-specific phosphorylation of tubulin.

The nuclear enzyme topoisomerase IIα (TopoIIα) is able to cleave DNA in a reversible manner and can facilitate transcription and replication of chromatin templates in mammalian cells. CK1ε phosphorylates TopoIIα on serine 1106 in G2/M cells, inducing the TopoIIα chromosome localization and decatenation function as well as sensitivity to TopoIIα -targeting drugs. Cdc7 cosilencing or CKIε overexpression in geminin-silenced cells restored TopoIIα chromosomal localization and prevented the formation of chromosome bridges. This finding suggests that CKIε is a positive regulator and Cdc7 is a negative regulator of TopoIIα chromosomal localization and function [41].

Numerous studies have demonstrated that CK1 might regulate cell cycle progression through interaction with the Wnt pathway and p53. CK1α formed a genetic and physical interaction with the murine double minute chromosome 2 (MDM2) oncoprotein resulting in degradation of the p53 tumour suppressor. Pharmacological inhibition of CK1 increases p53 protein level and induces cell death, whilst RNAi-mediated depletion of CK1α stabilizes p53 and induces growth arrest [25]. Therefore, the coordinated function of both CK1 and p53 could ensure the integrity of the centrosome and thereby maintain genomic stability. CK1δ is associated to the centrosome and related to Wnt3-dependent neurite outgrowth. CK1δ co-localizes with DVL2 at basal bodies and gradually accumulates at centrosomes when cells proceed through the cell cycle [42]. CK1δ and CK1ε are anchored at the centrosome through interaction with AKAP450 [31] and that CK1δ phosphorylates EB1, which is relevant for centrosome positioning during T-cell activation [43]. These further underlined that CK1 had regulatory roles at the centrosome.

The Role of CK1 Isoforms in Meiosis

In mammalian cells, CK1α was first reported to localize to vesicular cytolic structures and centrosome in interphase cells, and then associated with spindles in mitosis [9]; CK1δ was localized to centrosomes, co-localized with γ-tubulin and was associated with the mitotic spindles under DNA damage; and CK1ε was concentrated at centrosomes [31]. Cell treated with IC261 caused mitotic spindle defects and cell cycle arrest. All these results indicated that CK1 activity may be related to spindle assembly [38].

In *S. cerevisiae*, deletion of CK1 (HRR25 gene) results in a phenotype in which the yeast cannot enter meiosis or undergo division but are defective in the ability to segregate chromosomes and during mitosis a fraction of cells are defective in nuclear segregation [44]. Two other protein kinases from yeast have been cloned (YCK 1 and 2) that are homologous to the bovine CK1s; deletion of either of these CK1s results in no phenotypic change, but when both are deleted, the cells are not viable [45].

In budding yeast, CK1 δ/ε was a subunit of the monopolin complex required for the mono-orientation of sister kinetochores during meiosis I. The fission yeast CK1 δ/ε isoforms Hhp1 and Hhp2 were required for full levels of Rec8 phosphorylation and for efficient removal of Rec8 at the onset of anaphase I revealing a novel function of this evolutionarily conserved kinase (Figure 2). First, CK1δ/ε isoforms

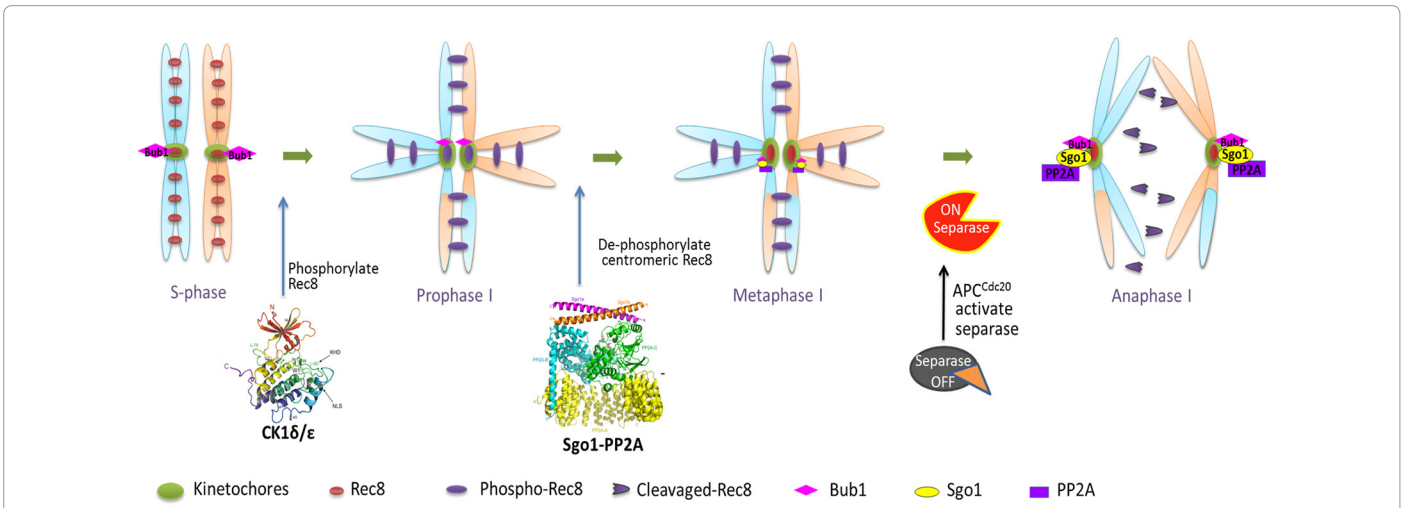

Figure 2: Model of CK1 in the segregation of chromosomes during meiosis I. First, CK1δ/ε isoforms phosphorylate Rec8. Second, a complex of Shugoshin (Sgo) and PP2A is recruited by Bub1 to centromeres to protect centromeric cohesin by dephosphorylating Rec8. Then, segregation of chromosomes during meiosis I was triggered by separase cleavage of phosphorylated Rec8 along chromosome arms, while centromeric cohesin was protected from separase cleavage during meiosis I by the Sgo1/PP2A complex.

phosphorylate Rec8. Second, a complex of Shugoshin (Sgo) and PP2A is recruited by Bub1 to centromeres to protect centromeric cohesin by dephosphorylating Rec8. Then, segregation of chromosomes during meiosis I was triggered by separase cleavage of phosphorylated Rec8 along chromosome arms, while centromeric cohesin was protected from separase cleavage during meiosis I by the Sgo1/PP2A complex. Centromeric cohesin was only cleaved during meiosis II, hence allowing segregation of sister chromatids. Rec8 phosphorylation promotes its removal at the onset of anaphase raises the possibility that the Sgo1/PP2A complex protects centromeric cohesin by dephosphorylating Rec8. *S. cerevisiae* Rec8 with phosphomimetic mutations is no longer protected from separase at centromeres during meiosis I and forced localization of excess CK1δ/ε at the pericentromeric region abrogates the ability of Sgo1/PP2A to protect centromeric Rec8. These results suggested that Sgo1/PP2A protects centromeric cohesin from separase cleavage by opposing CK1-dependent phosphorylation of Rec8. This mechanism may be evolutionarily conserved, because similar observations were made in budding and fission yeast, and both Rec8 and CK1 proteins are present in most eukaryotes.

Rec8 phosphorylation by CK1 and Cdc7-Dbf4 kinase regulates cohesin cleavage by separase during meiosis. In addition, previous studies showed that CK1α exists and is activated in mouse oocytes and that it regulates procession of interphase to mitosis during the first mitotic cell cycle in embryos [35].

In 2010, we used proteomics analysis to compare protein difference of mouse oocyte MII stage meiotic spindles between normal and spindles that form after somatic cell nuclear transfer (SCNT). We found that CK1α was significantly decreased in SCNT constructs, accompanied by chromosomes misalignment in SCNT embryos [46]. Then, we investigated function of CK1α during mouse oocyte meiotic maturation and early embryo development. The functions of CK1α on chromosome alignment also were studied by functional depletion, pharmaceutical inhibition and activation approaches. The results showed that both inhibition and activation of CK1α had negative effect on oocyte maturation and early embryo development. Depletion of CK1α with specific-morpholinos caused severe protein expression decreased and consequently observed defects in the interaction of microtubules and centromeres on chromosomes, plate thickness increased and failure of the first polar body extrusion. Blocking of CK1α function with D4476, (4[4-(2,3-dihydro-benzo[1,4]-dioxin-6-yl)-5-pyridin-2-yl-1-H-imidazol-2-yl]benzamidine) which is an ATP-competitive inhibitor, had the same results with the morpholino injection, such as expression of CK1a was decreased; chromosome congression failed; oocytes arrested at pro-MI phase; PB1 extrusion failed or extruded giant PB1; the ability of embryo development was impaired. However, mouse oocytes treated with pyrvinium pamoate (PP), which increased phosphorylation and allosteric activation of CK1α [47], induced oocyte meiotic maturation failure, severe congression abnormalities and misalignment of chromosomes. These results indicated that CK1α affect homologous chromosome alignment, congression and segregation in the oocyte meiosis and play an important role in early embryo development.

CK1α exhibited analogous functions with surviving,that correlated with the interaction of microtubules and centromeres on chromosomes in the process of oocyte meiotic maturation [48]. Casein kinase 2 (CK2), another casein kinase family member, directly interacted with survivin through β-catenin-Tcf/Lef-mediated transcription in Wnt signaling [49]. Meanwhile, CK1α also participated in the same pathway and allosteric activation of CK1α as an effective mechanism to inhibit Wnt signaling. Wnt signaling increases cytoplasmic levels of β-catenin, which enters the nucleus and interacts with other factors to activate a β-catenin-Tcf/Lef-mediated transcriptional program [47]. But high expression of β-catenin induced abnormal chromosome separation. Therefore we suppose that CK1α might involve in chromosome alignment through the Wnt signaling pathway. Furthermore, two homolog chromosomes were connected with each other at metaphase I, and cohesion together with Rec8 mediated the linkage between sister chromatid arms in the bivalent chromosomes [50]. Rec8 was phosphorylated along chromosomes and Sgo1-PP2A prevents the centromeric Rec8 dephosphorylation in meiosis. Once anaphase I is initiated, phosphorylated Rec8 on the chromosome arms was cleaved by separase, leading to chromosome separation [51]. Blocking CK1α may cut off the phosphorylation of downstream factors and then relevant proteins are unable to integrate with chromosome arms or centromeres. Consequently, chromosome dynamics were affected and anaphase I failed to proceed. However, constitutively activated Ck1α inhibits the function of the Wnt/β-catenin signaling pathway to affect the function of securin, which was a key factor in oocyte meiosis and a target of β-catenin transcriptional activation in colorectal adenomas [52]. CK1α phosphorylated Ser45 of β-catenin is the priming reaction of β-catenin proteasomal degradation [53]. And high expression of β-catenin induced abnormal chromosome separation [52]. Thus, constitutively activated Ck1α might disturb the securin inducing the PB1 extrusion failure and misalignment chromosome.

Our experimental data suggested that CK1α has a pivotal role in regulating chromosome congression and separation during oocyte meiotic maturation and early embryo development [33]. However, a recent study of Qi et al. was inconsistent with our results, which showed that CK1α, CK1δ and CK1ε were not essential for chromosome separation, as loss-of-function of CK1α, CK1δ and CK1ε either individually or simultaneously by RNAi or dominant-negative approach had no effect on homologous chromosome segregation and polar body extrusion. Furthermore, overexpression of exogenous CK1α, CK1δ and CK1ε, both individually and simultaneously, caused little effect on eitherthe meiosis process, spindle organization or chromosome alignment. Their report demonstrate that although CK1α, CK1δ and CK1ε were all localized to MTOCs or spindle poles, they were not the kinase responsible for Rec8 phosphorylation during mouse oocyte meiosis, indicating that CK1 was probably not essential for mouse oocyte meiotic maturation [34]. The contradictory results need further investigation.

Conclusions

Isoforms of CK1 family have been shown to phosphorylate key regulatory molecules involved in cell cycle, transcription and translation, the structure of the cytoskeleton, cell-cell adhesion and receptor-coupled signal transduction. Respecting this involvement in important cellular signal transduction pathways, it is reasonable that dysregulation of CK1 isoforms has been linked to the incidence of inflammatory and proliferative diseases but also to neurodegenerative disorders. In this review we are mostly interested in the cell division. CK1 isoforms play an important role in cell division, both mitosis and meiosis. In mammals, CK1 regulate the transition from interphase to metaphase in mitosis. In budding yeast and fission yeast, CK1 phosphorylate Rec8 subunits of cohesin complex and regulate chromosome segregation in meiosis. However, the functions of CK1 in mammalian oocyte meiosis remain elusive and more researches are still need.

Grant support

This work was supported by the National Natural Science Foundation of China to Y.Z. (31560333) and C.L. (31160243, 31371454); the Natural Science Foundation of Inner Mongolia Autonomous Region of China (2015JQ02) to C.L., Program of Higher Level Talents of Inner Mongolia University (SPH-IMU 135131) to Y.Z.

References

1. Matsuura S, Ito E, Tauchi H, Komatsu K, Ikeuchi T (2000) Chromosomal instability syndrome of total premature chromatid separation with mosaic variegated aneuploidy is defective in mitotic-spindle checkpoint. Am J Hum Genet 67: 483-486.
2. Musacchio A, Salmon ED (2007) The spindle-assembly checkpoint in space and time. Nat Rev Mol Cell Biol 8: 379-393.
3. Tuazon PT, Traugh JA (1991) Casein kinase I and II--multipotential serine protein kinases: structure, function, and regulation. Adv Second Messenger Phosphoprotein Res 23: 123-164.
4. Gross SD, Anderson RA (1998) Casein kinase I: spatial organization and positioning of a multifunctional protein kinase family. Cell Signal 10: 699-711.
5. Knippschild U, Gocht A, Wolff S, Huber N, Löhler J, et al. (2005) The casein kinase 1 family: participation in multiple cellular processes in eukaryotes. Cell Signal 17: 675-689.
6. Cheong JK, Virshup DM (2011) Casein kinase 1: Complexity in the family. Int J Biochem Cell Biol 43: 465-469.
7. Utz AC, Hirner H, Blatz A, Hillenbrand A, Schmidt B, et al. (2010) Analysis of cell type-specific expression of CK1 epsilon in various tissues of young adult BALB/c Mice and in mammary tumors of SV40 T-Ag-transgenic mice. J Histochem Cytochem 58: 1-15.
8. Bedri S, Cizek SM, Rastarhuyeva I, Stone JR (2007) Regulation of protein kinase CK1alphaLS by dephosphorylation in response to hydrogen peroxide. Arch Biochem Biophys 466: 242-249.
9. Brockman JL, Gross SD, Sussman MR, Anderson RA (1992) Cell cycle-dependent localization of casein kinase I to mitotic spindles. Proc Natl Acad Sci U S A 89: 9454-9458.
10. Budini M, Jacob G, Jedlicki A, Pérez C, Allende CC, et al. (2009) Autophosphorylation of carboxy-terminal residues inhibits the activity of protein kinase CK1alpha. J Cell Biochem 106: 399-408.
11. Agostinis P, Vandenheede JR, Goris J, Meggio F, Pinna LA, et al. (1987) The ATP,Mg-dependent protein phosphatase: regulation by casein kinase-1. FEBS Lett 224: 385-390.
12. Banerjee D, Chen X, Lin SY, Slack FJ (2010) kin-19/casein kinase Iî± has dual functions in regulating asymmetric division and terminal differentiation in C. elegans epidermal stem cells. Cell Cycle 9: 4748-4765.
13. Yang WS, Stockwell BR (2008) Inhibition of casein kinase 1-epsilon induces cancer-cell-selective, PERIOD2-dependent growth arrest. Genome Biol 9: R92.
14. Santos JA, Logarinho E, Tapia C, Allende CC, Allende JE, et al. (1996) The casein kinase 1a gene of Drosophila melanogaster is developmentally regulated and the kinase activity of the protein induced by DNA damage. J Cell Sci 109:1847-1856.
15. Rumpf C, Cipak L, Dudas A, Benko Z, Pozgajova M, et al. (2010) Casein kinase 1 is required for efficient removal of Rec8 during meiosis I. Cell Cycle 9: 2657-2662.
16. Gross SD, Loijens JC, Anderson RA (1999) The casein kinase Ialpha isoform is both physically positioned and functionally competent to regulate multiple events of mRNA metabolism. J Cell Sci 112 : 2647-2656.
17. Lee JY (2009) Versatile casein kinase 1: multiple locations and functions. Plant Signal Behav 4: 652-654.
18. Biswas A, Mukherjee S, Das S, Shields D, Chow CW, et al. (2011) Opposing action of casein kinase 1 and calcineurin in nucleo-cytoplasmic shuttling of mammalian translation initiation factor eIF6. J Biol Chem 286: 3129-3138.
19. Shanware NP, Hutchinson JA, Hwa KS, Lihong Z, Bowler MJ, et al. (2011) Casein kinase 1-dependent phosphorylation of familial advanced sleep phase syndrome-associated residues controls PERIOD 2 stability. J Biol Chem 286: 12766-12774.
20. Peters JM, McKay RM, McKay JP, Graff JM (1999) Casein kinase I transduces Wnt signals. Nature 401: 345-350.
21. Huart AS, MacLaine NJ, Meek DW, Hupp TR (2009) CK1alpha plays a central role in mediating MDM2 control of p53 and E2F-1 protein stability. J Biol Chem 284: 32384-32394.
22. Hutchinson JA, Shanware NP, Chang H, Tibbetts RS (2011) Regulation of ribosomal protein S6 phosphorylation by casein kinase 1 and protein phosphatase 1. J Biol Chem 286: 8688-8696.
23. Elyada E, Pribluda A, Goldstein RE, Morgenstern Y, Brachya G, et al. (2011) CKIÎ± ablation highlights a critical role for p53 in invasiveness control. Nature 470: 409-413.
24. Foldynová-Trantírková S, Sekyrová P, Tmejová K, Brumovská E, Bernatík O, et al. (2010) Breast cancer-specific mutations in CK1epsilon inhibit Wnt/beta-catenin and activate the Wnt/Rac1/JNK and NFAT pathways to decrease cell adhesion and promote cell migration. Breast Cancer Res 12: R30.
25. Huart AS, MacLaine NJ, Narayan V, Hupp TR (2012) Exploiting the MDM2-CK1Î± protein-protein interface to develop novel biologics that induce UBL-kinase-modification and inhibit cell growth. PLoS One 7: e43391.
26. Behrend L, Stöter M, Kurth M, Rutter G, Heukeshoven J, et al. (2000) Interaction of casein kinase 1 delta (CK1delta) with post-Golgi structures, microtubules and the spindle apparatus. Eur J Cell Biol 79: 240-251.
27. Zemp I, Wandrey F, Rao S, Ashiono C, Wyler E, et al. (2014) CK1Î´ and CK1Îµ are components of human 40S subunit precursors required for cytoplasmic 40S maturation. J Cell Sci 127: 1242-1253.
28. Knippschild U, Wolff S, Giamas G, Brockschmidt C, Wittau M, et al. (2005) The role of the casein kinase 1 (CK1) family in different signaling pathways linked to cancer development. Onkologie 28: 508-514.
29. Schittek B, Sinnberg T (2014) Biological functions of casein kinase 1 isoforms and putative roles in tumorigenesis. Mol Cancer 13: 231.
30. Milne DM, Looby P, Meek DW (2001) Catalytic activity of protein kinase CK1 delta (casein kinase 1delta) is essential for its normal subcellular localization. Exp Cell Res 263: 43-54.
31. Sillibourne JE, Milne DM, Takahashi M, Ono Y, Meek DW (2002) Centrosomal anchoring of the protein kinase CK1delta mediated by attachment to the large, coiled-coil scaffolding protein CG-NAP/AKAP450. J Mol Biol 322: 785-797.
32. Fish KJ, Cegielska A, Getman ME, Landes GM, Virshup DM (1995) Isolation and characterization of human casein kinase I epsilon (CKI), a novel member of the CKI gene family. J Biol Chem 270: 14875-14883.
33. Wang L, Lu A, Zhou HX, Sun R, Zhao J, et al. (2013) Casein Kinase 1 Alpha Regulates Chromosome Congression and Separation during Mouse Oocyte Meiotic Maturation and Early Embryo Development. Plos One 8: 402-402.
34. Qi ST, Wang ZB, Huang L, Liang LF, Xian YX, et al. (2015) Casein kinase 1 (Î±, Î´ and Îµ) localize at the spindle poles, but may not be essential for mammalian oocyte meiotic progression. Cell Cycle 14: 1675-1685.
35. Gross SD, Simerly C, Schatten G, Anderson RA (1997) A casein kinase I isoform is required for proper cell cycle progression in the fertilized mouse oocyte. J Cell Sci 110 : 3083-3090.
36. Knippschild U, Krüger M, Richter J, Xu P, García-Reyes B, et al. (2014) The CK1 Family: Contribution to Cellular Stress Response and Its Role in Carcinogenesis. Front Oncol 4: 96.
37. Nguyen HQ, Nye J, Buster DW, Klebba JE, Rogers GC, et al. (2015) Drosophila casein kinase I alpha regulates homolog pairing and genome organization by modulating condensin II subunit Cap-H2 levels. PLoS Genet 11: e1005014.
38. Behrend L, Milne DM, Stoter M, Deppert W, Campbell LE, et al. (2000) IC261, a specific inhibitor of the protein kinases casein kinase 1-delta and -epsilon, triggers the mitotic checkpoint and induces p53-dependent postmitotic effects. Oncogene 19: 5303-5313.
39. Greer YE, Westlake CJ, Gao B, Bharti K, Shiba Y, et al. (2014) Casein kinase 1Î´ functions at the centrosome and Golgi to promote ciliogenesis. Mol Biol Cell 25: 1629-1640.
40. Ben-Nissan G, Cui W, Kim DJ, Yang Y, Yoo BC, et al. (2008) Arabidopsis casein kinase 1-like 6 contains a microtubule-binding domain and affects the organization of cortical microtubules,. Plant Physiol 148: 1897-1907.

41. Lauren G, Rohit M, Yoshiko S, Nicole ME, Ishamy WM (2011) Geminin Overexpression Prevents the Completion of Topoisomerase IIa Chromosome Decatenation Leading to Aneuploidy in Human Mammary Epithelial Cells. Breast Cancer Res13: 53.

42. Lee KH, Johmura Y, Yu LR, Park JE, Gao Y, et al. (2012) Identification of a novel Wnt5a-CK1É›-Dvl2-Plk1-mediated primary cilia disassembly pathway. EMBO J 31: 3104-3117.

43. Zyss D, Ebrahimi H, Gergely F (2011) Casein kinase I delta controls centrosome positioning during T cell activation. J Cell Biol 195: 781-797.

44. Alexandru G, Uhlmann F, Mechtler K, Poupart MA, Nasmyth K (2001) Phosphorylation of the cohesin subunit Scc1 by Polo/Cdc5 kinase regulates sister chromatid separation in yeast. Cell 105: 459-472.

45. Hornig NC, Uhlmann F (2004) Preferential cleavage of chromatin-bound cohesin after targeted phosphorylation by Polo-like kinase. EMBO J 23: 3144-3153.

46. Han Z, Liang CG, Cheng Y, Duan X, Zhong Z, et al. (2010) Oocyte spindle proteomics analysis leading to rescue of chromosome congression defects in cloned embryos. J Proteome Res 9: 6025-6032.

47. Thorne CA, Hanson AJ, Schneider J, Tahinci E, Orton D, et al. (2010) Small-molecule inhibition of Wnt signaling through activation of casein kinase 1α. Nat Chem Biol 6: 829-836.

48. Shao-Chen S, Liang W, Mo L, Sheng-Li L, Bao-Zeng X, et al. (2009) Perturbation of survivin expression affects chromosome alignment and spindle checkpoint in mouse oocyte meiotic maturation. Cell Cycle 8: 3365-3372.

49. Tapia JC, Torres VA, Rodriguez DA, Leyton L, Quest AF (2006) Casein kinase 2 (CK2) increases survivin expression via enhanced beta-catenin-T cell factor/lymphoid enhancer binding factor-dependent transcription. P Natl Acad Sci USA 103: 15079-15084.

50. Lee J, Iwai T, Yokota T, Yamashita M (2003) Temporally and spatially selective loss of Rec8 protein from meiotic chromosomes during mammalian meiosis. J Cell Sci 116: 2781-2790.

51. Katis VL, Lipp JJ, Imre R, Bogdanova A, Okaz E, et al. (2010) Rec8 phosphorylation by casein kinase 1 and Cdc7-Dbf4 kinase regulates cohesin cleavage by separase during meiosis. Dev Cell 18: 397-409.

52. Hlubek F, Pfeiffer S, Budczies J, Spaderna S, Jung A, et al. (2006) Securin (hPTTG1) expression is regulated by beta-catenin/TCF in human colorectal carcinoma. Br J Cancer 94: 1672-1677.

53. Liu C, Li Y, Semenov M, Han C, Baeg GH, et al. (2002) Control of beta-catenin phosphorylation/degradation by a dual-kinase mechanism. Cell 108: 837-847.

Citation: Zhao YF, Liang CG (2016) The Role of Casein Kinase 1 in Meiosis. J Down Syndr Chr Abnorm 2: 106. doi:10.4172/2472-1115.1000106