—Original Article—

RNA-associated protein LSM family member 14 controls oocyte meiotic maturation through regulating mRNA pools

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Abstract. LSM family member 14 (LSM14) belongs to the RNA-associated protein (RAP) family that is widely expressed in different species, and whose functions include associating and storing mRNAs. In the present study, we found that LSM14b was essential for oocyte meiotic maturation. Lack of LSM14b caused oocyte meiotic arrest at metaphase, and misalignment of chromosomes, as well as abnormal spindle assembly checkpoint (SAC) and maturation promoting factor (MPF) activation. *Cyclin B1* and *Cdc20* mRNAs, whose contents changed with LSM14b expression, were likely direct targets of LSM14b. We conclude that LSM14b, by functioning as a container of mRNAs, controls protein expression, and thus regulates the oocyte meiotic maturation process.
Key words: CDC20, LSM family member 14 (LSM14), mRNA degradation, Oocytes, RNA-associated protein

O ocytes synthesize and accumulate a large pool of mRNAs in the form of ribonucleoprotein (RNP) particles. Some RNPs are actively translated, while others' translation is suppressed until oocytes reach subsequent developmental stages, which is what we describe as being 'masked'. Prior to zygotic transcription there is no mRNA synthesis, because maternal mRNAs direct all required protein synthesis; thus, translational control is the main mechanism that regulates gene activity during this early development. This translational control is generally correlated with polyadenylation or poly(A) [1–3]. The poly(A) on actively translated mRNAs is on average 100–200 nucleotides in length. The activation of masked mRNAs is associated with the elongation of their poly(A) in the cytoplasm to at least 100 nucleotides [4, 5]. Conversely, the inactivation of mRNAs during early development is associated with deadenylation, but unlike in somatic cells, it does not result in immediate mRNA degradation [6, 7].

Among these RNPs, there is a family member named RAP55 that is an RNA-associated protein of 55 kDa that was first discovered in the newt *Pleurodeles waltl* and in *Xenopus laevis* [8, 9]. It was initially thought to be specifically expressed in oocytes. There is a highly conserved domain called LSm14 [8] in the RAP55 protein sequence, so we also call RAP55A and RAP55B as LSM14a and LSM14b. The LSm14 domain is a well-conserved N-terminal domain, and previous research has determined that Sm and LSm proteins are generally involved in RNA metabolism [10]. The LSm domain forms a closed hetero-heptameric ring barrel using antiparallel β-strands and α-helix, and this kind of ring barrel is generally involved in recognizing and binding specific RNAs [11].

Human RAP55A was first detected in autoimmune patients together with P-bodies [12], which are cytoplasmic foci that contain translationally repressed mRNAs and part of the 5′-3′ mRNA decay-relating enzymes. The expression level of xRAP55 increases throughout oogenesis, together with the accumulation of maternal mRNAs; conversely, its level is constant during embryogenesis [13]. Therefore, we asked why do vertebrates exhibit two evolved types of the RAP55 protein ? Do they have different functions or distinct roles ? Possibly, one paralog is oocyte specific while the other is expressed in somatic cells. Considering that RAP55A is widely expressed in somatic cells [13], we propose that RAP55B may be important in germ cells.

Materials and Methods

Ethics statement

Mouse handling was conducted in accordance with policies promulgated by the Ethics Committee of the Institute of Zoology, Chinese Academy of Science. The ICR mice used in our study were housed in the animal core facility licensed by the experimental animal committee of the city of Beijing.

Mouse oocyte collection and in vitro maturation

The germinal vesicle (GV)-stage oocytes were isolated from the

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ovaries of 8–10-week-old ICR female mice without any drug treatment. We used a razor blade to dissect the ovaries, and collected denuded oocytes. These were then cultured in M2 medium for at least 12 h before maturation, in an incubator with environmental conditions comprising 5% CO_2, 37°C, and saturated humidity.

RNA interference

The GV oocytes, cultured in M2 medium containing 2.5 µM milrinone, were microinjected with 5–10 pl of 50-µM siRNA. The sequences of our siRNAs were as follows: *Lsm14a* siRNA 5′-GGUUCGUCGUCAUCUUCAUUTT-3′, *Lsm14b*-1 siRNA 5′-GCUAUGAGGGCAUCCUCUAUTT-3′, *Lsm14b*-2 siRNA 5′-GCAAUCGGAGAACAAGAAAUTT-3′. The injected oocytes were incubated for 24 h in M2 medium containing 2.5 µM milrinone. To resume meiosis, the oocytes were washed in milrinone-free M2 medium at least three times, and then incubated in M2 medium.

Real-time PCR

Total RNA was extracted from 70–80 oocytes using an RNeasy micro purification kit (Qiagen, Austin, TX, USA). We then generated single-strand cDNA with the cDNA synthesis kit (Takara, Otsu, Japan), using polyT primers. These cDNAs were used as templates to amplify *Lsm14a, Lsm14b, Cyclin B1,* and *Cdc20*, while *Ppia* was used as the reference. The primers we used were *Lsm14a*: 5′-AGATCCGCTACGAGGGCAT-3′ (forward), 5′-TCGAGGCGGTATTGGACGA-3′ (reverse); *Lsm14b*: 5′-CACCGACAACTCCACCGTG-3′ (forward), 5′-TCCCCGGAAAATGATGTACTCA-3′ (reverse); *Cyclin B1*: 5′-AAGGTGCCTGTGTGTGAACC-3′ (forward), 5′-GTCAGCCCCATCATCTGCG-3′ (reverse); *Cdc20*: 5′-TTCGTGTTCGAGAGCGATTTG-3′ (forward), 5′-ACCTTGGAACTAGATTTGCCAG-3′ (reverse). The PCR was performed in a Roche Light Cycler 480, using SYBR Premix (Kangwei, Beijing, China). Relative gene expression was measured using real-time quantitative PCR and the 2(-Delta DeltaC(T)) method.

Plasmid construction and LSM14a-myc overexpression

We used control cDNAs (reverse-transcribed as above) as templates for amplification. *Lsm14a* DNA was amplified via nest PCR, using outer primers comprising 5′-CGGGATCTGACTGAGTGCGA-3′ (forward), and 5′-CTCTACCATCCAGCACCCT-3′ (reverse), and primers with a restriction enzyme site comprising 5′-GAATTCGATGAGCGGGGGCACCCCTTA-3′ (forward with *Eco*RI site), and 5′-GGCGCGCCTTAGGGTCCAAAAGCCG-3′ (reverse with *Asc*I site). The fragment was linked to a T-vector (TAKARA), and digested with *Eco*R1 and *Asc*1 enzymes. The purified DNA fragment was then recombined into a PCS2+-myc plasmid digested with the same enzymes, and combined plasmids were transfected into Trans10 competent cells (TransGene, Beijing, China). Positive strains were selected to perform further experiments, and plasmids were extracted from bacteria using the TIANprep Mini Plasmid Kit (TIANGEN, Beijing, China), linearized with *Sal*I enzyme, and purified via the Gel and PCR Clean-Up System (Promega, Madison, WI, USA). Capped mRNAs were produced using the Sp6 mMessage mMACHINE Kit (Qiagen, Austin, TX, USA) and purified the RNeasy clean up kit (Qiagen). We used nuclease-free water to dilute mRNAs to a concentration of 2.0 mg/ml prior to injection. Injected oocytes were suppressed in M2 medium containing milrinone for 4 h, and then washed in milrinone-free M2 medium to resume meiosis.

Immunofluorescence (IF) labeling

Oocytes were first fixed in 4% paraformaldehyde for 30 min at room temperature, then permeabilized in phosphate-buffered saline (PBS) containing 0.5% Triton X-100 for 20 min. Next, oocytes were blocked in PBS with 1% BSA, either for 1 h at room temperature or overnight at 4°C, and then incubated with a primary antibody overnight at 4°C. The next day, after washing three times in washing buffer, oocytes were incubated with a secondary antibody at room temperature for 1 h, and DNA was stained with PI (Propidium Iodide). The stained oocytes were then mounted on slides. The antibodies we used included FITC-α-tubulin (F2618, Sigma, St. Louis, MO, USA), and Bub 3 (sc-28258, Santa Cruz, Dallas, TX, USA). The oocytes were observed under a laser-scanning confocal microscope (Zeiss LSM 780, Berlin, Germany).

Western blotting

To perform western blotting experiments, we collected, pooled, and boiled 150 oocytes for 5 min with 2 × loading buffer. Samples were subjected to 10% SDS-PAGE, and separated proteins were electrically transferred to polyvinylidene fluoride membranes. The membranes were then blocked in Tris Buffered Saline with Tween-20 (TBST)-buffer with 5% BSA at room temperature for more than 2 h, and incubated with a primary antibody overnight at 4°C. After three 10-min TBST washes, the membranes were incubated with a secondary antibody for 1 h at 37°C. Finally, the membranes were imaged using Super Signal West Pico Chemiluminescence Substrate Kit (Thermo, Rockford, IL, USA) , incubated with chemiluminescence substrate mix, Stable Peroxide Solution: Enhancer Solution as 4: 1, and were then photoed by ChemiDoc XRS+ (BioRAD, Hercules, CA, USA) system.

Chromosome spread staining

First, oocytes were transferred to acid Tyrode's solution (Sigma) to remove the zona pellucida; this was a quick reaction, and to avoid over-digestion, the whole process was monitored under a microscope. Oocytes without zona pellucidae were briefly recovered in M2 medium, and then transferred onto slides covered with fixing solution comprising 1% paraformaldehyde, 0.15% Triton X-100, and 3 mM dithiothreitol in distilled H_2O (pH 9.2). Slides were exposed to air to dry slowly, and then fixed oocytes were blocked with 1% BSA in PBS, either for several hours at room temperature or overnight at 4°C, before being incubated with the Bub3 (sc-28258, Santa Cruz) primary antibody overnight at 4°C. The slides were washed three times (briefly) with washing buffer, and then incubated with a secondary antibody for 2 h at room temperature. DNA was stained with Hoechst 33342 for 8 min. The slides were then mounted for immunofluorescence microscopy observation.

Data analysis

All experiments were repeated at least three times. Statistical analyses were performed using a Student's *t*-test, and data were shown as the mean ± SEM.
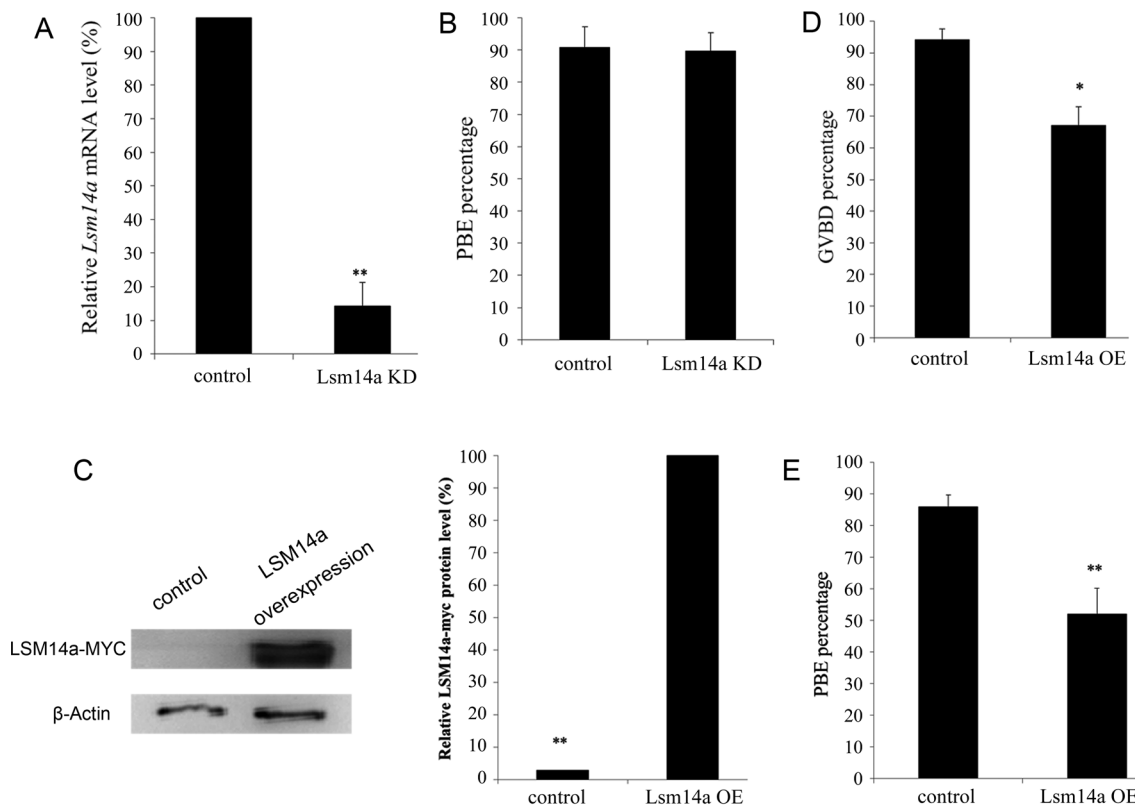
Fig. 1. Effects of *Lsm14a* knockdown (KD) and overexpression (OE) on oocyte meiotic maturation. (A) Oocytes were injected with *Lsm14a* siRNA (50 μM), incubated in M2 medium containing milrinone for 24 h, and then were washed with milrinone-free M2 medium to resume meiosis. Knockdown efficiency was measured via real-time PCR (RT-PCR). (B) The polar body extrusion (PBE) rate was observed after 14 h of maturation culture; both the RNAi group and control group contained over 200 oocytes. (C) The LSM14a-MYC expression level was analyzed by western blotting; oocytes were injected with *Lsm14a-myc* mRNA (2 mg/ml) and cultured for 4 h. (D) The GV oocytes were injected with *Lsm14a-myc* mRNA (2 mg/ml), incubated in M2 medium containing milrinone for 24 h, and then washed in milrinone-free M2 medium to resume meiosis. GVBD rates were analyzed after 3 h of culture. Each group contained over 150 oocytes. (E) The PBE rates of injected oocytes were analyzed after 14 h of culture.

Results

Lsm14a overexpression, but not Lsm14a knockdown, interrupts oocyte meiotic maturation

Using real-time PCR, we found that *Lsm14a* exhibited a high expression level in oocytes compared to *Ppia* (positive control); therefore, we first considered the effect of RNAi-silencing on *Lsm14a* mRNA expression. SiRNA (50 μM) or control ddH$_2$O was injected into the GV oocytes, and real-time PCR showed that $85.7 \pm 3.1\%$ of *Lsm14a* mRNA was depleted (P < 0.01, Fig. 1A). Oocytes injected with siRNA were incubated for 24 h in milrinone-containing medium, and the released oocytes exhibited normal maturation as indicated by the polar body extrusion (PBE) rate after culture (Fig. 1B).

Considering that LSM14a is an RNA-associated protein, and that its effect may either enhance or interfere with oocyte maturation, we next assessed the effects of *Lsm14a* overexpression in oocytes. *Lsm14a-myc* mRNA transcribed *in vitro* was injected into the GV oocytes at a concentration of 2 mg/ml, while control oocytes were instead injected with ddH$_2$O. Western blotting showed a high level of LSM14a-MYC protein expression (Fig. 1C). When *Lsm14a*

mRNA injected oocytes were incubated for 4 h and then released for maturation culture, they showed decreased germinal vesicle breakdown (GVBD) and maturation rates (Fig. 1D and E). The GVBD rate of oocytes with injected *Lsm14a* mRNA was $67.1 \pm 5.9\%$, while that of the control group was $94.2 \pm 3.4\%$ (P < 0.05). The PBE rates of the overexpressed and the control group were $52.1 \pm 8.1\%$ and $86.3 \pm 3.7\%$, respectively (P < 0.01). These data show that LSM14a protein may associate with the mRNAs of proteins that are essential for oocyte maturation, and form RNPs to store these mRNAs so that oocytes can control their maturation by regulating mRNA translation.

Lsm14b knockdown causes MI-stage arrest with either normal or abnormal chromosome alignment

In oocytes, there were two kinds of LSM14 protein. As described above, *Lsm14a* knockdown did not affect oocyte meiotic maturation, therefore we considered whether *Lsm14b* knockdown affected this process. First, we measured *Lsm14b* mRNA using real-time PCR, and found that like *Lsm14a* mRNA, it was highly expressed in the GV oocytes. After injecting different siRNAs, we decided to inject

a mix of two kinds of siRNAs at a concentration of 35 µM each. We collected 80 injection- and control-group oocytes to conduct real-time PCR, and found that 78.7 ± 5.3% of *Lsm14b* mRNA was depleted (P < 0.01) (Fig. 2A). RNA-interrupted oocytes went through the GVBD stage, but a considerable proportion of oocytes did not complete maturation; the percentage of RNAi oocyte maturation was 39.7 ± 5.1%, while that of the control ddH$_2$O-injected oocytes was 78.8 ± 3.6% (P < 0.05) (Fig. 2B). This phenotype indicated that, unlike LSM14a, LSM14b may regulate the oocyte meiotic maturation process.

IF staining of arrested RNAi oocytes after 14 h of culture showed that these oocytes were arrested at the MI stage, and that while the spindles of some of these metaphase-I arrested oocytes were well assembled, 30.9% (13/42) of oocytes showed abnormal spindles and disordered chromosomes (Fig. 2C). In contrast, control oocytes after 14 h of culture had already extruded their first polar bodies. In conclusion, we showed that LSM14b is essential for oocyte meiotic progression.

Lsm14b knockdown increases MPF and SAC activity as a result of Cdc20 mRNA non-degradation

From the above results, we concluded that *Lsm14b* mRNA-depleted oocytes had a lower PBE rate, and that oocytes that did not mature were blocked at the metaphase I (MI) stage. These predominantly exhibited typical MI spindles; however, some displayed an abnormal chromosome alignment, indicating that they had failed to undergo the metaphase-anaphase transition. For this transition process, maturation promoting factor (MPF) inactivation and spindle assembly checkpoint (SAC) silencing are the most important events, therefore we focused our attention on these two complexes.

Using Cyclin B1 as a marker to evaluate the MPF activity of oocytes in the metaphase-anaphase transition, we collected 150 each of RNAi and control oocytes after 9.5 h of culture [14]. Western blotting results revealed that control oocytes showed degraded Cyclin B1, while RNAi oocytes had some remaining Cyclin B1 (Fig. 3A). This suggested that the persistent MPF activity in the *Lsm14b*-depleted oocytes may have induced their failure to enter anaphase.

To analyze SAC activity in detail, we conducted chromosome-spreading staining with Bub3 as the SAC marker. We knew that after oocytes had started the anaphase transition, SAC on SAC centromeres would disaggregate so that homologous chromosomes could separate from each other. The chromosome-spreading staining showed that at 9.5 h of culture, Bub3 was no longer located on the centromeres of control oocytes, but was still observed on centromeres of *Lsm14b* RNAi oocytes arrested at the MI-stage after 14 h of culture (Fig. 3B). We then applied the chromosome-spreading staining to mature RNAi oocytes to analyze their euploidy. The result showed that 21.5% of mature RNAi oocytes were aneuploid compared to only 5.3% of control oocytes, and also identified some cytoplasmic equally-cleaved oocytes whose chromosomes were equally distributed (Fig. 3C and D). Together, these results show that *Lsm14b* knockdown causes oocyte MI-stage arrest, with abnormal spindle assembly and chromosome alignment as well as chromosome separation, by increasing MPF and SAC activity.

We next investigated the mechanism underlying LSM14b regulation of MPF activity. Considering that LSM14b is an mRNA-associated
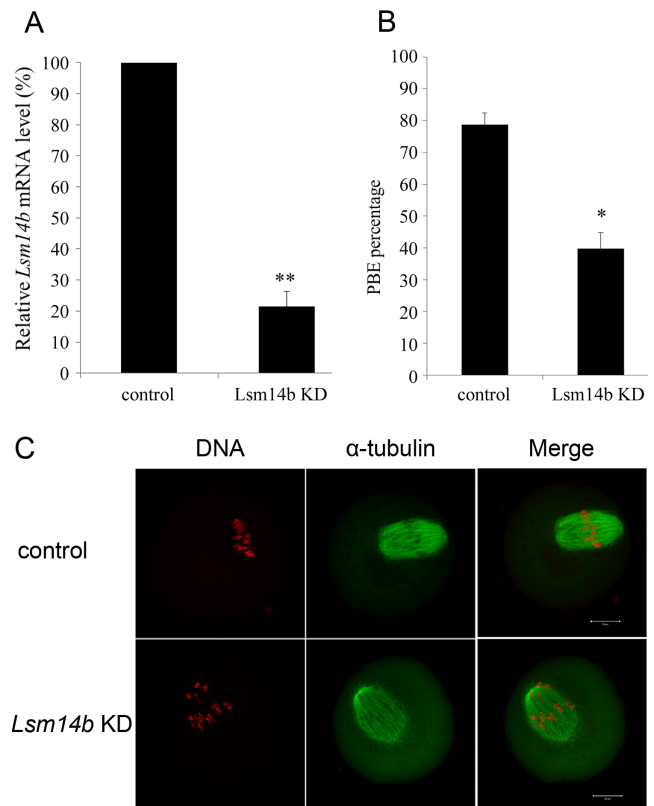
Fig. 2. *Lsm14b* knockdown causes oocyte arrest at the metaphase I stage. (A) Oocytes were injected with mixed *Lsm14b* siRNAs (70 µM), incubated in M2 medium containing milrinone for 24 h, and then washed with milrinone-free M2 medium to resume meiosis. Knockdown efficiency was measured via RT-PCR. (B) The polar body extrusion rate was observed after 14 h of maturation culture; both the RNAi and control group contained over 150 oocytes. (C) *Lsm14b* RNAi oocytes that were cultured for 14 h were fixed, and then used for confocal imaging to visualize spindles and chromosomes. Scale bar, 20 µm.

protein, we used real-time PCR to assess the mRNA levels of *Cyclin B1* (a structural subunit of MPF), and the APC/C regulatory protein CDC20. Oocytes injected with *Lsm14b* siRNA or ddH$_2$O were collected at the GV stage, after being arrested for 24 h in M2 medium containing milrinone. Unexpectedly, we found that following *Lsm14b* mRNA depletion, *Cyclin B1* and *Cdc20* mRNA contents were also reduced to the same level. The *Cyclin B1* mRNA level was reduced to 24.7 ± 4.3% compared to the control group (Fig. 3E), and the *Cdc20* mRNA level was reduced to 22.1 ± 5.1% compared to the control group (Fig. 3F). It is likely that *Cdc20* depletion caused APC/C inactivity, preventing Cyclin B1 from being degraded and thus the oocytes from completing the metaphase-anaphase transition. From our research data, we concluded that LSM14b associated and stocked mRNAs, including cell cycle-essential protein mRNAs such as *Cyclin B1* and *Cdc20*, and thus regulated the oocyte meiotic maturation process by controlling mRNA expression and degradation.
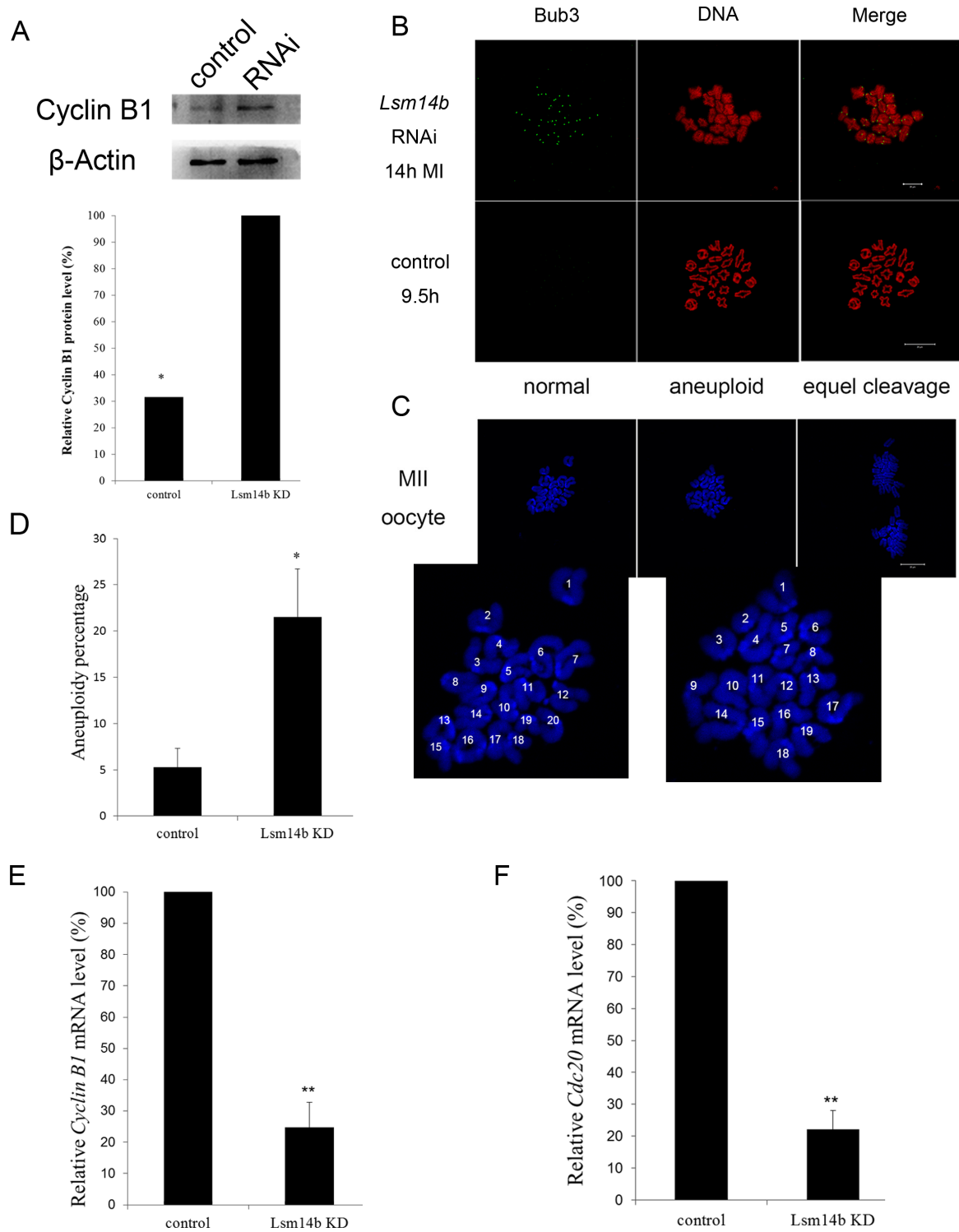
Fig. 3. Regulatory mechanism underlying the effect of LSM14b on oocyte meiotic maturation. (A) *Lsm14b* RNAi and control oocytes were collected after 9.5 h of culture, and analyzed via western blotting. The Cyclin B1 (CCNB) primary antibody was used to assay MPF activity, and β-Actin was used as the reference protein. (B) *Lsm14b* RNAi and control oocytes were subjected to chromosome spread staining after 9.5 h of culture. Bub3 (green fluorescence) was used as marker of SAC; the red crosses represent metaphase chromosomes stained by PI. (C) Mature oocytes were collected after 14 h of culture, and subjected to chromosome spread staining with Hoechst 33342 to analyze aneuploidy. (D) Aneuploidy rates were calculated from the chromosome spread staining. (E & F) Oocytes were injected with mixed *Lsm14b* siRNAs (70 µM), incubated in M2 medium containing milrinone for 24 h, and then washed with milrinone-free M2 medium to resume meiosis. *Cyclin B1* and *Cdc20* mRNA levels were measured via real-time PCR. Scale bar, 20 µm.

Discussion

Oocytes synthesize and accumulate maternal mRNAs in the ooplasm, and their time-dependent translation is important for oocyte meiotic maturation and early embryo development. In the present study, we showed that RNA-associated LSM14 family members may play important roles in regulating oocyte meiotic maturation. *Lsm14a* knockdown did not affect oocyte meiotic progression, but *Lsm14a* overexpression did cause reduced GVBD and maturation rates. The concentration of *Lsm14a* mRNA we injected into oocytes was high. We tried to inject lower concentrations of mRNAs, but the effect on the meiotic cell cycle was not evident. We thus believe that LSM14a did not specifically associate with mRNAs of cell cycle proteins, and that the phenotype may have instead been caused by the forced association of LSM14a with these mRNAs. This suggests that under physiological conditions, LSM14a is not a critical protein regulating oocyte meiotic maturation.

In contrast to *Lsm14a*, knockdown of *Lsm14b* caused significantly reduced polar body extrusion, and a high proportion of oocytes arrested at metaphase of first meiosis. Spindles were well constructed and chromosomes were aligned normally in some of the MI arrested oocytes, but in other cases, oocyte chromosomes were placed outside the metaphase plate, suggesting potentially abnormal microtubule and kinetochore linkage. A previously published study showed that during mitosis, LSM14 directly binds to tubulin, and is essential for spindle stability [15]. Chromosome-spreading staining showed that the SAC protein Bub3 was located on kinetochores even after a 14-h culture of *Lsm14b*-knockdown oocytes that had arrested at the MI stage. SAC is a checkpoint that cells use to ensure that spindles have properly assembled to make connections with chromosome kinetochores, before continuing cell division. The kinetochore localization of Bub3 indicated that SAC was still activated, and the chromosome kinetochores may exhibit linkage problems. Considering the function of LSM14b in regulating mRNA association, we interpret this to mean that some kinetochore proteins that help chromosomes to link were absent because of *Lsm14b* depletion. It would be interesting to determine which mRNAs are associated with LSM14b to control spindle microtubule association with chromosome kinetochores.

As we predicted, oocytes arrested at metaphase I exhibited incompletely degraded Cyclin B1, (degraded Cyclin B1 is a prerequisite for MPF inactivation). In our previous research, we discussed a similar phenotype where oocytes were also blocked in metaphase I and Cyclin B1 did not degrade, and reached the conclusion that MPF activity persisted due to APC/C^{cdc20} inactivation [14]. Therefore, in the present study, we wondered if LSM14b influenced APC/C function. APC/C is a complex consisting of a large catalytic APC subunit, with a regulatory CDH1 subunit during the GV phase, and a CDC20 subunit during anaphase. *Lsm14b* knockdown did not affect GVBD, and thus APC/C^{Cdh1} was functional. The MI-stage arrest after *Lsm14b* knockdown may have been caused by failed APC/C activation resulting from disturbed *Cdc20* expression or activation. Fortunately, we had confirmed that *Cdc20* mRNA was reduced to the same level as *Lsm14b* mRNA, and thus, it is very possible that LSM14b regulates oocyte maturation by controlling *Cdc20* expression to regulate APC/C activity. The reduction of *Cdc20* mRNA may be due to the deadenylation controlled by deadenylases

such as CNOT7, while dissociative mRNAs are more likely to be targeted and then degraded [16].

In the present study, we also analyzed *Cyclin B1* mRNA levels, and surprisingly found that they were also reduced to the same level as *Lsm14b* mRNA. Why did *Lsm14b*-depleted oocytes not show reduced GVBD compared to control oocytes? It is possible was that enough Cyclin B1 protein had already been synthesized and accumulated in the ooplasm to activate MPF as required for GVBD. Taken together, these results suggest that the LSM14 family, which functions by associating with mRNAs, is essential for oocyte meiotic maturation. LSM14b may associate with mRNAs encoding important cell cycle proteins such as *Cyclin B1* and *Cdc20*, to regulate meiotic cell cycle progression. It is now necessary to research the relationship between RAPs like LSM14 and mRNA expression, as this may be an important mechanism underlying mRNA post-transcriptional regulation.

Acknowledgements

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