Involvement of Aurora A Kinase during Meiosis I-II Transition in Xenopus Oocytes*

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The Aurora kinase family has been involved both in vivo and in vitro in the stability of the metaphase plate and chromosome segregation. However, to date only one member of this family, the protein kinase Aurora B, has been implicated in the regulation of meiotic division in Caenorhabditis elegans. In this species, disruption of Aurora B results in the failure of polar body extrusion. To investigate whether Aurora A is also required in meiosis, we microinjected highly specific α-Aurora A antibodies in Xenopus oocytes. We demonstrated that microinjected oocytes fail to extrude the first polar body and are arrested with condensed chromosomes on a typical metaphase I plate, which has not performed its normal 90° rotation. We additionally found that, although the failure of first polar body extrusion observed in α-Aurora A-microinjected oocytes is likely mediated by Eg5, the impairment of the metaphase plate rotation does not involve this kinesin-like protein. Surprisingly, although chromosomes remain condensed at a metaphase I stage in α-Aurora A-microinjected oocytes, the cytoplasmic cell cycle events progress normally through meiosis until metaphase II arrest. Moreover, these oocytes are able to undergo parthenogenetic activation. We conclude that Aurora A and Eg5 are involved in meiosis I to meiosis II transition in Xenopus oocytes.

From yeast to human, members of the Aurora/Ipl1 kinase family have been implicated in many mitotic cell cycle events ranging from centrosome separation and bipolar spindle assembly to chromosome segregation (1, 2). Thus, a direct interaction of the Aurora B kinase with the inner centromere protein (INCENP) has been reported as required for kinetochore disjunction and chromosome segregation during mitosis (2–4). The Aurora A kinase has also been involved in mitosis, because either RNA-mediated interference or mutational inactivation of the Drosophila Aurora A gene perturbs the formation of the embryonic bipolar mitotic spindle (5, 6). In Xenopus, the disorganization of a preformed mitotic metaphase plate in egg extracts can also be induced by adding either a specific antibody or a catalytically inactive mutant of the Aurora A kinase (7, 8). Additionally, the functional disruption of an Aurora A sub-

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‡‡ The abbreviations used are: GVBD, germinal vesicle breakdown; H1-kinase, histone H1-kinase; CHX, cycloheximide; CSF, cytostatic factor.© 2003 by The American Society for Biochemistry and Molecular Biology, Inc.
RESULTS

Microinjection of α-Aurora A Antibodies Does Not Inhibit Progesterone-induced Activation of Cdc2 Kinase—To assess the putative role of Aurora A in meiosis, we produced antibodies against recombinant Xenopus Aurora A. These antibodies specifically recognize and immunoprecipitate Xenopus Aurora A and do not cross-react with the other member of the Aurora A family, the Aurora B protein (Fig. 1, A and B; respectively; see also Ref. 4 and supplementary data in Ref. 18). Moreover, they do not inhibit Aurora A myelin basic protein kinase activity in vitro (data not shown). Indeed, previous reports have already shown mitotic spindle disorganization induced by treatment of Xenopus egg extracts with anti-Aurora A monoclonal antibodies that neither recognize the catalytic domain of the kinase nor inhibit its kinase activity (7). This suggests that such antibodies may exert their effect by perturbing the functional targeting of the kinase and/or the accessibility of its substrate.

We microinjected prophase-arrested Xenopus oocytes with either α-Aurora A antibodies (final concentration 13 ng/μl) or, as a control, the same amount of rabbit Ig. Then progesterone was added into the media to induce meiotic maturation. Both microinjected and control oocytes underwent germinal vesicle breakdown (GVBD) with similar kinetics (Fig. 2A). To determine the cell cycle status of the corresponding oocytes, we monitored the H1-kinase activity of Cdc2 at GVBD as a main landmark of the G2-M transition. No differences between control and treated oocytes were detected in the level of H1-kinase activity at the time of GVBD (Fig. 2B).

Hormone-stimulated Oocytes Microinjected with α-Aurora A Antibodies Are Arrested at Metaphase I—We then assessed the cytological status of microinjected α-Aurora A or control oocytes by Hoechst dye staining and direct observation under a UV epifluorescence microscope. As shown in Fig. 3, normal meiotic maturation in Xenopus oocytes is characterized by the formation of a first metaphase plate (20–60 min after GVBD), which is perpendicular to the surface of the oocyte, followed by rotation of the metaphase plate by 90° (60–80 min after GVBD). Finally, homologous chromosomes undergo anaphase (90–120 min after GVBD) leading to the extrusion of the first polar body and meiotic metaphase II arrest (180 min after GVBD). As shown in Fig. 2C (top left), control oocytes fixed 3 h after GVBD had reached second meiotic metaphase and arrested at this step. In contrast, oocytes microinjected with α-Aurora A antibodies (Fig. 2C, top right) were systematically arrested at metaphase I, with condensed chromosomes aligned on a metaphase plate oriented perpendicular to the oocyte surface. This arrested phenotype is in fact similar to the normal early metaphase plate before rotation of the first meiotic spindle in control oocytes at 1 h after GVBD (Fig. 2C, middle left, and Fig. 3; see Ref. 19). Moreover, no changes in α-Aurora A-microinjected oocytes were observed when they were fixed 10 h after GVBD (Fig. 2C, middle right), suggesting that chromosomes moved to form the first metaphase plate and did not proceed further in the first meiotic cell cycle. Arrest at the first meiotic metaphase before spindle rotation was a specific consequence of blocking some essential function of Aurora A protein, because microinjection of control antibodies had no such effect (Fig. 2C, bottom right). Moreover, no arrest at first meiotic metaphase was observed when oocytes were co-injected with an excess of recombinant Aurora A protein together with the native antibodies (Fig. 2C, bottom left). We know that under these conditions the recombinant Aurora A protein inhibits antigen-antibody binding because the Western blot signal corresponding to Aurora A kinase in interphase and metaphase II-arrested egg extracts dramatically decreased when these preabsorbed antibodies were used (Fig. 2D). The rotation of condensed chromosomes on the metaphase plate was never observed in α-Aurora A-microinjected oocytes arrested at the first meiotic cell cycle. However, suppression of chromosomes segregation does not appear to be associated necessarily with a failure of the metaphase plate rotation, because a very small proportion (see Table I) of injected oocytes escaped metaphase I arrest and progressed to first meiotic anaphase even though the metaphase plate failed to rotate (Fig. 4, A and B). Because these experiments were conducted on at least four different sets of oocytes, these latter results probably reflect a slight natural heterogeneity of the microinjected oocytes regarding
Fig. 2. a-Aurora A-microinjected oocytes readily underwent GVBD but arrested at metaphase I. A, oocytes were microinjected with either a-Aurora A antibodies or the same amount of rabbit Ig and then incubated in the presence of progesterone. The time of GVBD was established by scoring white-spot formation in the animal pole under a dissecting microscope. B, same oocytes as in A, but homogenates were prepared from one of these oocytes at 0 h (prophase) or at the time of GVBD, subjected to immunoprecipitation with anti-Aurora A antibodies, and analyzed by autoradiography for H1-kinase activity. C, maturing oocytes (left-hand panels, top and middle) and oocytes microinjected with anti-Aurora A antibodies (right-hand panels, top and middle), with anti-Aurora A antibodies preincubated with the recombinant Aurora A-His<sub>6</sub> protein (1 μg of antibody/5 μg of protein) (bottom left), and with control immunoglobulin (bottom right) were taken at the indicated times after GVBD. Subsequently, they were fixed, Hoechst-stained, and examined from the animal pole under an epifluorescence microscope. The arrows indicate the first polar body, and the scale bar represents 10 μm. D, 1 μl of interphase (INT) or metaphase II-arrested (CSF) Xenopus egg extracts were submitted to SDS-PAGE and Western blotting with either anti-Aurora A antibody (1/500 dilution; left panel) or the same serum previously preabsorbed using the recombinant Aurora A-His<sub>6</sub> cross-linked to CNBr-activated Sepharose (1/500 dilution; right panel).

These results indicate that Aurora A, likely through its substrate Eg5, controls metaphase I exit; however, they also indicate that the failure of metaphase I plate rotation in these oocytes is mediated mainly by Aurora A and does not involve the kinesin-like Eg5.

Microinjection of α-Eg5 Antibodies Induces Metaphase I Arrest in Maturing Xenopus Oocytes—The kinesin-like protein Eg5 is a well reported Aurora A substrate (9) in which homologues act as regulators of mitotic cytokinesis in different species. Disruption of Eg5 function in cell cultures by monastrol (10, 11) as well as Aurora A-antibody treatment of mitotic Xenopus egg extracts lead to a collapse of the mitotic metaphase plate (7). To investigate whether the role of Aurora A in meiosis is mediated by its substrate, Eg5, we tested for whether this kinesin-like protein is also required to develop complete meiotic division by microinjecting oocytes with highly specific α-Eg5 antibodies (see Fig. 3B) or with rabbit Ig in controls. Prophase oocytes microinjected with both rabbit Ig or α-Eg5 antibodies reached GVBD with the same kinetics when treated with progesterone (data not shown). However, only α-Eg5-microinjected oocytes failed to emit the first polar body (Table I and Fig. 4C) by the time controls had reached the second meiotic metaphase arrest (Fig. 4D), indicating that, like Aurora A, Eg5 is also required for the oocyte to resume meiosis. Moreover, although the first meiotic metaphase plate rotation was not impaired (Fig. 4C), the subsequent organization of condensed chromosomes was greatly perturbed, as observed in fixed oocytes 10 h after GVBD (Fig. 4, E and F). This disorganization of the condensed chromosomes resembles the previously described phenotype on the mitotic metaphase plate observed on cultured cells after monastrol treatment (10, 11).

The potential effect of microinjection. Additionally, this subset of oocytes that were less responsive to microinjection allow us to postulate that metaphase I plate rotation is not required for the metaphase-to-anaphase transition to take place.

Microinjection of α-Eg5 Antibodies Induces Metaphase I Arrest in Maturing Xenopus Oocytes—The kinesin-like protein Eg5 is a well reported Aurora A substrate (9) in which homologues act as regulators of mitotic cytokinesis in different species. Disruption of Eg5 function in cell cultures by monastrol (10, 11) as well as Aurora A-antibody treatment of mitotic Xenopus egg extracts leads to a collapse of the mitotic metaphase plate (7). To investigate whether the role of Aurora A in meiosis is mediated by its substrate, Eg5, we tested for whether this kinesin-like protein is also required to develop complete meiotic division by microinjecting oocytes with highly specific α-Eg5 antibodies (see Fig. 3B) or with rabbit Ig in controls. Prophase oocytes microinjected with both rabbit Ig or α-Eg5 antibodies reached GVBD with the same kinetics when treated with progesterone (data not shown). However, only α-Eg5-microinjected oocytes failed to emit the first polar body (Table I and Fig. 4C) by the time controls had reached the second meiotic metaphase arrest (Fig. 4D), indicating that, like Aurora A, Eg5 is also required for the oocyte to resume meiosis. Moreover, although the first meiotic metaphase plate rotation was not impaired (Fig. 4C), the subsequent organization of condensed chromosomes was greatly perturbed, as observed in fixed oocytes 10 h after GVBD (Fig. 4, E and F). This disorganization of the condensed chromosomes resembles the previously described phenotype on the mitotic metaphase plate observed on cultured cells after monastrol treatment (10, 11).

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a-Aurora A Antibodies Do Not Impair the Kinetics of Cyclin Degradation during Either Meiotic Maturation or Meiotic Metaphase II to Anaphase II Transition—During meiosis of Xenopus oocytes, a window of cyclin degradation occurs in the course of the metaphase I to metaphase II transition (20–22). This window of degradation is, however, naturally hidden by a dynamic cyclin neosynthesis. Following this window, a progressive cyclin stabilization step occurs, which culminates with a final high and permanent level of Cdc2 activity. This cyclin stabilization results directly from an accumulation of cytostatic factor activity (CSF) during meiosis and allows the installation of a permanent Cdc2 kinase activity (21, 22). Thus, because they contain CSF activity, vertebrate oocytes fail to degrade cyclin B and are arrested at the second meiotic metaphase until fertilization. It is well known that protein synthesis is not required after GVBD for completion of the first meiotic cell cycle in Xenopus oocytes. Thus, to facilitate detection of cyclin degradation, we used cycloheximide (CHX) to suppress protein synthesis during progesterone-induced meiotic maturation after GVBD. We then assessed whether the first metaphase I arrest observed in α-Aurora A-microinjected oocytes could be the result of a cyclin stabilization, leading to a possible cell cycle arrest, by monitoring both cyclin B2 degradation and Cdc2 activity during meiosis. Progesterone-treated oocytes, previously microinjected or not with α-Aurora A antibodies at...
the prophase stage, were transferred at the indicated times after GVBD for 1 h in a buffer containing CHX. Then they were homogenized to allow immunoblotting detection of cyclin B2 as well as H1-kinase assay on Cdc2 immunoprecipitates. As shown (Fig. 5A), cyclin B2 disappeared in control and α-Aurora A-microinjected oocytes by 30–60 min after GVBD. As a result of a nonreplacement of degraded cyclin due to the CHX, Cdc2 kinase activity remained low. Moreover, a high Cdc2 activity and no cyclin B2 degradation were detected in both α-Aurora A- and rabbit Ig-microinjected oocytes when CHX was added 100 min or more after GVBD, by the time that oocytes were already arrested at metaphase II. The normal degradation of cyclin B2 observed in α-Aurora A-microinjected oocytes indicates that the metaphase I arrest in those oocytes is not the result of a general block of the meiotic cell cycle. Moreover, despite the fact that the time course establishment of the CSF activity, as revealed by cyclin B2 accumulation and an increase in Cdc2 activity (Fig. 5A), remained unaffected in α-Aurora A-treated oocytes, the cytological condensed status of meiosis I-arrested chromosomes did not evolve during this period (Fig. 2C, middle right).

When Xenopus metaphase II-arrested oocytes are electrically stimulated or treated with ionophore A23187, two procedures that mimic fertilization, they resume anaphase II concomitantly with cyclin degradation (13). We then asked whether cyclin B2 degradation could be impaired in α-Aurora A-microinjected oocytes upon ionophore treatment. To test this possibility, prophase Xenopus oocytes were microinjected with either α-Aurora A or the same amount of rabbit Ig. Then progesterone was added to resume meiotic maturation, and by the time that control oocytes arrested at metaphase II, oocytes were activated with the ionophore. Time course degradation of the endogenous cyclin B2 was then monitored by immunoblot. As shown in Fig. 5B, both treated and control oocytes normally degraded cyclin B2. Our results indicate that, in α-Aurora A-microinjected oocytes, the failure to resume the first meiotic anaphase does not impair subsequent cyclin degradation upon parthenogenetic activation.

**DISCUSSION**

A growing number of reports indicate a requirement for Aurora A and B in mitosis. Aurora A kinase has been implicated in the maintenance of mitotic metaphase plate as well as in cytokinesis (1, 2). Moreover, the Aurora kinase family seems to be also involved in meiotic division because maternal Aurora B depletion in C. elegans results in the failure of polar body extrusion during first meiosis (12). We investigated here whether Aurora A, which is expressed at low levels at first meiotic prophase in Xenopus oocytes (8, 23, 24), plays a role in the meiotic cell cycle. We used microinjection of highly specific α-Aurora A antibodies to target Aurora A kinase in vivo. We show here that, upon microinjection, the ability of the recipient oocytes to resume meiotic maturation upon GVBD and to activate the Cdc2 kinase is not impaired. However, we were surprised to find that the same recipient oocytes failed to emit the first polar body and were arrested at meiosis I with condensed chromosomes. Moreover the condensed chromosomes remain aligned on a metaphase plate oriented perpendicular to the oocyte surface. Although spindle rotation was never observed in meiosis I-arrested oocytes, we showed, in some limited cases, however, a small proportion of α-Aurora A-microinjected oocytes that escaped metaphase I arrest and progressed to first meiotic anaphase. This subset of particularly less responsive oocytes probably indicates that metaphase I to anaphase I transition in Xenopus oocytes does not need a previous 90° rotation of the metaphase plate. On the basis of these results, we concluded that Aurora A kinase is involved in separation of homologous chromosomes and spindle rotation during meiosis. Because Aurora A is a protein kinase it makes sense to hypothesize that this protein exerts its role by phosphorylating one or several key substrates controlling meiosis resumption. Microinjection of α-Aurora A antibodies into Xenopus oocytes may inhibit phosphorylation of the Aurora A targets. However, we (data not shown) and others (7) have demonstrated that these antibodies have no effect on Aurora A kinase activity, indicating that their blocking activity is probably mediated by perturbation of functional targeting and/or substrate accessibility.

The kinesin-like Eg5 is a well reported substrate of Aurora A in Xenopus (9). Disruption of this protein in different cell lines leads to a disorganization of the mitotic metaphase plate (10, 11) similar to that observed when Aurora A is blocked (9). As described previously herein for α-Aurora A antibodies, α-Eg5-microinjected oocytes fail to emit the first polar body and are arrested with condensed chromosomes organized on a rotated metaphase plate. Thus, because α-Eg5-microinjected oocytes always succeed in the 90° metaphase I plate rotation, we conclude that Aurora A may be involved in two different mechanisms, the first one regulating metaphase I exit, which is likely mediated by Eg5, and the second one, independent of Eg5, which may control metaphase I plate rotation. In this regard, there is no data concerning the regulation of spindle rotation by this kinesin-like protein either in Xenopus oocytes or other cell types. Unlike Eg5, the role of the dynein-dynactin complex in spindle rotation during embryogenesis and cell division has...
Aurora A and Meiosis in Xenopus Oocytes

TABLE I

α-Aurora A microinjection arrests Xenopus oocytes at meiosis I

| Phenotype                          | α-Aurora | α-Aurora + antigen | Rabbit Ig | α-Eg5 |
|------------------------------------|----------|--------------------|-----------|-------|
| Arrest at metaphase of meiosis I   | 49       | 3                  | 0         | 43    |
| Arrest at anaphase of meiosis I    | 2        | 0                  | 0         | 0     |
| Arrest at metaphase of meiosis II  | 3        | 28                 | 39        | 0     |
| Total scored oocytes               | 54       | 31                 | 39        | 43    |

Fig. 4. First meiotic anaphase in the absence of spindle rotation and requirement of the kinesin-like Eg5 for metaphase I exit. A and B, oocytes injected with α-Aurora A antibodies were fixed 10 h after GVBD and examined from the animal pole as in Fig. 2. Pictures of the same oocyte were taken at two different focal planes (A and B) to allow detection of separate sets of chromosomes. C-F, oocytes injected with either α-Eg5 antibodies (C, E, F) or the same amount of rabbit Ig as a control (D) were taken 3 (C, D) or 10 h (E, F; two different oocytes) after GVBD and examined by epifluorescence as described in the legend for Fig. 2. The scale bar represents 10 μm.

been reported largely in several species, for example in C. elegans (23) or Saccharomyces cerevisiae (24, 25) or in mammalian cells (26). The current model proposes that astral microtubules interact with cortical actin and dynactin through one dynactin subunit, the actin-capping protein. Then, tethered by dynactin, dynein interacts with the plus-end of microtubules and pulls toward the anterior cortex by minus-end-directed motility causing spindle rotation (23). According to this hypothesis, dynactin is localized to the cortical microtubules capture site, and both dynactin and dynein are required for spindle rotation (27). Several evidences indicate that the dynein-dynactin-dependent mechanism of spindle orientation is regulated by phosphorylation of both complexes. Thus, phosphorylation of p150(Glued), a subunit of the dynactin complex, mediates dynamic binding to microtubules (28). Moreover, dynein-dynactin interaction is also regulated by phosphorylation of the dynein intermediate chain (29). Spindle rotation in Xenopus oocytes at metaphase I may also be mediated by a dynactin-dynein mechanism. Accordingly, cortical F-actin is required for spindle rotation because rotation of metaphase I spindle is completely inhibited upon cytochalasin B treatment (30). Moreover, two components of the dynein-dynein complex undergo coordinated phosphorylation during oocyte maturation (17). In this work we found that inhibition of Aurora A kinase blocks metaphase I plate rotation and metaphase I exit.

We propose that the first phenomenon may be mediated by phosphorylation of one or several constituents of the dynactin-dynein complex, as for example the p150(Glued) protein or the intermediate chain of dynein.

In an effort to characterize the second mechanism controlling meiosis I exit, we investigated whether the metaphase I arrest observed in α-Aurora A-microinjected oocytes was the result of a general block of the cell cycle. We found that normal meiotic cytoplasmic events, such as cyclin B2 degradation, proceeded normally. Thus, we conclude that the arrest at first meiotic metaphase observed in α-Aurora A-microinjected oocytes is not correlated with a general block of the cell cycle.

Meiotic metaphase II-arrested oocytes of vertebrates are believed to exit naturally this stage when cyclin degradation is triggered by fertilization or parthenogenetic activation (13). Our results indicate that oocytes with a metaphase I-arrested phenotype normally degrade cyclin B2 upon parthenogenetic activation. However, despite the fact that our data show a requirement of Aurora A during first meiosis in Xenopus oocytes, we could not assess whether this kinase is also involved in the second meiotic anaphase because of the early phenotype observed. We conclude that the disruption of Aurora A in vivo does not imply a block of the normal meiotic cytoplasmic cell cycle events. Moreover, our data reveal, for the first time, a
requirement of the Aurora A kinase and Eg5 in the meiosis of Xenopus oocytes. We ask now how Aurora A impinges on the segregation of homologous chromosomes at first meiosis in Xenopus. Thus, the Aurora kinases seem to be key factors not only in controlling mitotic events but also in the regulation of meiotic division.

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