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to spindle-checkpoint inactivation. We found that nocodazole, a microtubule-depolymerizing agent, effectively induced the G2 arrest in chicken DT40 cells that stably express a temperature-sensitive form of the kinase-dead allele of Mps1 (mps1-T68A) mutant. In contrast, cells expressing the mps1-T68A mutant did not arrest in G2 after short-term exposure to nocodazole at non-permissive temperature, although the control cells eventually arrest in G2 after long-term exposure. This suggests that the sensitivity of the spindle checkpoint depends on the duration of exposure of cells to nocodazole. To investigate the signaling pathways downstream of Mps1, we found that nocodazole induced the degradation of Bub1 and Mad1 as well as the activation of Chk1 and Chk2 in DT40 cells. These results suggest that both Bub1 and Mad1 are essential for the maintenance of G2 arrest and the activation of both Chk1 and Chk2 is necessary for induction of checkpoint arrest in response to impaired mitosis.Q: Как получить положение рукава в прил 6d1f23a050

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