The Impact of Deuterium Oxide on the Cell Cycle of RBL Cell during Interphase via Tubulin-mediated Antimitotic Action

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I previously investigated the effects of deuterium oxide on cell growth. However, I could not discover what factors attributed to a delay in the cell growth of rat basophilic leukemia (RBL-2H3) cells. I investigated the cell cycle and vesicle transport to determine whether or not these components had contributed to the delayed cell growth. The diffusion coefficient alone could not explain the declination observed. My first experiment, conducted with the flowcytometer, displayed a reduced number of deuterium oxide cells found in the G2 (Interphase) cell cycle of the cells. The G2 phase is responsible for the preparation of mitosis (cell division). When I performed single particle tracking (using a TIRF microscope), a slower velocity was shown from the vesicles within the deuterium oxide cells. When colchicine was added to the cells, a significant decrease in speed (more than 75%) was observed from vesicles within normal water cells. Despite colchicine's microtubule-destabilizing abilities, the vesicles of the deuterium oxide cells remained at the same velocity. These experiments allow me to conclude that deuterium oxide causes cells to halt in growth during their G2 phase. This is due to the slower velocity of the kinesin motors which are responsible for the transportation of vesicles. Additionally, deuterium oxide's microtubule-stabilizing capabilities were able to counteract the destabilizing aspects of colchicine when it was added to the cells. I have analyzed possible interpretations of these results and the importance for future studies on possible applications regarding deuterium oxide's microtubule-stabilizing abilities.

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