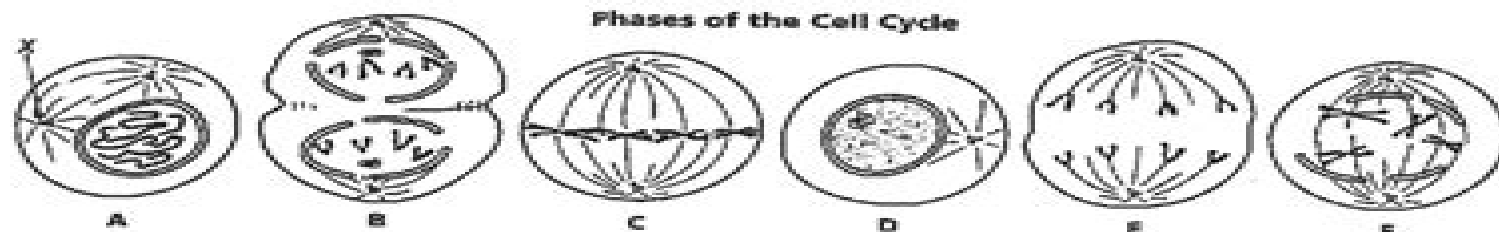


Mitosis Worksheet

The diagram below shows six cells in various phases of the cell cycle. Note the cells are not arranged in the order in which mitosis occurs and one of the phases of mitosis occurs twice. Use the diagram to answer questions 1-7.



- 1) Cells A and F show an early and a late stage of the same phase of mitosis. What phase is it?
A - early prophase, F - late prophase
- 2) Which cell is in metaphase?
C
- 3) Which cell is in the first phase of mitosis?
A (if consider prophase 1st) but D (if consider interphase 1st)
- 4) In cell A, what structure is labeled X?
Centrioles
- 5) Which cell is in the "in between" phase of mitosis?
D (interphase, where the cell is being the cell it was created to be!)
- 6) Place the diagrams in order from first to last.
A, F, C, E, B, D (some may put D first. Remember this is a cycle so how the order is viewed is subjective)
- 7) Are the cells depicted plant or animal cells? Explain your answer.
Animal - round in shape
- 8) What is the longest phase of the cell cycle?
D
- 9) Why is mitosis important?
Allows cells to reproduce, making exact copies of themselves

Answer Sheet For Cell Division By Mitosis

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Answer Sheet For Cell Division By Mitosis:

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Blended learning has gained significant attention recently by educational leaders practitioners and researchers i Flex a variation of blended learning is based on the premise that certain non interactive teaching activities such as lecturing can take place by students without teachers direct involvement Classroom time can then be used for educational activities that fully exploit teacher student and student student interactions allowing for meaningful personalized feedback and scaffolding on demand Revolutionizing K 12 Blended Learning through the i Flex Classroom Model presents a well rounded discussion on the i Flex model highlighting methods for K 12 course design delivery and evaluation in addition to teacher performance assessment in a blended i Flex environment Emphasizing new methods for improving the classroom and learning experience in addition to preparing students for higher education and careers this publication is an essential reference source for pre service and in service teachers researchers administrators and educational technology developers **Botany (Paper 1) Cytogenetics, Plant**

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Dikic, 2018-02-09 DNA damage response DDR is a term that includes a variety of highly sophisticated mechanisms that cells have evolved in safeguarding the genome from the deleterious consequences of DNA damage It is estimated that every single cell receives tens of thousands of DNA lesions per day Failure of DDR to properly respond to DNA damage leads to stem cell dysfunction accelerated ageing various degenerative diseases or cancer The sole function of DDR is to recognize diverse DNA lesions signal their presence activate cell cycle arrest and finally recruit specific DNA repair proteins to fix the DNA damage and thus prevent genomic instability DDR is composed of hundreds of spatiotemporally regulated and interconnected proteins which are able to promptly respond to various DNA lesions So it is not surprising that mutations in genes encoding

various DDR proteins cause embryonic lethality malignancies neurodegenerative diseases and premature ageing The importance of DDR for cell survival and genome stability is unquestionable but how the sophisticated network of hundreds of different DDR proteins is spatiotemporally coordinated is far from being understood In the last ten years ubiquitin ubiquitination and the ubiquitin relative SUMO sumoylation have emerged as essential posttranslational modifications that regulate DDR Beside a plethora of ubiquitin and sumo E1 activating enzymes E2 conjugating enzymes E3 ligases and ubiquitin sumo proteases involved in ubiquitination and sumoylation the complexity of ubiquitin and sumo systems is additionally increased by the fact that both ubiquitin and sumo can form a variety of different chains on substrates which govern the substrate fate such as its interaction with other proteins changing its enzymatic activity or promoting substrate degradation The importance of ubiquitin SUMO systems in the orchestration of DDR is best illustrated in patients with mutations in E3 ubiquitin ligases BRCA1 or RNF168 BRCA1 is essential for proper function of DDR and its mutations lead to triple negative breast and ovarian cancers RNF168 is an E3 ubiquitin ligase which creates the ubiquitin docking platform for recruitment of different DNA damage signalling and repair proteins at sites of DNA lesion and its mutations cause RIDDLE syndrome characterized by radiosensitivity immunodeficiency and learning disability In addition recently discovered the ubiquitin receptor protein SPRTN is part of the DNA replication machinery and its mutations cause early onset hepatocellular carcinoma and premature ageing in humans Despite more than 700 different enzymes directly involved in ubiquitination and sumoylation processes only few of them are known to play a role in DDR Therefore we feel that the role of ubiquitin and the ubiquitin related SUMO in DDR is far from being understood and that this is the emerging field that will hugely expand in the next decade due to the rapid development of a new generation of technologies which will allow us a more robust and precise analyses of human genome transcriptome and proteome In this Research Topic we provide a comprehensive overview of our current understanding of ubiquitin and SUMO pathways in all aspects of DDR from DNA replication to different DNA repair pathways and demonstrate how alterations in these pathways cause genomic instability that is linked to degenerative diseases cancer and pathological ageing

Mathematical Modelling of the Cell Cycle Stress Response Elahe Radmaneshfar, 2013-10-08

The cell cycle is a sequence of biochemical events that are controlled by complex but robust molecular machinery This enables cells to achieve accurate self reproduction under a broad range of conditions Environmental changes are transmitted by molecular signaling networks which coordinate their actions with the cell cycle This work presents the first description of two complementary computational models describing the influence of osmotic stress on the entire cell cycle of *S cerevisiae* Our models condense a vast amount of experimental evidence on the interaction of the cell cycle network components with the osmotic stress pathway Importantly it is only by considering the entire cell cycle that we are able to make a series of novel predictions which emerge from the coupling between the molecular components of different cell cycle phases The model based predictions are supported by experiments in *S cerevisiae* and

moreover have recently been observed in other eukaryotes Furthermore our models reveal the mechanisms that emerge as a result of the interaction between the cell cycle and stress response networks

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