Dna Structure And Replication Answers

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there has been a sea change in how we view genetic recombination when germ cells are produced in higher organisms genetic recombination assures the proper segregation of like chromosomes in the course of that process called meiosis recombination not only assures segregation of one chromosome of each type to progeny germ cells but also further shuffles the genetic deck contributing to the unique inheritance of individuals in a nutshell that is the classical view of recombination we have also known for many years that in bacteria recombination plays a role in horizontal gene transfer and in replication itself the latter by establishing some of the replication forks that are the structural scaffolds for copying

dna in recent years however we have become increasingly aware that replication which normally starts without any help from recombination is a vulnerable process that frequently leads to broken dna the enzymes of recombination play a vital role in the repair of those breaks the recombination enzymes can function via several different pathways that mediate the repair of breaks as well as restoration of replication forks that are stalled by other kinds of damage to dna thus to the classical view of recombination as an engine of inheritance we must add the view of recombination as a vital housekeeping function that repairs breaks suffered in the course of replication we have also known for many years that genomic instability including mutations chromosomal rearrangements and aneuploidy is a hallmark of cancer cells although genomic instability has many contributing causes including faulty replication there are many indications that recombination faulty or not contributes to genome instability and cancer as well the nas colloquium links between recombination and replication vital roles of recombination was convened to broaden awareness of this evolving area of research papers generated by this colloquium are published here to encourage the desired interactions of specialists we invited some contributions that deal only with recombination or replication in addition to contributions on the central thesis of functional links between recombination and replication to aid the nonspecialist and specialist alike we open the set of papers with a historical overview by michael cox and we close the set with a commentary on the meeting and the field by andrei kuzminov

genotoxic carcinogens can lead to dna mutations with the potential to cause cancer typically a series of mutation events are needed before malignancy occurs so a single small exposure may not result in disease also cells have an armoury of defence mechanisms which to a degree counter the effects of mutagens distinguishing the point at which exposure to a carcinogen increases mutation rates beyond the background level is challenging in fact there is now general agreement that for genotoxic carcinogens no specific threshold can be identified however noaels no observed adverse effect levels may be used in the process of establishing a dose response relationship these denote the level of exposure at which there is no significant increase in adverse effects in the exposed population when compared to an appropriate control such a scientifically defendable threshold allows us to propose health based exposure limits for genotoxic carcinogens this book describes the various cellular defence mechanisms individually and explains how they are regulated the processes covered include metabolic inactivation epigenetic regulation scavenging mechanisms dna repair and apoptosis it also considers dose dependent threshold mechanisms of carcinogenesis and the rate limiting parameters aimed at graduate level and above the book discusses the consequences of genotoxic evaluation and urges readers to question the idea that even low exposures present a cancer risk

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 ubiqutin and sumo e1 activating enzymes e2 conjugating enzymes e3 ligases and ubiquitin sumo proteases involved in ubiquitination and sumoylation the complexity of ubiqutin 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

dna damage is a major threat to genomic integrity and cell survival it can arise both spontaneously and in response to exogenous agents dna damage can attack most parts of the dna structure ranging from minor and major chemical modifications to single strand breaks ssbs and gaps to full double strand breaks dsbs if dna injuries are mis repaired or unrepaired they may ultimately result in mutations or wider scale genome aberrations that threaten cell homeostasis consequently the cells elicit an elaborate signalling network known as dna damage response ddr to detect and repair these cytotoxic lesions this research topic was aimed at comprehensive investigations of basic and novel mechanisms that underlie the dna damage response in eukaryotes

discrepant results are the most common results of mental health research and may even occur multiple times in a single study but what do discrepant results mean how are researchers to make sense of them do they tell us something important about mental health and if they do how would we know discrepant results in mental health research challenges all that we think we know about mental health

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