[Name of the Writer]

[Name of Instructor]

[Subject]

[Date]

Prevalence of Obesity in USA

Introduction

With the advent of the science and technology, many things that could have been considered inconceivable or impossible few years ago are now done quite easily. DNA repair is one of them. It is a process through which is the cell can identify and correct the damage that has happened to DNA with the passage of time and how the encoding that is being done in the DNA can be repaired with the passage of time (Wood et al, 2001). The process was needed since with the environmental damage as well as the metabolic activities that are carried out in the region, the basic structure of the DNA is being damaged. Most of the times, it is the structural damage that is caused to the DNA is the most serious one and there was an effort for a very long time to make sure that the degree of control is achieved in this regard. Therefore the DNA repair process was needed to make sure that the normal wear and the other damages to the DNA structure can be repaired.

Discussion

Most o the times, when the DNA repair process is activated, it works in the manner that how the activities are carried out in the system that respond to the damage that is happening in the DNA structure at the given point of time. It must be noted that when the normal repair process fails, and the apoptosis of the cell is not occurred, then the DNA repair process is most active. The most damaging of them all is the double strand break as well as the DNA cross linkages that are carried out at the point of time (Wood et al, 2001).

Rate of the DNA repair

There are many factors that are involved in the whole process that tend to make sure that the process of the DNA repair is being sped up or halted. For instance, the cell that has accumulated the large amount of DNA damage or the one that is not able to repair or function in the normal manner goes through three states which are as followed.

* An irreversible state of the dormancy which is also called as the senescence
* The cell suicide, in which the programmed cell death occurs
* Unregulated cell division, which is the most damaging of them all as it is likely to cause the formation of the tumor that is bound to be cancerous.

It must be noted that the ability of the DNA to repair itself is quite important when it comes to the integrity of the genome and thus this process has to be carried out all the cost. The functionality of many organisms that are prevailing in the body. There were many genes that were initially shown to have an influence on the overall life span of the entities were the ones that were involved in the DNA repair system (Wood et al, 2001).

Mechanism of the DNA repair

It has to be noted that the cells cannot function with themselves if the DNA damage is such that they are corrupted and the integrity and the accessibility of the information genome is missing during the course of the whole process. It must be noted that even during this process, the cell remains to the functional at the superficial level specially when there is a case that the non essential gene is damaged or missing during the whole process. The whole thing is also dependent on the type of damage that is inflicted on the DNA’s double helical structure. To combat these, there are variety of strategies are being and the core premises of these strategies is to make sure that the information does not have to be lost during the whole process. The other possibility that is needed to be kept in mind is that how the cells are going to be using the unmodified complementary strand of the DNA or even the sister chromatid. The idea behind it is to make sure that the template is being created that allows the recovery of the initial information that was stored in the DNA. If there is no access to the template, the cells are then going to be using the trans lesion synthesis to make sure that the whole thing is being done as a matter of the last resort of all the other available options of the DNA repair are not working out (Wood et al, 2001).

Direct Reversal

It has been known that how cells are supposed to be making sure that they eliminate three types of the damages that is happening to the DNA and the way it is being done is that the chemical process is being used as a rationale to reverse the whole process. Most of the times, these mechanisms are not requiring a template due to the fact that the type of the template that they are supposed to be counteracting is such that it is likely to occur in only three to four basis at the particular point of time. The other thing that must be noted about these direct reversal mechanisms is that they are quite specific in terms of the way they work and how the type of damage that is incurred during the whole process is going to be avoided at the particular point of time. The formation of the pyrimidine dimers also plays an important role during the whole process (Wood et al, 2001). One key aspect of this process is that how the photo reactivation is going to be carried out. Most of the times what happens is that the direct reversal of the damage is needed to be done in order to make sure that the enzyme that comprises of the photolyase process is carried out. It is other matter that how its activation is going to be done and how the dependency on the enzyme is also supposed to be carried out based on the energy constraints at the particular point of time.

Single Strand Damage

If there is a case that only one or two strands are damaged during the process of the DNA repair, then the double helix effect must be there and the underlying defect at that point of time has to be resolved in an appropriate manner (Farmer et al, 2005). What happens most of the times in such processes is that the other strand can be used as a template to make sure that there is guidance in terms of the way correction of the underlying issues is carried out. Not only that, in order to ensure that the corresponding damage to the cell has been repaired, the effort is needed to be made to ensure that at least one of the two paired molecules of the DNA and its composition must be appropriate. The next thing that can be done is to make sure that the removal of the damaged nucleotide must be done and then it has to be replaced with DNA strand that is not damaged (Farmer et al, 2005). While large numbers of epigenetic alterations are found in cancers, the epigenetic alterations in DNA repair genes, causing reduced expression of DNA repair proteins, appear to be particularly important. Such alterations are thought to occur early in progression to cancer and to be a likely cause of the genetic instability characteristic of cancers.

Double Strand Breaks

If there is a case of the double strand break, the both the strands are being repaired with the help of the double helix. The idea behind the whole process is to make sure that the hazardous cells are covered as there is a likelihood that they can lead towards the rearrangement of the genome. It has been noted in some of the studies that how the double strand break that is witnessed in the given issue acts as a cross linkage that is joining both the strands and how the whole point is going to be irreparable. This is happening since how none of the strand can be used as a template for the repair. If that is the likely scenario, then what happens most of the times is that the cell is going to die in the next mitosis. There are some rare instances that even the mutation of the cells is being carried out (Farmer et al, 2005). The other thing that must be noted about the whole process is that how the damage that occurs in these cases is intermediate in nature. Classically, cancer has been viewed as a set of diseases that are driven by progressive genetic abnormalities that include mutations in tumour-suppressor genes and oncogenes, and chromosomal aberrations. However, it has become apparent that cancer is also driven by epigenetic alterations.

Trans lesion Synthesis

Trans lesion synthesis which is also known as the TLS is one of the major ways through which the whole process of the DNA repair is being carried out. The idea is to make sure that the DNA’s must be developing damage tolerance. This damage tolerance is going to allow them to make sure that the DNA replication mechanism ca be placed so that the thymine dimmers and the functionality of the AP sites works in the right manner. Not only that, the whole process work out in the manner that is about making sure that the regular DNA polymerases is going to be done and the specialized trans lesion about the polymerases is carried out at the particular point of time (Farmer et al, 2005). The other purpose of the whole exercise is to make sure that there should be facilitation of the insertion of the opposite damaged nucleotides during the course of the whole process. What happens next is that the polymerase are going to be mediated along with the other factors (Farmer et al, 2005). The post translational modification of the replication and the processivity factor of the PCNA is a major factor that is needed to be kept in mind during the whole process. What it does is that when the lesion is encountered, then the replication fork is going to be stalled. What happens next is that the PCNA would be making the switch from the processive polymerase to the TLS polymerase so that the lesion can be fixed (Farmer et al, 2005). The PCNA is then going to make sure that the extension in the given case is being managed in the right manner. The last thing that is going to be done during the whole process is that the switch to the processive polymerase is going to be made to make sure that the process of the replication is continued in the given point of time.

Global Response to the DNA Damage

The important factor that must be kept in mind during the course of the whole process is to make sure that the ionization of the radiation and the usage of the ultraviolet lights is going to be used (Cleaver, 1968). There are instances where even the usage of the chemical is being talked about as a major process to control the damage that is happening to the cells at that point of time. The next thing is that how the global response to the damage is going to be working out and how the management of the multiple pathways. It also includes the repair of the lesion bypass as well as the development of the tolerance level and apoptosis during the whole process (Cleaver, 1968). DNA damage response mechanisms trigger cell-cycle arrest, and attempt to repair DNA lesions or promote cell death/senescence if repair is not possible. Replication stress is observed in preneoplastic cells due to increased proliferation signals from oncogenic mutations. Replication stress is characterized by: increased replication initiation/origin firing; increased transcription and collisions of transcription-replication complexes; nucleotide deficiency; increase in reactive oxygen species (ROS).

DNA repair Mechanism and Cancer

At the moment, there are some inherent limitations to the process as far a the way DNA repair mechanism is supposed to work. If the humans are living long enough, at times, the process of the repair work is augmented and thus it is bound to cause cancer to the person. There are about 34 inherited gene mutations that are likely to increase the risk of the cancer. What happens is that many these mutations work in the manner that it causes the DNA repair. Specially if one talks about the hereditary non polyposis colorectal cancer, then it is strongly associated with the mutations that are rather specific in nature and thus cause the DNA mismatch repair pathway to be broken at the particular point of time. Then there are cancer therapy procedures such as the chemotherapy and radiotherapy that might function in the manner that the they can overwhelm the capacity of the DNA repair process to be faulted and thus resulting in the death of the cell. Not only that, the cells that are rapidly dividing, which are mostly cancer cells are going to be affected preferably due to that. The important thing that has to be noted in this regard is that how the side effect in the given case is going to work in the manner that the rapid division of the cells act as a progenitor that are placed in the gut, skin and some of the other hematopoietic systems of the body that are affected in this manner. The good thing is that there are many drugs that are sorting out the residual DNA repair mechanism and the likelihood is on the higher side that the how the commonly found things and cancer causing processes are going to be controlled (Cleaver, 1968). The prevalence of DNA damage response mutations differs across cancer types; for example, 30% of breast invasive carcinomas have mutations in genes involved in homologous recombination.

Conclusion

There are replication errors and the DNA damage that is going on in the cell all the time as well as our bodies. The idea is to make sure that how the element of control is needed to be developed to make sure that these mutations can be controlled. Most of the times they are detected and the DNA itself works in the manner that the proofing is carried out and the structure fixed itself. If that is not happening, then cells would go the programmed death which is known as the process of the apoptosis (Cleaver, 1968).

# Works Cited

Cleaver, J. E. "Defective repair replication of DNA in xeroderma igmentosum." *nature* 218.5142 (1968): 652.

Farmer, Hannah, et al. "Targeting the DNA repair defect in BRCA mutant cells as a therapeutic strategy." *Nature*434.7035 (2005): 917.

Wood, Richard D., et al. "Human DNA repair genes." *Science*291.5507 (2001): 1284-1289.