

*Review Article*

## **IMPORTANCE OF NUTRIENTS IN DNA REPAIRING PROCESS**

**R. Yasothai**

Veterinary University Training and Research Centre,  
Tamilnadu Veterinary and Animal Sciences University,  
Erode – 638004, Tamilnadu, India

Deoxyribonucleic acid (DNA) is a nucleic acid that contains the genetic instructions used in the development and functioning of all known living organisms. A main role of DNA molecules is the storage of information. Chemically, DNA is a long polymer of simple units called nucleotides with a backbone made of sugars and phosphate groups joined by ester bonds. Attached to each sugar is one of four types of molecules called bases. It is the sequence of these four bases along the backbone of DNA that encodes information. Three nucleotides (nucleic acids) in DNA encode for three nucleotides in ribonucleic acid (RNA), which encode one protein molecule when the DNA is read (translated) from RNA to protein in the ribosome.

DNA furnishes a template or blueprint for the production of RNA that is translated into proteins. DNA is often compared with a set of blueprints, since it contains the instructions needed to construct other components of cells such as proteins and RNA molecules. The DNA segments that carry this genetic information are called genes, but other DNA sequences have structural purposes or are involved in regulating the use of this genetic information.

The information contained in DNA is read using the genetic code, which specifies the sequence of the amino acids within proteins. The code is read by copying stretches of DNA into the related nucleic acid RNA in a process called transcription. Most of these RNA molecules are used to synthesize proteins, but others are used directly in structures such as ribosomes and spliceosomes.

There are two kinds of nucleic acids: DNA and RNA. DNA is found in the chromosomes of a cell's nucleus and it carries hereditary information. RNA is located in the cell, but not in the nucleus. Just as proteins consist of long chains of amino acids, DNA and RNA consist of nucleic acid chains called nucleotides.

Nucleotides are composed of three units: base, sugar (monosaccharide) and phosphate. Bases are found in both DNA and RNA. As seen below they are adenine, cytosine, guanine,

*Received July 27, 2016 \* Published Oct 2, 2016 \* [www.ijset.net](http://www.ijset.net)*

thymine and uracil. They are abbreviated (A, C, G, T, U). Three of the bases (A, G, C) are found in both DNA and RNA. However, uracil (U) is found only in RNA and thymine (T) is found only in DNA.

Within cells, DNA is organized into structures called chromosomes. These chromosomes are duplicated before cells divide in a process called DNA replication. Eukaryotic organisms such as animals, plants and fungi store their DNA inside the cell nucleus, while it is found in the cell's cytoplasm in prokaryotes such as bacteria. Within the chromosomes, chromatin proteins such as histones compact and organize DNA, which helps control its interactions with other proteins and thereby control which genes are transcribed.

If DNA repair does not take place, defective DNA is present in the cell and this leads to defective RNA and the translation of defective RNA into non-functional or defective proteins in the ribosome.

DNA has the remarkable ability to proof read itself and to correct many errors before they become permanent. However the nutrient deficiencies can interfere with both the synthesis and repair of DNA.

Folate is an essential water-soluble vitamin occurring naturally in select foods as well as in the synthetic form (folic acid) used in supplements and in food fortification programs. There are many critical cellular pathways dependent on folate as a 1-carbon source including DNA, RNA, and protein methylation as well as DNA synthesis and maintenance. Folate can be a limiting factor in all these reactions

For example low intake of folic acid interferes with the production of thymine, one of DNA's four bases. When cells cannot make thymine they replace it in DNA with uracil. But when DNA repair enzymes scan the DNA, it removes the uracil and leaves breaks in the DNA.

The breaks in the single strand of DNA can usually be repaired. However folic acid deficiency leads to large number of uracil deposits in DNA. So many deposits that they dramatically increase the risk of double strand DNA breaks. Double strand breaks are not as easily repaired and are more likely to result in permanent damage to DNA.

Some research findings say that in folate deficient people increased folate intake may decrease the risk of many types of cancer.

Schantz (1997) found that smoking cigarettes generate large numbers of free radicals, which increased the number of DNA mutations. People who relatively take few antioxidant – rich foods. (fruits and vegetables) and those with poor DNA repair mechanisms were far

more likely to develop head and neck cancers. In contrast people who consumed large amounts of antioxidants particularly vit C, vit E and lycopene were more resistant to DNA damage and less likely to develop head and neck cancers.

### **Genetic variation, dietary cholesterol and plasma cholesterol level:**

It has been known for at least 20 years that the response of plasma cholesterol concentration to cholesterol feeding is heterogeneous, although the mechanisms only recently are being understood.

In certain situation the response to diet appears to be determined by the genetic variant of apolipoprotein as for example, is the case with apolipoprotein E (Apo E). On a low fat / high cholesterol diet individuals with Apo E4/4 phenotype respond with an increase in serum cholesterol, whereas those with Apo E2/2, Apo E3/2 do not show an increase. On a Low-fat/low cholesterol diet all variants show a decrease in serum cholesterol.

Apo E4 allele is associated with hyper cholesterolemia. Where as the Apo E2 protects against high cholesterol level. However in the presence of the obesity, hypothyroidism and diabetes the variant form of Apo E2 is associated with the development of type III hyper lipoproteinemia and accumulation of chylomicron and VLDL remnants in plasma. Only one person in 50 with the Apo E2 variant develops hypertriglyceridemia. Because the triglyceride removal is genetically determined, increase in energy intake, trans fatty acid intake or carbohydrate intake (particularly in women) leads to hyper triglyceridemia.

Additional studies show that women of the Apo E3/2 phenotype stand to benefit the least from high polyunsaturate. Because of reduction in the more protective high-density lipoprotein (HDL) cholesterol, where as men of the Apo E4/3 phenotype showed the greatest improvement in the LDL and HDL ratio. Therefore a general recommendation to increase the polyunsaturated content of the diet to decrease plasma cholesterol level and the risk for coronary artery disease is not appropriate for women with the Apo E3/2 phenotype.

### **References**

- [1] Bailey, L.B., 2003. Folate, methyl-related nutrients, alcohol, and the MTHFR 677C3T polymorphism affect cancer risk: intake recommendations. *J.Nutr.*, 133: 3748S–3753S.
- [2] Boland, M.P., P. Lonergan and D.O' Callaghan, 2001. Effect of nutrition on endocrine parameters, ovarian physiology and oocyte and embryo development. *Theriogenology*, 55: 1323–1340.

- [3] Dwyer, J.H., H. Allayee and K.M. Dwyer, 2004. Arachidonate 5-lipoxygenase promoter genotype, dietary arachidonic acid and atherosclerosis. *New England J. Medicine*, 350: 29-37.
- [4] Eitenmiller, R., L.W. Folate, 1999. In: Rea W L, editor. *Vitamin Analysis for the Health and Food Science*. Boca Raton: CRC Press. p. 411–465.
- [5] Kelly, T.K, D.D. De Carvalho and P.A. Jones, 2010. Epigenetic modifications as therapeutic targets. *Nat Biotechnol.*, **28**:1069–1078.
- [6] O'Broin JD, I.J. Temperley, J.P. Brown and J.M. Scott, 1975. Nutritional stability of various naturally occurring monoglutamate derivatives of folic acid. *Am J Clin. Nutr.*, **28**:438–444.
- [7] Schantz, S.P., Z.F. Zhang and M.S. Spitz, 1997. Genetic susceptibility to head and neck cancer: interaction between nutrition and mutagen sensitivity. *Laryngoscope*, 107: 765-781.
- [8] Schwerin, M., U. Dorroch, M. Beyer, H. Swalve, C.C. Metges, and P. Junhans, 2002. Dietary protein modifies hepatic gene nutrient interactions. *Annual review of Nutr.*, 25: 499-522.
- [9] Yang, X, F. Lay, H. Han and P.A. Jones, 2010. Targeting DNA methylation for epigenetic therapy. *Trends Pharmacol. Sci.*, **31**:536–546.