Factors That Influence The Process Of Ageing

Ageing is defined as the progressive loss of function accompanied by decreasing fertility and increasing mortality with advancing age (Kirkwood, et al., 2000). A gene, as described by The Editors of Encyclopaedia Britannica (2019) is a unit of hereditary information and thus, is continuous; passed down from generation to generation. There have been various discussions on the effect of genes on ageing. Whilst some believe there's no effect, it has been proven by recent scientific findings that ageing is under (some) genetic control. However, ageing is also said to be affected by environmental factors such as exercise, diet, sleep and stress. Another belief is that epigenetics also plays a part in how organisms age.

It's now a common belief that genetics has a role in how an organism ages due to newfound evidence introducing genes that can affect longevity. It is important to note that, different species have different lifespans (Kirkwood, 2008) and so the genes that affect ageing differs among organisms. Firstly, it has been noted in vertebrate species that insulin, and the insulin-like growth factor pathway have a crucial role in controlling longevity (Van Heemst, 2010). This fact is portrayed by a popular example of the roundworm, Caenorhabditis elegans, mutations decreasing the function of daf-2 (which encodes for IGF-1) more than doubles the lifespan of the roundworm (Kenyon, 2010). Additionally, the gene FOXO3, encoding the transcription factor forkhead box O-3 (FOXO3), is one which genetic polymorphisms has exhibited consistent associations with longevity in diverse human populations (Morris, et al., 2015). In invertebrates such as the roundworm, daf-16, activates genes that extend longevity by promoting resistance to oxidative stress (Carter, et al., 2007). Even though the research for the effect in humans is still ongoing, it can be compared to the study of mice, due to their close genetic and physiological similarities. Mice that are deficient for either the insulin receptor, or the insulin-like growth factor receptor-1 can live up to 30% longer than wild-type mice, suggesting that FOXO factors could be involved in mammalian longevity (Carter, et al., 2007). FOXO factors a range of cellular responses, including resistance to oxidative stress, a phenotype highly coupled with lifespan extension (Greer, et al., 2007).

Despite evidence that ageing is influenced by genes, it is also widely believed that social and physical environmental conditions and non-genetic factors also play a vital role in the development of an organism, and the progress at which the organism ages. As stated by Karol (2009), the aging process is largely under genetic control but is highly responsive to diverse environmental influences. The environmental factors believed to make an impact on ageing include lifestyle, such as smoking in humans, physical activity and nutrition. For example, a poor diet containing excess sugar and saturated fats contributes directly to the burden of damage with which cells must deal, whereas a Mediterranean?style diet may contribute protective factors such as dietary antioxidants (Kirkwood, 2008). A Mediterranean-style diet might directly modulate the insulin/IGF-1 and mTOR pathways, which are known to be linked to ageing and longevity (as mentioned above), due to the low content of animal protein and low glycaemic index (Vasto, et al., 2014). Another experiment that displayed that environmental factors were of influence was an experiment with Drosophila and dietary restrictions, demonstrating that dietary restriction produces a rapid decrease in the mortality rate, and so leading to the result that restrictions in the diet counteract the causes of ageing (Mair, et al., 2003).

Another phenomenon linked to ageing is epigenetics. Epigenetics is generally known as the study of changes in gene function that's heritable and doesn't cause a change in DNA sequence (Wu, et al., 2001 cited in Dupont, et al., 2009). It is said that telomere shortening is attributed to ageing in humans. Telomeres continuously cut during the meiotic cycle and once they reach a certain shortened length they senesce and stop growing (Aubert, et al., 2008). Telomeric DNA is lost in humans through many pathways such as end replication problems, nucleolytic processing of 5? template strands following DNA replication to create a 3? single strand overhang, and failed repair of oxidative DNA damage to telomeric DNA (Verfaille, et al., 2002). This issue is significant because loss of telomeric DNA leads reduces the amount of telomere function within the body, and it has been discovered that loss of telomere function brings consequences both for aging and carcinogenesis (Stewart, et al., 2006 cited in Aubert, et al., 2008). Nevertheless, telomere length related to ageing cannot be proven for mice, yeast, plant and roundworms as with the loss of telomeric DNA, they can withstand several generations (Aubert, et al., 2008). Whilst, telomere length is attributed to ageing, it can only be accounted for in humans.

The idea that ageing is purely caused by genes or outside factors can also be disputed by the fact that there may be stochastic occurrences that can arise randomly and have no heritable predisposition. One example is somatic gene mutation but other errors on an epigenetic, telomeric, proteomic or even cellular level are also possible (Steves, et al., 2012). An example is that in which genetically identical marbled crayfish raised in identical environmental conditions still differ in development (Vogt, et al., 2008 cited in Steves, et al., 2012). Thus, indicating that even in genetically identical twins, the ageing process may differ in each due to the stochastic occurrences that can be enhanced by one's environment, as well as the somatic mutations that can arise due to damage to cells.

Many theories have been put forward by scientists suggesting that ageing is multifaceted (though genetics seems to be the biggest influencer) such as the wear and tear theory which present the idea that cells and tissues have vital parts that wear out resulting in ageing (Kunlin, 2010).

To conclude, ageing is highly genetically controlled, however according to recent discoveries is also dependable on external and environmental factors such as antagonistic pleiotropy, stochastic occurrences and epigenetic alterations. Therefore, ageing is genetic but to an extent. It is a complex multifactorial process, not regulated or caused by a single universal basic mechanism (Von Hahn, 2003), but is as a result of various factors acting simultaneously and each factor essentially falls into one of the two following categories: programmed or damage-related/error factors (Jin, 2010).