With rate of the loss in nerve cells.

Technology, Development

With research, Scientists have found out that a certain drug; created for type two diabetes, helps with memory loss within Alzheimer's patients. The drug is currently being tested on mice, at the Lancaster University in the UK and is being fully evaluated for further tests, because alzheimer's has not had a drug or any kind of new knowledge for 15 years now. Alzheimer's is the most common of diseases that deal with dementia, and the numbers are continuing to rise, with two million people expected to have the disease within the UK alone, come 2051. The drg is only being used on mice currently, but researchers have found something within the drugs and the activity of the mice, that they would like to continue to advance the drug in hopes it will soon be for treating alzheimer's. The drug is a triple receptor drug that is not used often if any; the drug acts towards protecting the brain from degeneration and contains GLP-1, GIP, and Glucagon which are all used for growth.

The growth factor within the brain of alzheimer's patients have problems regenerating, and signals are not passed through as in patients without the disease. The study used mice that have human mutated genes that cause alzheimer's, these genes are found in people who have a form of alzheimer's that can be inherited. The APP/PS1 mice were aged transgenic and in the advanced stages of neurodegeneration, when treated. Results found, included enhanced levels of brain growth which protects nerve cell functioning, reducing the amount of amyloid plaques in the brain linked with alzheimer's, reduced chronic inflammation and oxidative stress, and slowed down the rate of the loss in nerve cells. Studies show that, the older version of the drug still has promising results; so the outcome, if of course they

improve on the drug, should be effective to a more advanced level than before.

While the search for new drugs for alzheimer’s patients have been going on, a new study on technology is emerging. Thus, being a brain imaging research will allow scientists to key on a certain protein that is killing off the brain cells, and the objective is to stop the spreading before it grows abroad, this may actually prevent the disease from forming in the first place. With about an estimated 44 Million people affected by the disease today, scientist have been working with alzheimer’s and its complexities, as it continues to grow and affect many people not only within the Country but worldwide. The disease is caused when two proteins grow inside the brain and begin to kill off brain cells as time goes on. The disease will cause memory loss, changes in a person’s behaviour or loss of independence; the two proteins, amyloid beta, and tau.

It is said that the amyloid phase occurs first and later influences the tau to form. The tau on the other hand appears throughout the brain and as the disease grows, so does the tau. This is known as transneuronal spread, which is tested on mice through injections, and have confirmed the protein does spread; more rapidly in mice, compared to humans which is the complete opposite and will not spread as fast. Just about a couple years ago the only way of studying the spread and growth of these proteins is through a study of people who have had the disease but have passed away. Today, scientists have a positron emission tomography (PET) scanning, which requires an injection of a radioactive ligand; a tracer molecule that will bind with the tau,

then will be able to detect it through the scanner. Recent studies have shown that there are more genes involved with Alzheimer's with leading functional and structural changes in the brain and within the proteins in cerebrospinal fluid(CSF).

This study focused on individual groups in specific regions across the cognitive spectrum system, and identified several novel genetic associations within subgroups. The researchers say the genes may be more attractive targets for drug development, the effective drugs will be given to patients before or shortly after they have developed the cognitive impairment. The researchers tested the comparisons between alzheimer's related brain MRI measures, memory test scores and CSF levels of the two proteins, amyloid-beta and tau; with millions of genetic markers (SNPs) across the genome of 1, 189 patients of the ADNI(Alzheimer's Disease Neuroimaging Initiative). They then examine the SNPs and genes using several datasets that show the gene expression that affects areas of the brain. Two main genes were identified in the normal functioning group, SRRM4 and MTUS; these genes are involved in neuronal signaling, development, and loss.

One gene, GRIN2B encoded a subunit that has a role in resilience of neurons and memory. A school in Sweden, the Karolinska has provided new evidence that higher education attainment is strongly linked with a lower risk of developing the disease; yet the cause of alzheimer's are unknown and the trials for treatment have failed, reducing the disease can be helped by targeting modifiable factors. Another method used, Mendelian randomisation used the genetic information and indicates interference between potential

risk factors with disease. If the gene has a specific impact on a risk factor, then this indication means they are associated with each other. Using the Mendelian method, a professor tested to see if education and different lifestyles link with the risk factors and disease. The analysis included more than 900 generic variants shown as risk factors, these variants were then compared among 17, 000 patients with Alzheimer's, and 37, 000 healthy controls revealed association for genetic variants that predict education.

The results, the strongest evidence provided this far on higher education will lower the risk of alzheimer's. A professor has discovered that boosting our cell's mitochondria defense system against a particular form of protein, may not only prevent growth of amyloid plaques, but also protect themselves from toxins. Through the aging process, our cells tend to breakdown and get weaker, this makes it very difficult for the protection of the mitochondria. Without the mitochondria, our brain cells will not be provided with the energy needed, and therefore are easier targets for disease.

While experimenting, the professor was able to identify the two main mechanisms that control mitochondria basically. Mitochondrial unfolded protein response(UPRmt) which protects the mitochondria, and the Mitophagy, which will recycle defective mitochondria; these both will be the focus points on delaying or preventing the damage in the future. From evidence is the past, it was known that the mitochondria is dysfunctional during alzheimer's, they did not know that it will defend itself with help. The help? Boosting control pathways, pharmacologically activate them.

The team tested antibiotic doxycycline and the vitamin nicotinamide riboside(NR), which can turn on the UPRmt and mitophagy defense systems. When treated with animals with Alzheimer’s the plaque formation was greatly reduced, and most importantly the scientist observed similar results when tested on human’s with the disease.