"Obesity our biggest challenge

When teaching students about the Liver and liver disease I usually share my belief that the liver it is the most important organ of the body and wax lyrical about the large number of functions it performs whilst regularly comparing the liver to a factory that produces, distributes, stores and removes waste.

When I entered nursing the main issues affecting the liver appeared to involve alcohol, drugs, cancer and infection however during my career there have also been numerous letters from the alphabet   added to hepatitis. Now whilst these issues continue to be problematic I believe that the greatest threat to the liver today is obesity. I say this because obesity rates have continued to rise over the last decade and issues related to this public health problem include cirrhosis and non-alcoholic steato hepatitis (Suraweera et al 2017).

It is it is estimated that approximately 1.9 billion people are obese worldwide (World Health Organisation 2015). This is a significant health problem that in turn leads to a wide range of health issues such as type two diabetes, cardiac disease and liver disease. In the UK it is estimated that approximately 30,000 people die every year in the UK as a result of being obese (James 2008).

When we specifically look at obesity and liver disease it is evident that non-alcoholic fatty liver disease is one of the leading indications for transplantation (Koppe 2014). It is also the most common chronic liver disease in children (Braumbough and Friedman 2014).

However, all is not as dire as it would initially seem as even a small weight loss of approximately 5-10% will reduce the health risks associated with obesity (Tsigos *et al.,* 2008). This is supported by Jenson et al (2013) who point out that this small reduction in weight will in fact improve blood pressure, liver function, glycemic control, cholesterol levels and may even reduce chronic pain. It has also been shown that for every 1 kg of weight lost the risk of developing diabetes is reduced by 16% (Hamman et al 2006).

Therefore, it is imperative that we tackle this problem in any way we can. The tools in our armoury include diet modification, increased exercise and bariatric surgery and whilst these are very useful approaches they do not suit everyone, Therefore newer ways to tackle this problem are required and unless we can come up with a viable solution my fear is that we will not tackle this significant health problem effectively.

 My rationale for the above statement is a follows; Whilst bariatric surgery has been found to reduce the mortality of patients with morbid obesity (Adams *et al.,* 2007) there are problems and complications associated with this. This is because the majority of patients undergoing surgery are morbidly obese and therefore this increases the risk during surgery as well as the development of postoperative complications (Flum *et al.,* 2005).

Diet modification is another approach and whilst there is no shortage of advice or diets available no one in particular has been shown to be better than the others. It is also a sad fact that many people who lose weight whilst on a diet usually put it back on within a year. It has also been found that the adherence to energy restriction diets is usually unsuccessful (Sumithran et al 2011).

Therefore it is clear that we need to try something different or a combination of approaches when dealing with obesity. However, Friedmann (2017;p4732) argues that there are alternative explanations to the commonly held belief that that obesity is “simply a lack of willpower that can be treated by advising patients to eat less and exercise more”. We now know that genetics plays apart and it is the identification of mutant genes in animal studies would suggest that mutant genes in humans may cause obesity.

When reviewing the numerous diets available one such diet which has proved to be somewhat effective at helping people lose weight is the high protein diet. It has been postulated that this works because it causes the body to expend more energy expenditure and because it leads to sustained satiety as well as increasing sensitivity to circulating leptin within the central nervous system (Westerterp-Plantenga et al 2009, Weigle 2005 ). It has been suggested that high protein diets cause the body to release appetite suppressing hormones (Larnkjær et al 2015). The hormone Leptin which is produced by adipocytes decreases feelings of hunger. It does this by alerting the brain that the body’s current energy store level is good which in turn leads to a suppression of food intake and increased energy expenditure (Bates & Myers, 2003). Leptin promotes glucose metabolism (Kusakabe *et al.,* 2009) and it also plays a role in regulating the metabolism of fat and carbohydrates in skeletal muscle (Ceddia *et al.,* 2001). Leptins effect on skeletal muscle also helps maintain energy stores and body weight. It therefore, maintains homeostatic control of adipose tissue mass (Friedman 2016). It has also been found that mutations in either leptin or the leptin receptors increase food intake and a corresponding reduction in energy expenditure (Friedman & Halaas, 1998). The discovery of Leptin in 1994 and its role in appetite suppression offered the opportunity to banish obesity to the annals of history. This was further supported when it was found that a reduction of leptin can lead to the development of obesity which may in in some cases reversible with leptin therapy (Ghouse et al 2016).

Yet 23 years since the discovery of leptin the obesity problem continues and this “magic bullet” has not provided the solution we hoped. However, as Farr (2015) points out **leptin** administration has proven ineffective for inducing weight loss on its own. However when used with something else the effects may be more significant. We also know now that leptin resistance also plays a part in obesity and whilst it may not be the main cause of obesity it does develop as a consequence of it (Parton *et al.,* 2007). It may also explain why lepin on its own has not been effective. Therefore, is this the end of the dream for a wonder drug? I would like to think not because whilst lepin on its own has not worked scientists are currently exploring issues around leptin resistance and this exciting work offers further insight into the development of obesity and the possibility of a "magic bullet" to treat this significant problem in the future. Further exciting developments is the use of Botulism toxin (BTX-A) and from early results it would suggest that within a month of administration there is significant weight loss that continues over a six month period. It has also been found to be a safe treatment, and well tolerated by patients (Albani et al 2005).

To conclude is evidently clear that whilst early studies have shown promise in the treatment of obesity we are still a long way off developing a “miracle cure” and therefore nurses and other health care professionals need to continue promoting the need to reduce sugar, fats from our diets as well as encouraging as much exercise as possible especially in our children if we wish to tackle this public health issue effectively.

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