ARTP SLEEP

S-NEWS

Dreaming of a better night's sleep

In this issue:

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Welcome

Welcome to this now wintery edition of S-News. Apologies for the lateness of getting this “Autumn” edition ready. I hope it will have been worth the wait. This issue has a bit of an obesity theme and the second article was written, re-written and shortened many times (contributing to the delay in me pulling the whole newsletter together). I could have written so much more and have been fascinated in what I have learnt in writing it. I would whole heartedly encourage further reading from the reference list and beyond. The information not only has importance for those with weight issues but for the health of the world as a whole. Don’t forget if you have anything you would like including in the next edition to send your news, articles and pictures to S-News@artp.org.uk. Enjoy!

SLEEP PEOPLE

Professor John Stradling

This issue’s sleep person is Prof John Stradling. John was born in London and trained at the Middlesex hospital. He gained a 1st class honours Bachelors of Science in physiology in 1973. From the outset, John excelled in his studies, gaining 1st year overall prize, 1st physiology and 1st biochemistry prizes in his second year and 1st BSc and 1st clinical year prizes in his 3rd year. In 1976 he gained his MB BS (with honours), followed by his MRCP in 1978. In 1981 John completed his MD and in 1984 undertook an MRC travelling research fellow post at the University of Toronto. One of his first publications related to sleep apnoea was in the Lancet in 1980, entitled “Apnoea alarms. Description of a new design.” In 1985, John returned to the UK and took up the position of Wellcome Senior Research Fellow and Consultant Physician at the University of Oxford. Since 1989 John has worked as a Consultant Respiratory Physician at the Churchill Chest Unit and was the Director of the Respiratory Sleep Service. In 1999 he was awarded a personal chair at the University of Oxford. John has been involved at a high level in numerous societies, including co-chair of the Sleep and Breathing Disorders working group, SEPCR, honorary secretary of the Breathing Club, Chair of the British Sleep Society and Vice Chair of ARTP-SLEEP. He is an international lecturer of great repute and has published widely on many aspects of Obstructive Sleep Apnoea with over 260 publications to his name. He has been an invited speaker and chair at many national and international meetings. He has been involved in developing many of the national guidelines and pathways including providing clinical expertise and interpretation of evidence used in the National Institute of Health and Clinical Excellence Health Technology Appraisal of the use of CPAP, the 18 week wait pathway for sleep and the RCP map of medicine project. John has always been a strong supporter of the British Society of Dental Sleep Medicine and the role that GDPs have in screening for Obstructive Sleep Apnoea. He was the lead author of the 'Snoring and the Role of the GDP: British Society of Dental Sleep Medicine pre treatment screening protocol' which was published in the British Dental Journal in 2009.

Dates for your diary

Obstructive Sleep Apnea in Dentistry
http://heauni.ee
7-8th January 2015, Tallin, Estonia

ARTP Annual Conference 2015
http://www.artp.org.uk/en/meetings/artp-annual-conference-2015/index.cfm
22nd-23rd January 2015, Blackpool, UK

London Sleep Medicine Training Course
http://www.mahealthcareevents.co.uk/cgi-bin/go.pl/conferences/detail.html?conference_uid=467
12-13th March 2015, London, UK

Edinburgh Sleep Medicine Course
http://www.ed.ac.uk/schools-departments/clinical-sciences/sleep-research-unit/courses/sleep-medicine
16-20th March, Edinburgh, UK

6th World Congress on Sleep Medicine
http://wasmcongress.com
21st-25th March, 2015, Seoul, Korea

Sleep and Breathing
http://www.sleepandbreathing.org
16-18th April 2015, Barcelona, Spain

Send your articles to S-NEWS@artp.org.uk

John has and has been supervisor for 14 PhD/MDs as well as clinical supervisor for many other clinicians and researchers. He has been awarded in excess of 50 research grants and has been integral to increasing the understanding of OSA, its consequences and its treatment. John has been sub-editor and peer reviewer for a number of highly renowned journals including Journal of Sleep Research, Clinical Science, Thorax, American Journal of Respiratory and Critical Care Medicine and European Journal of Respiratory Sleep Medicine to name a few.

Over his career John has won many awards including Hospital Doctor of the Year in 2002, BTS Silver Jubilee Award in 2007, the Ludwig Engel Visiting Professor, University of Sydney, 2009 and ARTP award for services to Respiratory Medicine in 2010. In 2012 he won the American Academy of Sleep Medicine annual William C. Dement Academic Achievement award for members of the sleep field who have displayed exceptional initiative and progress in areas of academic research. This year has also been a successful one with the ARTP Lyn Davies award, an Honorary Doctorate from Grenoble University (Honoris Causa) and the BTS Annual Medal for services to Respiratory Medicine and Research. John has now allegedly retired, however he is still very active in both in a research and speaking capacity.

SLEEP IN THE NEWS

Don't worry, be happy: Just go to bed earlier

http://www.springer.com/gp/about-springer/media/springer-select/don-t-worry--be-happy--just-go-to-bed-earlier/41926

Researchers at Binghampton University in the US have linked late evenings to repetitive negative thoughts. The researchers stated "When you go to bed, and how long you sleep at a time, might actually make it difficult for you to stop worrying." They asked 100 young adults at Binghamton University to complete a battery of questionnaires and two computerised tasks. In the process, it was measured how much the students worry, ruminate or obsess about something – three measures by which repetitive negative thinking is gauged. The students were also asked whether they were more habitual morning or evening types, preferring to hold regular hours or to have a sleep-wake schedule that is more skewed towards later in the day.They found that people who sleep for shorter periods of time and go to bed very late at night are often overwhelmed with more negative thoughts than those who keep more regular sleeping hours. The findings have been published in the journal Cognitive Therapy and Research.

Obesity, Diabetes and Bariatric surgery:

by Debbie Smith, RNS, Oxford

Obesity & diabetes cost the NHS £billions a year. *NICE draft guidelines out for consultation, has recommended lowering the threshold of eligibility for weight loss surgery, and obese people with type 2 diabetes should be offered weight loss (bariatric) surgery. If obesity levels continue to rise at their current rates, it is estimated that by 2050 the annual cost of treating obesity-related complications will be £50 billion, more than half the current NHS budget for England.

The expansion of bariatric surgery is controversial, but NICE says the move is supported by the latest evidence that surgery can reduce the effects, or even reverse, type 2 diabetes. In

particular, NICE advises that those with recent-onset type 2 diabetes who fulfil certain body mass index (BMI) criteria should have surgery.

Currently, NHS funded bariatric surgery is offered to people with a BMI of 40 or more, or those with a BMI between 35 and 40 if they have another significant and possibly life-threatening disease that could be improved if they lost weight, such as type 2 diabetes or high blood pressure. Patients must have tried and failed to achieve clinically beneficial weight loss by all other appropriate non-surgical methods and be fit for surgery.

The updated draft guidelines include additional recommendations on bariatric surgery for people with recent-onset type 2 diabetes, which include:

- Offering an assessment for bariatric surgery for people who have recent-onset type 2 diabetes and are also obese (BMI of 35 and over)
- Considering an assessment for bariatric surgery for people who have recent-onset type 2 diabetes and have a BMI between 30 and 34.9. People of Asian origin will be considered for surgery if they have a lower BMI than this as the point at which the level of body fat becomes a health risk varies between ethnic groups. Asian people are known to be particularly vulnerable to complications of diabetes.

What is Bariatric Surgery?

Bariatric surgery consists of a range of techniques used (gastric band, bypass, sleeve gastrectomy and duodenal switch) but all usually based on the principle of surgically altering the digestive system so it takes less food and makes the patient feel fuller quicker after eating.

The two most common types, usually performed using keyhole surgery are:

- Gastric banding - a band is used to reduce the size of the stomach so a smaller amount of food is required to make someone feel full.
- Gastric bypass – where the digestive system is rerouted past most of the stomach so less food is digested, which makes the person feel full.

What are the risks:

As with all types of surgery, weight loss surgery carries a risk of complications, which include:

- internal bleeding
- a blood clot inside the leg (DVT)
- a blood clot or other blockage inside the lungs (PE)

It is estimated the risk of dying shortly after gastric band surgery is around 1 in 2,000, and a gastric bypass carries a higher risk of around 1 in 100. Other side effects include excess skin (removal not normally available on NHS), gallstones, gastric band slippage and psychosocial effects.

Main concerns:

There is concern about how many people will be eligible for treatment under the new guidelines and how much it will cost, with Diabetes UK estimating that 850,000 people could be eligible for surgery, but NICE expects the figure to be nearer 20,000 additional operations a year!

Bariatric surgery should only be considered as a last resort if serious attempts to lose weight have been unsuccessful and if the person is obese. Bariatric surgery can lead to dramatic weight loss which in turn may result in a reduction in the amount of type 2 diabetes medication required, or in some cases no medication will be needed at all. However, this does not mean that type 2 diabetes has been cured. These people will still need to eat a healthy balanced diet and be physically active.

The recommendations also provide guidance on the use of very low-calorie diets (800kcal per day or less). These include:

- Not routinely using low-calorie diets to manage obesity.
- Only considering very low-calorie diets for a maximum of 12 weeks as part of a multicomponent weight management strategy with on-going support.

- Giving counselling and assessing people for eating disorders or other mental health conditions before starting them on a very low-calorie diet – to ensure the diet is appropriate for them.

The risks & benefits of surgery should also be discussed. Patients should be made aware that very low-calorie diets are NOT a long-term weight management strategy and that regaining weight is likely, but not because of a failure on their or their clinician's part.

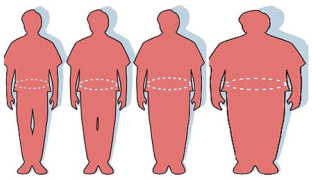
Bariatric patients have clinically significant OSA:
The majority of bariatric surgery patients have clinically significant OSA, but report fewer symptoms than other sleep disorder patients, according to a study in the journal of Sleep and Breathing by lead author Dr Sharkey. The report's author warned that patients with severe obesity need evaluation for OSA as they under-report symptoms and self-report measures are not an adequate substitute for objective assessment and clinical judgement when evaluating bariatric patients for OSA.

Bariatric surgery reduces OSA in severely obese patients:
Bariatric surgery results in a reduction in the symptoms of OSA according to the results of a randomised clinical trial in Brazil & Italy (Aguiar et al). The findings of the study demonstrate that weight loss following bariatric surgery for severe obesity effectively reduces neck and waist circumference, improves pulmonary function, improves sleep architecture and reduces respiratory sleep disorders especially OSA via a reduction of apnoea-hypopnea index.

Therefore in summary, Sleep services across the country should be prepared for an increase in pre-bariatric surgery diagnostic sleep studies and subsequent CPAP set-ups!

Find out more:

- Obesity: Identification, assessment and management of overweight and obesity in children, young adults and adults. National Institute for Health and Care Excellence (*NICE) draft guideline for consultation
- http://www.nice.org.uk/Guidance/GID-CGWAVE0682/Consultation
- NHS choices, Your health, your choices
- http://www.nhs.uk/news/2014/07July/Pages/Offer-weight-loss-surgery-to-diabetics-says-NICE.aspx
- Diabetes UK
- www.diabetes.org.uk
- NICE draft on obesity guidelines update 2014
- www.nice.org.uk/guidance/GID-CGWAVE0682/Consultation
- Sharkey *et al:* (2013)Subjective sleepiness and daytime functioning in bariatric patients with obstructive sleep apnea *Sleep and Breathing* **17 (1):**267-274 http://www.mrmjournal.com/content/9/1/43
- Aguiar *et al.* (2014) Obstructive sleep apnea and pulmonary function in patients with severe obesity before and after bariatric surgery: a randomized clinical trial. *Multidisciplinary Respiratory Medicine* **9:**43. http://www.mrmjournal.com/content/9/1/43

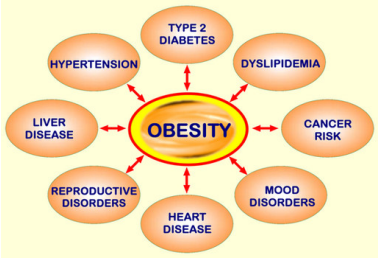


Obesity - The big problem that keeps on growing!

by Dr Vicky Cooper, Principal Respiratory Physiologist, Salford Royal

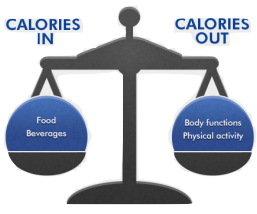
The world is getting fatter and it is also getting sicker. There has been an overwhelming increase in the cluster of chronic diseases termed metabolic syndrome, which include obesity, type 2 diabetes, hypertension, hypercholesterolaemia, and cardiovascular disease. As those reading this newsletter will know, this increase in obesity has also led to an increase in the incidence of

obstructive sleep apnoea (OSA). OSA itself is associated with numerous other health problems. Other obesity-associated diseases include non-alcoholic fatty liver disease, kidney disease and polycystic ovarian syndrome. Obesity is also associated with orthopaedic problems, gallstones and depression. Obesity and its associated health problems is now the biggest health concern in the world. So the world is getting fat, sick and sad!



The rapid increase in obesity has really only occurred over the last 35 years. The number of overweight and obese individuals worldwide increased from 857 million in 1980, to 2.1 billion in 2013.The 2013 rates for the UK were 66.6% of men and 57.2% of women were either overweight or obese. It's not just adults who are suffering. There has been a 47.1% increase in the worldwide prevalence of overweight and obesity combined in children in the same time period with the UK rates in 2013 being 26.1% of boys and 29.2 % of girls[1]. Teens with type 2 diabetes used to be unheard of, now they make up over 10% of all new diagnoses in the USA[2]. The problem of obesity and its associated illnesses are global, not only affecting wealthy Western countries, but, increasingly affecting under developed countries at an alarming rate. India, which has 270 million people living below the poverty line and still has massive problems with malnutrition, has the second highest incidence of diabetes in the world, with 65.1 million effected[3] and the 3rd highest incidence of obesity[1]. This is in terms of numbers of people affected rather than percentage of population, so as one of the largest countries in the world these headline figures are somewhat biased. Nevertheless, at 19.5% of men and 20.7% of women in India being overweight or obese the figures are not to be ignored. The International Diabetes Federation predicts a 55% increase in the worldwide incidence of diabetes between 2013 and 2035 to a whopping 592 million people.

So what is causing this global epidemic and what can we do to stop it? Surely we just need to "do more and eat less", for that is the mantra of almost every piece of diet advice you will come across (including, disappointingly, the latest draft of the NICE obesity guidelines). But are all overweight people really just gluttons and sloths and is this the characteristic of over half the UK population? If the answer is really so simple why is it so difficult for people to lose weight and keep it off? Why does every study on calorie restricted diets show poor success in the long term, with more often than not weight regain and then some? What if the very advice on how to lose weight and the diet products we are encouraged to consume are the very things causing the problem? In this article I will explore some eye opening revelations and the myths surrounding healthy eating, obesity and weight loss.



The first myth – energy in equals energy out, therefore eat less and do more = weight loss

It is an almost universally held belief that the only way to lose weight is to eat less and/or do more. We read this on many respected websites and guidelines. For example the BBC advice website states[4]: "There is only one cause of obesity: we eat more than we need and we store the extra calories as fat." The website patient.co.uk[5] states "In some respects, the cause sounds quite simple. Your weight depends on how much energy you take in (the calories in food and drink) and how much energy your body uses (burns) up:

- If the amount of calories that you eat equals the amount of energy that your body uses up, then your weight remains stable.
- If you eat more calories than you burn up, you put on weight. The excess energy is converted into fat and stored in your body.
- If you eat fewer calories than you burn up, you lose weight. Your body has to tap into its fat stores to get the extra energy it needs.

Even the British Nutrition Foundation[6] states: “Your body weight is determined by the amount of energy obtained from your food compared to the amount of energy that your body is using. The surplus energy you take in from food and drink is mostly stored as fat. To lose weight, the energy you take in from food must be less than the energy you use, in other words eat less and exercise more.”

The following statement is taken from the British Dietetic Association in their leaflet *Want to lose weight & keep it off?”* “one pound of fat contains 3,500 calories, so to lose 1lb a week you need a deficit of 500 calories a day”. Similar statements can be found in many authoritative publications such as the joint BBC / Welsh Assembly Government campaign *The Big Fat Problem* (2004),Wardlaw & Smith’s Contemporary Nutrition (often thought of as the ‘bible’ of nutrition), BUPA, *Healthy Living booklet*, NHS booklet *Your weight, your health*, NICE guideline 43 (Obesity), the American Department of Health and Human Services, The American Academy of Family Physicians, the American weight loss programme Anne Collins, even Wikipedia states “there are 3500 calories in 1lb (0.45Kg) of body fat…if someone has a daily allowance of 2500 calories, but reduces their intake to 2000, then the calculations show a one pound loss every seven days.” This weight loss advice is also quoted in the majority of diet books lining the shelves of book stores[7]. The precision to which this formula is applied is ludicrous, particularly because one pound of fat does not equal 3,500 calories, but in itself is an approximation of an approximation[7].

One example of how calorie counting is taken to a ridiculous extreme is in book *The obesity Epidemic and its management* written by Terry Maguire and Professor David Haslam (chair of the National Obesity Forum)[8] which states “Small daily excess in calories, as little as 50kcal per day, can lead to large accumulation of fat deposits over a number of years. This excess can occur through an increase in energy intake (eating too much calorie-dense foods) or a reduction in energy expenditure (too little physical activity) or both.” The question is without precisely measuring your basal metabolic rate on a daily basis (and taking into account activity levels on that day) how would you know if you are eating 50 calories too much (or in that fact too little?). A quick “google search” (other search engines are available) reveals that for 50 calories I could eat 2 Laughing Cow Light Cheese Triangles, 12 green olives, 2 medium satsumas, a small scoop of vanilla ice-cream, or a single Jaffa cake (45 calories). With the varying calorific needs it would be hard to determine whether eating (or not) any of these small portions would amount to a 50 calorie excess (or deficit). However, if you apply this formula, then the 50 daily calorie excess would lead to a 5lb gain in weight per year and over 3.5 stones in 10 years. Conversely, applying this as a deficit this is the weight loss you would expect over 10 years by not having that Jaffa cake. This of course does not take into account as you gain weight your energy requirements increase and as you lose weight they decrease which then gets us stuck with a circular reference calculation.

This underlying principal of eat more do less is based on the conservation of energy in the first law of thermodynamics: the total energy of an isolated system is constant; energy can be transformed from one form to another but cannot be created or destroyed. The problem is that this has been oversimplified. The human body is not a closed system in thermal equilibrium. This simplified view neglects to take into account “insensible energy” that is used in the making of energy and energy unavailable or lost to the system in any way. There are significant implications to ignoring this. The other frequently made error is the assumption of a direction of causation. It is assumed that too much energy in and /or too little energy out causes weight gain. The reverse is not considered, that weight determines energy in (heavier people need more fuel) and energy out (heavier people find it more difficult to move). Thus we push the propensity that energy in and/or out determines weight but we neglect to consider how weight may determine energy in and/or out. This then readily pushes the blame onto the individual who is obviously eating too much, doing too little and has no self control!

The first study of the effects of giving someone insufficient energy was by Francis Benedict[9] in 1918. This study showed that less energy in is more likely to result in less energy out than in the system using itself up (ie losing weight). Notwithstanding the fact that the first law is about energy conservation then it is logical that less energy in will equate to less energy out.

The definitive study for this may be attributed to the American Epidemiologist, Ancel Keys[10] who published his Minnesota starvation experiments in 1946. These studies were carried out on conscientious objectors during the war. He recruited 36 healthy young men of normal weight. They were accommodated in the University football stadium and starved for 24 weeks. Dependent on their starting body weight they were restricted to between 1600 and 1800 calories a day whilst still working and performing physical exercise, walking 15km per week. The purpose of the studies was to simulate the starvation that would be occurring in war-torn Europe. The researchers would then re-feed the men according to different schedules so that the US had real data on the best diet for the recovery of Europe. As would be expected, the men experienced significant weight loss (an average of 25%). Their diet was strictly controlled, so it makes sense that if they were fed a lot less than they needed, their body had to compensate. It did so by losing both muscle and fat mass. However, the other symptoms were less expected. The men immediately lost all sex drive, they were constantly hungry, became tired and lethargic and slept as much as they were allowed. They reported feeling cold all the time and struggled even to lift their feet over the gutter when crossing the road. Eventually the researchers noted that their subjects developed significant apathy, irritability, loss of cognitive function and depression.

More recent research on the effects of highly restricted calorie diets on over-weight subjects has come from reality TV shows such as Biggest Loser. Researchers from the Pennington Biomedical Research Centre measured body composition and energy expenditure in individuals of one such televised weight loss competition[11]. All participants were morbidly obese with an average BMI of 49.4kg/m^2. By week 6 participants had lost more than 10% of their body weight and by week 30 they had lost nearly 40%. However, they also found that the contestant's metabolism slowed much more than might be expected. As one loses weight the metabolism slows down since less energy is needed to be produced as quickly because there is less weight to carry around. However, the researchers showed that by week 6 the contestants' metabolism had slowed by 244 calories per day more than would have been expected by weight loss alone. By week 30 they were burning a massive 504 calories per day less than would be expected. Subjects with the greatest weight loss had the greatest metabolic adaptation. What this effectively means is they'd have to eat 500 calories less (one less meal) per day than someone who was naturally at the same weight, in order to maintain the same weight and eat even less to lose more weight. This is perhaps why so many of them find it is impossible keep the weight off. Whilst we may not be able to deny that more energy in than is used is going to lead to energy storage (weight gain), the reverse cannot always be held true. Whilst in the short term calorie deficiency may lead to weight loss, there are clear metabolic adaptations that act to attenuate this loss and this makes further weight loss or even maintenance of weight loss most difficult.

The second myth – a calorie is a calorie

In addition to the myth that it is all about the energy in versus energy out, a frequently mis-held belief is that all calories are equal, and thus it doesn't matter what you eat so long as you cut the number of calories. However, not all calories are created equal and what your body does with those calories (and how much energy it uses in the process) very much depends on the macro-nutrient from whence those calories came. Of course, it is quite logical that the amount of insensible energy used will depend on the process of digestion, which is quite different for fats, proteins and carbohydrates.

As early as 1932 Lyon and Dunlop[12] noted that patients on isocaloric diets lose more weight when the largest proportion of calories was from fat than when it was supplied by carbohydrates. In a series of 3 experiments in 1956, Kekwick and Pawan[13] demonstrated beautifully how the type of macro-nutrient is just as, if not more important than the calorie content. In their first experiment patients were given a "normal" 47:33:20 carbohydrate:fat:protein ratio. The proportions were kept constant but the calories per day were decreased from 2000 to 1500, to 1000 and finally 500 per day. They found a relationship between lower calories and weight loss but it was by no means a straight line and certainly did

not find a pound of fat loss for every 3500 calorie deficit (as the energy balance equation predicts). The second experiment entailed giving patients 1000 calories consisting of either 90% fat, 90% protein or 90% carbohydrate. The 90% fat group lost the most (2.3Kg), with the 90% protein just a little behind (1.7Kg) whilst the 90% carbohydrate group had negligible weight change. In the third experiment 5 patients were observed to maintain their weight on a 2000 calorie 47:33:29 carb:fat:protein diet. They then put them on 2000 calories of carbohydrate per day and they all gained weight. They subsequently put 5 people on 2600 calories of fat/protein and all but one lost weight (the one that didn't was a woman who retained more than 3 litres of water during menstruation). Further studies have shown low carbohydrate diets are much more effective than low calorie diets[14,15].

The third myth – eating fat makes you fat and meals should be based on carbohydrates

Standard nutritional advice is that we should eat less fat, (particularly saturated fat) and the majority readily follow this advice, buying more expensive lean cuts of meat than their cheaper full fat counterparts and the chemical concoction that is margarine rather than more natural and cheaper butter. Our supermarket shelves are stacked with "light" and "low fat" products with "less than x % fat" (preferably as low as possible) or "y% fat free" (preferably as high as possible) being major advertising points on the front of packaging.

There are two reasons why fat has been demonised. The first is that in the calorie counting world, gram for gram fat contains over twice as many Calories (approximately 9 Calories per gram) than protein or carbohydrate (both 4 Calories per gram). Therefore you can eat twice as much carbohydrate as fat for the same calorie intake. What this narrow-sighted view forgets is that fat is actually much better at making you feel full (satiated) than carbohydrate and therefore you are less likely to eat as much. Many people can easily eat a big bowl of pasta or sugary cereal, only to feel hungry again an hour or two later.

The second reason for demonization of fat, particularly saturated fat is that of its apparent link to heart disease. The first proponent of the idea that fat could be the cause of coronary thrombosis was the very same man of the starvation studies above: Dr Ancel Keys. In the 1940's Keys postulated that the apparent epidemic of heart attacks in middle-aged American men was related to their diet. In 1958 Keys began his seven countries study[16] in which 12,763 males aged 40-59 years were enrolled from seven countries across the United States, Nothern Europe, Southern Europe and Japan. Subjects were recruited over a 6 year period, but the study continued for a period spanning over 50 year with many publications resulting from it[17-19].The Seven Countries Study suggested that the association between blood cholesterol level and coronary heart disease risk from 5 to 40 years follow-up is found consistently across different cultures. It also suggested Cholesterol and obesity were associated with increased mortality from cancer. However, what Dr Keys has never explained is what made him choose those particular countries when the data existed for a number of other countries. If the data from these other countries is added, then the beautiful correlation of higher fat consumption with higher mortality from coronary disease is less than clear cut. He also chose to leave out of his studies indigenous tribes such as the Inuit of North America, who ate only animal fat and have amongst the lowest prevalence of heart disease on earth. Correlation does not necessarily mean causation. Keys himself noted in his 500 page mega-opus "the incidence rate of coronary heart disease was significantly correlated with the average percentage of calories from sucrose in the diets" but went on to day that this "is explained by the inter-correlation of consumption of sucrose and saturated fat." So Keys simply discounted the sugar issue and did no further studies to show which was the driving force the sugar or the fat.

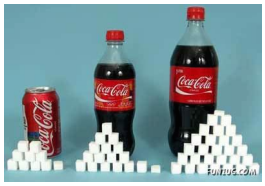
It is true that dietary fat increases blood LDL (low density lipoprotein) levels. However, another issue with the Keys conclusions are they assume that all LDLs are bad. In fact, there are two types of LDLs: large buoyant LDL (type A LDL), driven by dietary fat and small dense (type B LDL) driven by dietary carbohydrate. Type A LDL float in the blood stream and are too big to get underneath the cells lining the blood vessels to start the atheroslcerotic process and hence this type of LDL are neutral in terms of heart disease. The small dense type B LDL sinks and is small enough to get underneath the blood vessel cells and has been specifically implicated in the formation of atherosclerotic plaques.

Another complicating factor is there are many types of dietary fat and all have different effects on the body. For example Omega -3 fatty acids such as those found in fish, have been found to be protective against heart disease. However, Omega-6 fatty acids found in seed oils and in farm reared animals fed on corn and soy, are pro-inflammatory and are implicated in atherosclerosis, insulin resistance and immune dysfunction. Trans fats (such as margarine) which have since been shown to be significantly implicated in heart and metabolic disease[20]. So swapping out butter for margarine and lard for vegetable oil may not be so healthy after all.

There have been numerous large scale studies that have questioned the long held views that heart disease is all about fat and cholesterol levels, yet standard nutritional advice has not changed.

The majority of dietary guidelines, including food pyramids and other pictorial guidance suggest that the majority of our food intake should come from carbohydrates. However, as the studies described above show, diets high in carbohydrate can drive weight gain and / or curtail weight loss even when there is a calorie deficit. Triglyceride is human fat. This is a structure of three fatty acids joined to a backbone of glycerol. Glycerol is formed from glucose and glucose is provided by carbohydrates. Thus, carbohydrates can be formed into fat. Ingestion of carbohydrates leads to release of insulin to counteract the increase in blood glucose that would otherwise occur. Insulin is the fat storage hormone and therefore a diet high in carbohydrate is going to promote fat storage. Without insulin (ie in a low carbohydrate diet) the body will break down fat to release the necessary glucose for energy, and hence weight loss can occur.

The real culprit



There are various kinds of diets that can all be successful in weight loss. Low carbohydrate, high fat or, low fat, high carbohydrate, whilst seemingly diametrically opposite have one thing in common when successful and that is low sugar. It is much more difficult to be low fat and low sugar, because fat removal equals flavour removal, and in most cases that flavour is returned to products in the form of sugar. (Next time you are in the supermarket look at the sugar content of the extra-light mayo compared to the full fat version). There has been a lot of media attention recently about the adverse effects of sugar. However, this concept isn't new. John Yudkin, a British physiologist and nutritionist, first determined that over consumption of sucrose was most closely associated with heart disease over 40 years ago. He was the first to show that sugar uniquely raises serum triglycerides and insulin levels and first publish his seminal work on the subject in 1972 in a book called "Pure, White and Deadly[21]". He has since published numerous papers on the biochemistry of sugar, in particular the molecule fructose and its effects on coronary heart disease, diabetes, GI disease, eye disease and other inflammatory diseases[22].

Sugar is the most common and successful food additive the world over. It is highly addictive and adds palatability to foods making us buy and consume more. It is also cheap and is therefore added to almost every known processed food. We have also been duped into believing that one source of sugar is more healthy than another. For example, most of us are aware that sweets,

cake and chocolate are unhealthy, but when things are sweetened by fruit juice concentrate or "natural" products such as honey we are led to believe they are a healthy alternative. With the constant push to get your 5 a day (or other amount per day depending on which country you live in) we are drinking juice and giving our kids congealed fruit bars and snacks believing we are giving them something healthy. Because, let's be honest, how many children do you know that are happy to eat 5 portions of vegetables every day. I'm lucky to get one or two down my children. However, it doesn't matter where the fructose came from, fruit, honey, syrup or table sugar (sucrose) the effects on the body are the same. When you juice, dry or concentrate any fruit you take away the fibre content (which stops you over consuming it) and concentrate the sugar content. Therefore a glass of apple juice is not really any better for you than a can of coke. Yes it may contain a small amount of vitamin C but you are far better off eating an apple and having a glass of water to drink.

So what is the problem with sugar anyway? Sucrose is a molecule of glucose bound to a molecule of fructose. Unlike glucose, which can be metabolised by all organs of the body, the primary site of fructose metabolism is the liver[23].This means that fructose has major effects on the liver, increasing its energy requirements, depleting the liver cells of adenosine triphosphate (ATP). ATP depletion leads to the generation of the waste product uric acid – which causes gout and increases blood pressure[24]. Fructose enters the bloodstream more slowly than glucose and its levels are much lower, but they persist longer in the circulation[22].

Several studies have pointed to the deleterious effect of fructose on glucose metabolism and insulin sensitivity. Indeed, a high-fructose diet has been found to increase glucose and insulin responses to a sucrose load[25] increase fasting glycemia[26], and lead to hepatic insulin resistance in healthy men[27]. Insulin resistance is closely linked to lipid metabolism disorders; more specifically, insulin-resistant subjects have higher ectopic lipid deposition, which may generate toxic lipid-derived metabolites, such as diacylglycerol, fatty acyl CoA, and ceramides. The presence of these metabolites in the intracellular environment leads to a higher serine/threonine phosphorylation of insulin receptor substrate-1 (IRS-1), which has been shown to reduce insulin signalling[28]. Insulin resistance and its associated abnormalities are of utmost importance in the pathogenesis of NIDDM (non-insulin dependent diabetes mellitus), hypertension, and coronary heart disease[29].

Insulin resistance leads to high levels of circulating insulin (the pancreas needs to produce more to have the same effect on lowering blood glucose), which in turn can drive the growth of many cancers[30]. There are clear links between insulin resistance, diabetes and dementia.

Fructose is metabolised in a similar way to ethanol and therefore not surprisingly increased fructose intake is associated with non-alcoholic fatty liver disease (NAFLD)[31-34]. NAFLD is the most common type of liver disease and its incidence has paralleled the increased incidence of obesity. However, you don't need to be fat on the outside to be suffering from NAFLD. So if you are lucky enough to maintain a slim figure despite a high sugar diet, be warned of the hidden damage you may find inside. You may well be what is known as a TOFI (Thin on the Outside, Fat on the Inside).

So fructose is associated with obesity, diabetes, liver dysfunction, cancer and dementia. To make matters worse it acts on both the starvation and reward pathways, tricking your brain into wanting more. It blocks leptin signalling so your brain thinks you are still hungry and it doesn't decrease ghrelin (the hunger hormone). It therefore disrupts the appetite control system which can lead to over-eating, weight gain and obesity[35].

In summary, the standard "healthy" nutritional advice of low fat, high carbohydrate diets is not working. The world is getting fatter and sicker. Whilst ever, we still bang the drum that the way to lose weight and prevent or cure obesity is to eat a such a "healthy" diet, and to cut calories the world will continue to get fatter, as will the profits of the big food companies. It's time to wake up and smell the fructose!

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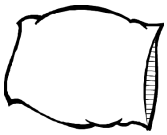
Further Recommended Reading.
Robert Lustig (2012) Fat Chance, The Bitter Truth About Sugar, Obesity and Disease *Harper Collins Publishers*
David Gillespie (2012) Big Fat Lies *Penguin Books*
Zoe Harcombe (2010) The Obesity Epidemic: What Caused It? How Can We Stop It? *Columbus Publishing*



Pillow Talk manufacturers news, new equipment and a bit of gossip!

There are a number of new technologies and masks that have been launched since the last issue of SNEWS, however none of the manufacturers sent us any info, so we shall await an update in the next edition.

Alan Moore has been checking out the news stories regarding the world of Sleep Manufacturers:

SAN DIEGO and TAIPEI, Taiwan, Sept. 4, 2014/PRNewswire/ -- ResMed Inc. and APEX Medical Corp. have agreed to a confidential settlement resolving their outstanding patent disputes. Under the agreement, APEX has the right to sell and import globally its Wizard masks, as well as its XT and iCH flow generators. APEX agreed to dismiss all validity challenges to ResMed patents, and ResMed agreed to dismiss pending litigation. Additional details of the settlement are confidential.

In accordance with the settlement, ResMed is requesting termination of its lawsuits against APEX's products. Similarly, APEX has requested dismissal of its challenges to ResMed patents pending in the United States, Europe, Germany, Japan, and China. The settlement brings to a conclusion a worldwide legal conflict that began in March 2013, when ResMed filed a patent infringement suit against APEX with the U.S. International Trade Commission and in federal court in Los Angeles.

"As the premier innovator in the sleep-disordered breathing market, it's critical that we defend our innovations," said David Pendarvis ResMed chief administrative officer and global general counsel. "We welcome healthy, fair competition in this growing market. This agreement with APEX is a good result that meets our goals and allows ResMed to continue to focus on improving lives for millions of people."

"As one of the leading innovators in respiratory therapy and pressure area care sectors, APEX believes in healthy, fair competition in the market place. Those in need of respiratory therapy benefit when efforts are directed towards innovations to improve quality of life," said APEX's Vice

President of Sales, PJ Hsueh. "We view today's global resolution of disputes as a victory for people suffering from sleep apnea."

Breas Medical AB has recently announced the takeover of B&D Electromedical Ltd, manufacturers of the NIPPY range of ventilator/cough assist products:

> *" We would like to share with you the news of the acquisition of B&D Electromedical, Ltd. by Breas Medical AB. Breas Medical, headquartered in Sweden, is a leading manufacturer of home respiratory ventilators and sleep apnoea products for the global home healthcare market sold in more than 40 countries. Founded in 1991, Breas employs more than 100 people in nine countries and operates a 30,000 square-foot manufacturing/office facility in Sweden. Breas Medical is a portfolio company of PBM Capital Group. This is a positive and significant business win for both companies. With the B&D acquisition Breas Medical becomes the market leader in yet another European home ventilation market, through the respected and established NIPPY portfolio, alongside its leading positions in Germany, Iberia, France, Benelux, Italy, and Nordics. Breas customers in the UK will also benefit from the expanded capabilities and B&D's excellent customer support, service and education. For B&D this means access to the vast Breas channel globally, as well as different quality, regulatory and manufacturing processes which Breas Medical is experienced in. Breas Medical will help accelerate growth of the Clearway portfolio in Europe and globally as well as the continued innovation of the NIPPY Ventilator platform. B&D and Breas Medical do to some extent share a similar company history and in many ways share the same company culture: Providing customer focused innovative products, building their business on close and lasting customer relations as well as offering personal and excellent customer support. B&D's founders, Peter Bachelor and Noel Davis, will remain with the company to develop new, innovative NIPPY respiratory devices and airway clearance products for the UK and global markets. Your relationship with the commercial team will remain intact, to continue delivering first class customer service, education and support. NIPPY will be the key Brand and product offering for Breas Medical in the UK and Ireland, now and in the foreseeable future. To this Breas adds a portfolio of complementary home mechanical ventilation and Sleep therapy products. The Breas Medical management team will work together with B&D to establish our combined forward going product offering in the UK and Ireland. Until the practical steps are finalised for merging our operations and support functions, please continue your communication with us as normal for any enquiries. We are committed to continue the product innovation and close customer relationships with the combined strengths of the two companies and look forward to our continued cooperation."*

Sleep disordered breathing in the spotlight at the European Society of Cardiology.

by Emma Braithwaite

Following on from Prof Mary Morrell's keynote speech at the ARTP conference in 2014 and "What role does the identification and treatment respiratory-sleep disorders play in the management of cardiology patients? " the spotlight turned to sleep disordered breathing at the recent European Society of Cardiology Congress in Barcelona. Topics covered included the impact of sleep apnoea on outcomes in coronary heart disease and heart failure, the need to recognise it in the cardiac patient how it can be identified and treated today and potentially in the future.

Impact of CPAP on outcomes in patients with coronary heart disease and heart failure
A study using the German Statutory Health Insurance (SHI) database covering ~5% of the German population showed that the three-year mortality of people with sleep apnoea – a prevalent co-morbidity in coronary heart disease (CHD) and heart failure (HF) – was significantly lower in patients who were treated with positive airway pressure (PAP) devices compared to a comparable cohort that received no PAP treatment. Mortality was reduced by 37.9% in patients with CHD (p=0.0002) and by

31.6% in patients with HF (p<0.0001). "Sleep apnoea is a highly prevalent co-morbidity in both coronary heart disease and heart failure, yet it remains frequently undiagnosed and thus under-treated," said Professor Michael Böhm, Professor of Cardiology, University of the Saarland, Homburg, Germany and co-author of the analysis. "The results from this analysis highlight just how important it can be to identify and appropriately treat this condition, not only to improve quality of life, but also patient survival. It is vital that, as a community, cardiologists do more to recognise this and explore how we can ensure patients receive respiratory device therapy when needed."
A group of patients with sleep apnoea being treated with PAP therapy was chosen (4,068 patients). Propensity score was used to define a control group of an equal number of patients with sleep apnoea who received no PAP treatment. Patients were followed over three years after initiation of their PAP therapy with results showing that the three-year mortality rate was significantly lower in patients treated with PAP compared with the no PAP group (4.5% vs 7.2%, 37.5% reduction; p<0.0001). Three-year rates for CHD mortality (4.5% vs 7.2%, 37.9% reduction; p=0.0002) and HF mortality (14.7% vs 21.4%, 31.6% reduction; p<0.0001) were also significantly lower in the PAP vs. no PAP group.

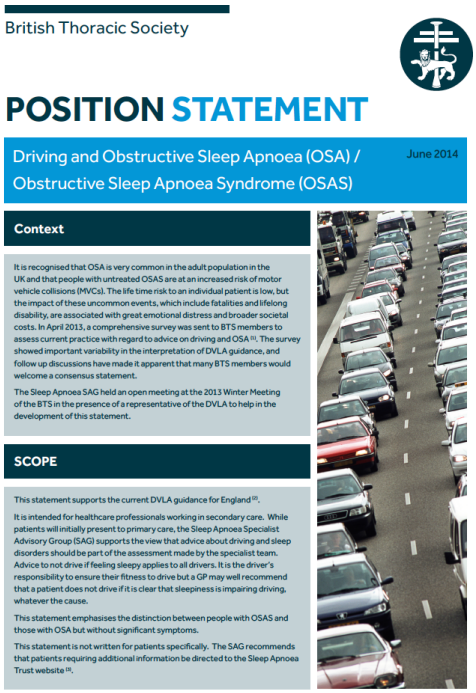
Diagnosing Sleep Apnoea in the future
An additional study, highlighted that, in the future, HF patients with sleep-disordered breathing could be more accurately diagnosed through the use of the at-home, contactless, bedside SleepMinder™ device.

SDB is typically diagnosed in patients with HF by calculating the Apnoea-Hypopnea Index (AHI), typically from a single overnight study. However, HF patients commonly experience volume load changes that can make AHI highly variable in this population over time. Mean AHI assessment, over a longer period is therefore likely to be more accurate.[ii] SleepMinder™ is a non-contact, bedside, nocturnal respiratory monitor that can be placed next to a patient's bed to collect data on SDB over longer periods. Two weeks represent a useful period for SDB assessment to be gathered. This study involved 39 adult patients with HF who were assessed over 12 months.

The study also assessed how SleepMinder™, when used over two weeks, compared to a single night assessment via inpatient polysomnography (PSG). The investigating team reported that, after using SleepMinder™ for two weeks, 57% of patients were consistently above a threshold that would require treatment for their SDB (AHI≥15). This rose to 74% in patients who were followed up for 12 months.[ii]

"Sleep disordered breathing is the most common heart failure co-morbidity, yet the diagnosis is often missed," said Professor Martin Cowie, Professor of Cardiology, Royal Brompton Hospital, London, UK and co-author of the study. "This condition affects millions of heart failure patients across Europe and can lead to significantly worse outcomes if not correctly diagnosed and treated. This study showed that, by using a device that can gather longer-term data, we can offer a means of diagnosis that is both practical for patients, doctors and health services and may well be more accurate than existing techniques that require inpatient care. It is vital that cardiologists explore how they can help to establish more routine use of this technology in the heart failure patient pathway. These results can have significant implications for the routine diagnosis of this prevalent condition in HF.

Finally, there was a satellite symposium entitled "Sleep-disordered breathing in heart failure; time for cardiologists to take control". This session had more attendees than seats (in excess of 400) and the overwhelming message was that cardiologists should actively look for SDB and treat OSA in their patient population while awaiting the results of the ResMed Serve-HF randomised clinical trial which will answer the question of what to do with those heart failure patients who experience predominantly central sleep apnoea. The trial is expected to report in 2016, in the meantime do not be surprised if one of your cardiology colleagues starts talking to you about sleep.


British Thoracic Society

POSITION STATEMENT

Driving and Obstructive Sleep Apnoea (OSA) / Obstructive Sleep Apnoea Syndrome (OSAS) June 2014

Context

It is recognised that OSA is very common in the adult population in the UK and that people with untreated OSAS are at an increased risk of motor vehicle collisions (MVCs). The life time risk to an individual patient is low, but the impact of these uncommon events, which include fatalities and lifelong disability, are associated with great emotional distress and broader societal costs. In April 2013, a comprehensive survey was sent to BTS members to assess current practice with regard to advice on driving and OSA [1]. The survey showed important variability in the interpretation of DVLA guidance, and follow up discussions have made it apparent that many BTS members would welcome a consensus statement.

The Sleep Apnoea SAG held an open meeting at the 2013 Winter Meeting of the BTS in the presence of a representative of the DVLA to help in the development of this statement.

SCOPE

This statement supports the current DVLA guidance for England [2].

It is intended for healthcare professionals working in secondary care. While patients will initially present to primary care, the Sleep Apnoea Specialist Advisory Group (SAG) supports the view that advice about driving and sleep disorders should be part of the assessment made by the specialist team. Advice to not drive if feeling sleepy applies to all drivers. It is the driver's responsibility to ensure their fitness to drive but a GP may well recommend that a patient does not drive if it is clear that sleepiness is impairing driving, whatever the cause.

This statement emphasises the distinction between people with OSAS and those with OSA but without significant symptoms.

This statement is not written for patients specifically. The SAG recommends that patients requiring additional information be directed to the Sleep Apnoea Trust website [3].

BTS Position Statement on Driving and Obstructive Sleep Apnoea (OSA) / Obstructive Sleep Apnoea Syndrome (OSAS)

In June The British Thoracic Society published its position statement on driving and obstructive sleep apnoea and obstructive sleep apnoea syndrome. This statement gives welcome advice on what can be a difficult area for those treating patients with sleep apnoea. It helps give some clarity as to when we should be advising patients of the need to inform the DVLA giving clear distinction between those that are symptomatic (OSAS) and those with abnormal sleep studies but without significant sleepiness (OSA). Further information can be found here: https://www.brit-thoracic.org.uk/document-library/about-bts/documents/bts-position-statement-on-driving-and-obstructive-sleep-apnoea/

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