Dyslipidaemias | HEART UK 33rd Annual Medical and Scientific Conference REVIEW

Wednesday 3 – Friday 5 July 2019, University of Warwick

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The HEART UK conference this year focused its attention on the pharmacological and non-pharmacological ways of identifying and managing dyslipidaemia.

Dr David Unwin a GP from Southport and his wife Dr Jen Unwin, a clinical psychologist explained how one patient who reversed their Type 2 Diabetes Mellitus (T2DM) was pivotal to inspiring change across their entire practice population. This was achieved using the GRIN model which focuses on establishing the person's **G**oals, exploring the persons support, **R**esources and resilience, gaining small **I**ncrements towards agreed goals and encouraging the patient and the clinician to notice and reflect upon progress (**N**oticing).

Dr David Unwin shared examples of good practice in which giving people information to help them make good dietary choices improved their triglyceride levels by 30% and resulted in a 40% T2DM remission rate.

Suzanne Watkins, Lead Lipid Clinical Nurse Specialist from Cardiff shared some examples of good practice from her clinic. She used case studies to show the effectiveness of combination therapy including PCSK9 inhibitors, the role of drug therapy in combination with lipoprotein apheresis and the future potential for anti-sense therapy for Lp(a).

Alison Pottle, Consultant Nurse at Harefield Hospital, demonstrated the importance of trialling different drugs and how a switch of PCSK9 inhibitor can sometimes help. She also showed that 'it is not a case of saying its better its can we do better' with a young man whose life time exposure to cholesterol will be vital and how extra control was achieved in the lipid clinic with a combination of a statin, ezetimibe and a PCSK9 inhibitor.

Professor Steve Humphries (London, UK) led the audience through an update on FH genetics, explaining that mutations in any of three genes (*LDLR, APOB* and *PCSK9)* that cause autosomal dominant FH. Knowing the gene defect that causes FH is important because it helps predict the severity of the disease. Compared to FH patients where no mutation can be found, having an *LDLR* mutation roughly doubles the odds of having a cardiac event, having an *APOB* mutation increases risk 3 fold while with a *PCSK9* mutation there is a 20 fold increase, because of the long term extreme cholesterol burden in these people.
Historically the prevalence of FH was said to be 1/500 but UK data collected from the screening of over 10,000 children gave a 1/270 ratio. The 2019 NHS long term plan sets an ambition to increase the number of FH patients known for its current level of 7% of the predicted number to 25% within 5 years.

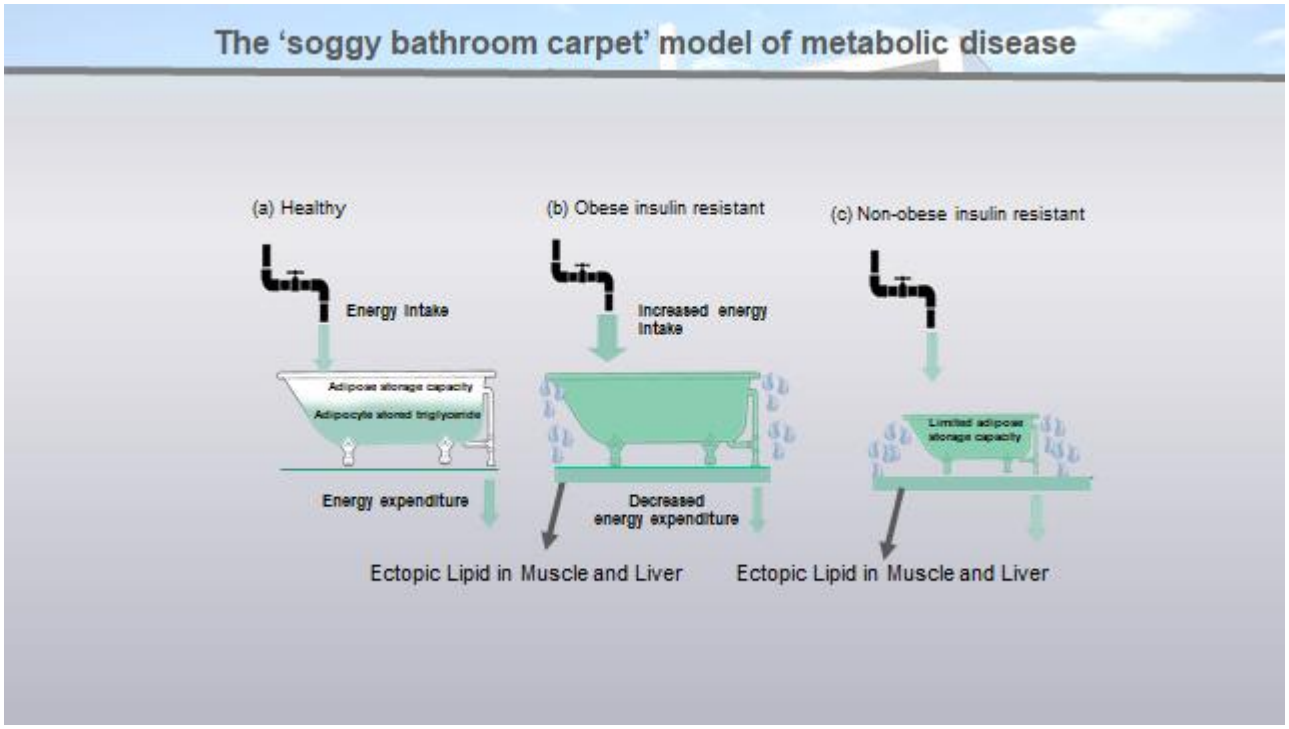
Dr Maggie Williams (Bristol, UK) described the work underway to get whole genome testing live within the NHS with seven genomic laboratories and a national genomic test directory now available on line with around 300 indications for testing rare diseases. The new Patient Choice consent model under development for patients has an opt in for research studies as part the national genomics research library. There are designated gene panel tests within the test directory for FH and Lipoprotein Lipase Deficiency (FCS) and familial/relative testing.

Professor Mike Khan (Coventry, UK) described gene silencing as like turning off the tap rather than just mopping the decks but went onto explain that this works well if you can get to the tap safely i.e. get the drug safely into the cell where the disease causing protein is being made. In the future 'different cargo' mRNA, gene editing and different oligonucleotide chemistry or different delivery systems may come into use.

Professor Charalambos Antoniades (Oxford, UK) described the use of coronary computed tomography angiography (CCTA), a new first line, non-invasive test for investigating chest pain endorsed by NICE.

It is interesting; however, that 50 % of the people who experience a heart attack do not have obstructed coronary arteries. CCTA and new biomarkers such as the Fat Attenuation Index (FAI) have been developed using AI and imaging to identify higher risk people.

In the **Myant Lecture this year** **Professor Sir Stephen O'Rahilly** **(Cambridge,UK)** tackled the causes and consequences of human obesity and challenged the view of metabolic disease as eating too much or expending too little. Work now suggests perhaps we should focus more on the concept of people having different sized baths – some cannot cope with a positive energy balance. You can then turn the tap down in people with small baths with treatments such as bariatric surgery



Metformin is a diabetic drug that is now being investigated as a cancer prophylactic, a CVD prophylactic and a dementia prophylactic. In obese, pre diabetic people metformin can reduces body weight mostly from fat.

Professor Sir O'Rahilly explored why some people are more prone to the weight gain and the metabolic consequences of over-nutrition than others. The role of human genetics is central in understanding these questions and will have broad ranging implications in the future.

Dr Uma Ramaswami **(London,UK)** presented data collected from just over 3000 children across Europe. The proportion of children older than 10 years who were receiving statin therapy ranged from 99% in Greece to 54% in Czech Republic. Overall, statin treatment reduced LDL-C by between 30-57%. Over 10 years of age, 25% of on-treatment children still had LDL-C levels over 3.5mmol/l and 67% of those not on a statin had LDL-C levels over 3.5mmol/l. In children over 10 years of age, the addition of ezetimibe reduced LDL-C to < 3 mmol/l for most individuals.

A new consensus statement provides a comprehensive care pathway for children and young people with heterozygous FH.