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Editorial

Conference on 'Optimal diet and lifestyle strategies for the management of cardio-metabolic risk'

Optimal diet and lifestyle strategies for the management of cardio-metabolic risk

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Cardio-metabolic risk (CMR) embodies a clustering of metabolic abnormalities that increase the likelihood of developing CVD in the large arteries of the heart, peripheral tissues and brain. These abnormalities share a common origin of insulin resistance, which manifests typically as excess visceral adipose tissue in the abdominal cavity, and within cells of key metabolic tissues (ectopic fat), including the liver, pancreas, heart and skeletal muscle. As expected, the increased risk of CVD that can be attributed to CMR factors is alarmingly high in overweight and obese populations, but this risk can be reduced by reversing many of the inappropriate diet and lifestyle behaviours that underlie its development. The Nutrition Society's 2018 Winter Meeting at the Royal Society of Medicine addressed the topic of the 'Optimal diet and lifestyle for managing cardio-metabolic risk', with the aim of providing mechanistic insights into the impact of macronutrients, dietary patterns and meal timing in key metabolic tissues. The 2-d programme concluded with a summary of its main outcomes, and an overview of their implications for dietary policy in the UK.

Obesity: Visceral fat: Dietary fatty acids: Carbohydrates: Ectopic fat

Cardio-metabolic risk (CMR) describes a constellation of metabolic abnormalities, including dyslipidaemia, dysglycaemia and hypertension, which increase CVD risk in a dose–response manner. These abnormalities are inextricably linked to excess visceral adipose tissue (VAT), ectopic fat and insulin resistance, and are most familiar to us as components of the metabolic syndrome[1]. The clinical utility of this syndrome has been questioned, because its whole may be of less value than its parts in terms of diagnostic power and efficacy of drug therapy[2]. However, this overlooks the subclinical and insidious development of central adiposity as a warning sign of disease risk, '*…like the sea retreating before the tsunami of metabolic syndrome, and diabetes and CVD roll in*'[3]. Chronic energy imbalance from the passive overconsumption of food and a sedentary lifestyle is fundamental to the accumulation of central body fat that drives the development of CMR. While this can be redressed by energy-restriction and physical activity, the quality and timing of macronutrient intake are also emerging as important determinants of CMR, and potential therapeutic modalities for the management of this highly prevalent source of CVD risk.

In his opening plenary lecture, Professor Jean-Pierre Després delivered a compelling overview of the clinical and health implications of CMR. As a renowned champion for the key role of VAT in the development of CMR, Professor Després emphasised the need to move beyond BMI, which provides no information about visceral adiposity, and thus variation in CMR between

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individuals. By comparison, waist circumference provides a superior metric of central adiposity and VAT mass, which when combined with a measure of elevated serum TAG ('hyper-triglyceridaemic waist'), offers significantly greater diagnostic power to discriminate VAT and its associated CMR, than waist circumference alone[4].

The first symposium on dietary fatty acids was opened by Professor Julie Lovegrove who provided an update on the evidence to support our National dietary guideline to reduce intake of saturated fat to 10% of total energy and to reduce CVD risk[5]. Much of the benefit of this dietary guideline has been based previously on the lowering of serum LDL-cholesterol, a causal risk factor for CVD. More recently, the replacement of dietary SFA with unsaturated fatty acids has been linked to improvements in CMR, including reductions in serum TAG, HbA1c, vascular dysfunction, and most critically, cardiovascular events. The most potent dietary fatty acids to exert a favourable impact on CMR are the long chain (LC) *n*-3 PUFA, namely EPA and DHA, which human subjects or man obtain mainly from marine sources. Professor Erik Berg Schmidt reviewed the main findings from several follow-up studies that used adipose tissue as an objective biomarker to study exposure to LC *n*-3 PUFA in relation to atherosclerotic CVD. Intake of these fatty acids was shown to be inversely associated with risk of myocardial infarction and ischaemic stroke caused by atherosclerosis and peripheral arterial disease, while evidence for the impact of plant derived α-linoleic acid on CVD events was more equivocal[6].

Various strategies for achieving energy-restricted weight loss have been described, one of which is by inducing mild ketosis by a high intake of dietary fat and exclusion of carbohydrate. Professor Stephen Phinney presented data on the impact of a 1-year, well-formulated, ketogenic diet in reducing body weight (11 %) and on CVD risk factors[7]. Mild ketosis has been associated with the promotion of weight loss through several plausible physiological mechanisms, including suppression of appetite and increased sympathetic tone. In this case, as in others, it is possible that the impressive loss in body weight from the energy restriction alone, made a substantial contribution to the favourable effects on CVD risk factors. Interestingly, and in contrast to the established beneficial effect of weight loss in lowering serum cholesterol, the ketogenic diet produced a consistent increase in serum LDL-cholesterol (10 %), presumably because of its high content of SFA.

The second symposium turned attention to the impact of diet on specific tissues, as key therapeutic targets for the modification of CMR, namely the liver, VAT and skeletal muscle. Dietary fat has been linked to the development of non-alcoholic fatty liver disease, a condition known as the liver's expression of metabolic syndrome. Professor Leanne Hodson presented evidence for a hierarchy of effects of dietary fatty acids on liver fat accumulation, with therapeutic implications for replacing SFA with unsaturated fatty acids, especially LC *n*-3 PUFA, which appear to be the most effective in lowering liver fat[8]. Dr Paul Petrus then discussed the differential effects of dietary fatty acids on regional fat depots, and presented evidence to suggest that PUFA and MUFA may exert greater benefit in reducing VAT and SAT, respectively, while replacing LC SFA with medium chain SFA may be an even more effective strategy for reducing VAT[9]. The age-related loss of skeletal muscle mass (sarcopenia) may exacerbate the development of CMR factors, most notably in the presence of obesity (sarcopenic obesity). Dr Oliver Witard addressed the role of LC *n*-3 PUFA in combating the adverse effects of sarcopenic obesity. Supplementation with these fatty acids expands their expression in skeletal muscle and by doing so, potentially primes muscle to respond to anabolic stimulation in the form of feeding and resistance exercise. However, evidence in support of a favourable effect on these fatty acids in promoting muscle protein synthesis in sarcopenic obesity is still lacking and in need of further work[10]. The first day concluded with a highly informative and thoroughly entertaining Keynote Lecture by Professor Eric Rimm from Harvard University on 'The optimal diet – so many pieces to that pie!'

Day 2 opened with the third symposium on dietary carbohydrates. Professor Ian MacDonald, Chair of the UK SACN Report on Carbohydrates and Health (2015), provided an overview of the recommendations for the reduction in intake of free sugars to no more than 5 % of total energy[11]. It is clear that the consumption of free sugars in excess of 20 % total energy intake, typically in hyper-caloric diets, can exert adverse effects on VAT, liver fat and CMR, and that free sugars at lower levels of intake contribute to the passive overconsumption of energy and thus body weight. However, since the average intake of free sugars in the UK is closer to 12 % energy, this is highly unlikely to reproduce the adverse metabolic effects of sugar fed in excess of 20 % energy in supplementation trials. Dr Denise Robertson then outlined developments in the field of resistant starches, compounds that avoid digestion in the small intestine and are fermented such as dietary fibres in the colon. Resistant starches have been associated with favourable effects on the regulation of body weight and CMR through changes in the gut microbiota and bile acid metabolism, and may harbour significant potential as components of an optimal diet for reducing CMR[12]. Dr Charlotte Evans closed the session by reviewing the impressive epidemiological evidence for the protective effects of dietary fibre on non-communicable diseases, and CMR factors, including blood lipids, blood pressure and type-2 diabetes[13]. It is possible that dietary fibres harbour greater potential for reducing CMR than other macronutrients. While our current evidence-based dietary guideline of consuming 30 g/d is realistic for affecting CMR, stronger policies need to be in place to reach this challenging target.

The fourth and final symposium addressed other lifestyle factors. Professor Antonis Zampelas examined the efficacy of whole dietary patterns in reducing CMR and endpoints of CVD, using 'Diet to Stop Hypertension' and the Mediterranean diets as the most well documented examples[14]. Dr James Betts highlighted the importance of the temporal pattern of food ingestion in relation to biological rhythms and metabolic regulation, using intermittent fasting and re-feeding as a model to illustrate the

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potential benefits of nutrient timing on cardio-metabolic health[15]. Professor Andrew Salter closed the session by addressing the question of whether our existing dietary guidelines for the prevention of CVD are relevant to people on cholesterol-lowering drug therapy, and raised the possibility that non-compliance could have negative implications for cardiovascular health[16].

The meeting culminated with an address from Public Health England's Chief Nutritionist, Dr Alison Tedstone, who addressed the theme and outcomes of the meeting within the context of our current dietary guidelines and future National policies for the prevention of CVD[17].

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Conflicts of Interest

None.

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