WORLD OBESITY

HOT TOPIC CONFERENCE:

Dietary Sugars, Obesity & Metabolic Disease Risk 2015

COURSE GUIDE

WORLD OBESITY

Welcome to Berlin and the World Obesity Federation Hot Topic Conference on Dietary Sugars and Obesity.

Consumption of dietary sugars have increased in the population and sugars have emerged as a major dietary component linked with obesity and associated metabolic outcomes. There appear to be separate and common pathways linking dietary sugars to obesity and related metabolic outcomes that can be due to the caloric load of excess sugars as well as the way sugars are metabolized, and these processes are relevant during all stages of the life cycle. The issues surrounding dietary sugars have become of global importance and have significant implications for the development of suitable local, national and global policies that might prove successful in combating obesity and associated chronic diseases. These issues include sugar/soda taxes, warning labels on foods/beverages that are high in sugars and global trade policies. The science behind the links between dietary sugars and obesity/metabolic outcomes is rapidly developing and I am delighted that the program over the next two days will address these issues.

I am excited at the prospect of scientific dialogue that brings together some of the leading researchers as well as more junior researchers and trainees working in this rapidly developing field. There are also numerous evolving controversies and discrepancies in this field of study that we will address and hopefully begin to resolve by bringing world experts together into an atmosphere of collegial sharing and consensus building. I would also like to thank my co-chairs (Barry Popkin, USA; Luc Tappy, Switzerland; Martin Wabitsch, Germany), Natasha Joyner from the World Obesity Federation, and all the generous sponsors for helping to make this happen. Thank you all for being here and I look forward to an exciting, inspiring and invigorating two days

Michael Goran
Scientific Chair

CONTENTS

Useful Information

Wifi Details

Login: myfarvouritehotel
Password: wififorfree

Professional Development

Attendance Provides 4 SCOPE points and 16 CPD points

Poster display times

All poster presenters are required to put their posters up on Monday 29th June during the first morning coffee break.

Poster presenters are required to remove their posters by the end of the last coffee break on Tuesday 30th June (15:00-15:30). Any posters not removed by 15:45hrs will be removed.

Venue information

The conference will take place at the Ameron Hotel Abion Spreebogen Berlin.
All scientific sessions will take place in the Köpenpick Room.
Refreshments, posters and exhibit stands are located in the Pankow Room.

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World Obesity would like to thank The Dr Robert C & Veronica Atkins Chair in Childhood Obesity & Diabetes Fund for providing a number of registration and travel bursaries to participants.

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World Obesity

Address:

World Obesity Federation, Charles Darwin House, 12 Roger Street, WC1N 2JU, London, UK

Contact:

Natasha Joyner
enquiries@worldobesity.org

World Obesity Federation represents professional members of the scientific, medical and research communities from over 50 regional and national obesity associations. Through our membership we create a global community of organisations dedicated to solving the problems of obesity.

Our mission is to lead and drive global efforts to reduce, prevent and treat obesity. We collate, conduct and disseminate world-leading research into obesity, its impact, causes, treatment and prevention. We influence policy of academics, government and business at global, regional and national levels. We bring rigour, consistency and credibility to the field through educational programmes, practical training, publications, conferences and accreditation.

World Obesity offers an internationally recognised online obesity education programme for health professionals, providing evidence-based content developed by leading obesity experts.

www.worldobesity.org

SPONSOR AND EXHIBITOR PROFILES

German Association for the Study of Obesity (DAG e.V.)

Address:

Fraunhoferstr. 5, 82152 Martinsried, Germany

Contact:

Beatrix Feuerreiter
www.adipositas-gesellschaft.de

The German Association for the Study of Obesity (DAG e.V.) was founded in 1984 and has since represented the interests of German specialists active in the field of obesity nationally and internationally.

The German Association for the Study of Obesity (DAG e.V.) sees itself as an advanced, science-based professional association.

It organises major scientific conventions and heads a variety of working groups such as the study group AGA - Obesity in Children and Adolescents.

The goal pursued by the German Association for the Study of Obesity (DAG e.V.) and its members is to jointly propagate and put in practice the knowledge about obesity prevention and therapy and thus make an important contribution to the health status in the German population.

Sable Systems

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SCIENTIFIC PROGRAMME

Monday 29th June

	Start Time
Registration	07:45
Session 1: Overview and Scope of the problem *Session Chair Michael Goran*	**08:30**
Welcome & Overview *Michael Goran, University of Southern California, USA*	08:30
Welcome *Martin Wabitsch, Universitätsklinik für Kinder- und Jugendmedizin, Germany*	08:45
Global consumption patterns, policies, taxes and other issues *Barry Popkin, University of North Carolina, USA*	09:00
Effects of sugars on diabetes risk and fatty liver *Luc Tappy, University of Lausanne, Switzerland*	09:45
Refreshment Break	10:30
Session 2: Effects of Sugars on Obesity in Early Development *Session Chair: Kristina Rother*	**11:00**
Effects of sugars on obesity during development and early life *Michael Goran, University of Southern California, USA*	11:00
Development of sweet taste preferences *Julie Menella, Monnel Center, USA*	11:45
Lunch	12:30
Session 3: Effects of Sugars on the Brain *Session Chair: Luc Tappy*	**13:30**
Diet-induced obesity and the brain: Role of dietary sugars *Matthias Tschöp, Helmholtz Zentrum München, Germany*	13:30
Effects of sugars on the brain *Jonathan Purnell, Oregon Health & Science University, USA*	14:14
Sugars, reward, addiction *Serge Ahmed, Université de Bordeaux, France*	15:00
Refreshment Break	15:45
Session 4: Oral presentations from submitted abstracts Session Chairs: Kimber Stanhope & Richard Mattes	**16:15**
Networking reception in exhibition and poster area	18:00
Day 1 close	20:00

SCIENTIFIC PROGRAMME

Tuesday 30th June

	Start Time
Session 5: Effects of Sugars in the GI system *Session Chair: Jonathan Purnell*	08:45
Welcome & Recap *Michael Goran, University of Southern California, USA*	08:45
Effects of sugars on CV risk *Kimber Stanhope, UC Davis, USA*	09:00
Dietary sugars and gut health *Remy Burcelin, University of Toulouse, France*	09:45
Refreshment Break	10:30
Session 6: Non-Caloric Sweeteners *Session Chair: Barry Popkin*	**11:00**
Overview of existing non-caloric sweeteners *Kristina Rother, National Institute of Health, USA*	11:00
Interventions involving sugars reduction including use of non-caloric sweeteners *Richard Mattes, Purdue University, USA*	11:45
Lunch	12:30
Session 7: Oral presentations from submitted abstracts *Session Chair: Julie Menella & Remy Burcelin*	**13:30**
Refreshment Break	15:00
Session 8: Action and Next Steps *Session chair: Michael Goran*	**15:30**
Roundtable discussion Guided discussion on research priorities and needed solutions	16:15
Event close	17:00

SCIENTIFIC COMMITTEE & ABSTRACT REVIEWERS

Michael Goran (Chair)

University of Southern California, USA

Barry Popkin

University of North Carolina, USA

Martin Wabitsch

President, German Association for the Study of Obesity, Ulm, Germany

Luc Tappy

University of Lausanne, Switzerland

ROUNDTABLE DISCUSSION PARTICIPANTS

Dr. Michael Goran (Roundtable chair)

University of Southern California, USA

Dr. Barry Popkin

University of North Carolina, USA

Dr. Dietrich Garlichs

CEO, German Diabetes Association and Speaker of the German NCD Alliance

Dr. Jörg Spieldenner

Head of Public Health Nutrition at the Nestlé Research Center

Dr. Klaus Heider

Director-General for Food Policy, Product Safety and Innovation

Dr. Luc Tappy

University of Lausanne, Switzerland

Prof. Dr. Martin Wabitsch

President, German Association for the Study of Obesity, Ulm, Germany

SPEAKER BIOGRAPHIES

Barry Popkin

Barry M. Popkin, PhD, an economist and nutrition epidemiologist, W. R. Kenan, Jr. Distinguished Professor, University of North Carolina. He has developed the concept of the Nutrition Transition (studies on dynamic shifts in dietary intake, physical activity and obesity patterns and trends and impact on nutrition-related non-communicable diseases). His research program has included studies of added sugar in our global diet: analysis of US sales and purchases; beverage patterns and trends in many countries; two adult RCTs (the US and Mexico on water replacement of caloric beverages); and the design and evaluation of SSB taxes and other large-scale regulatory efforts.

Luc Tappy

Luc Tappy obtained his MD degree at the University of Lausanne in 1981, and was trained in the Department of Internal Medicine and the Service of Endocrinology, Centre Hospitalier Universitaire Vaudois (CHUV) and in the Diabetes section, Temple University Hospital, Philadelphia, PA.

Since 1988, he has been a senior researcher at the Institute of Physiology, Lausanne University School of Medicine and from 1991-1997 he received a Career Development Award from the Max Cloëtta Foundation to develop research projects in the field of human metabolism. His studies have focussed on nutrition, physical exercise and metabolism in healthy individuals as well as in various clinical conditions, such as diabetes, obesity, organ transplant patients and critically ill patients. In 2002, he was appointed full professor of physiology and associate physician at the Division of Endocrinology and Metabolism at the CHUV. He was invited professor at the Centre Hospitalier Sart Tilman in Liège, Belgium (1998-2001), and in the Department of Nutrition at the University of California at Berkeley (1995).

His present research is essentially aimed at identifying the environmental factors involved in the present epidemics of obesity and type 2 diabetes. A number of studies are being conducted to evaluate the role of dietary sugars (more specifically fructose in carbonated beverages) in the development of obesity and insulin resistance, while others are aimed at assessing and evaluating the role of sport and physical activity in the prevention of fructose-induced metabolic disorders. Luc Tappy has published more than 200 original articles and review papers in international scientific journals.

SPEAKER BIOGRAPHIES

Michael Goran

Dr. Goran is Professor of Preventive Medicine and Pediatrics at the Keck School of Medicine, University of Southern California in Los Angeles. He is the founding Director of the USC Childhood Obesity Research Center and holds the Dr Robert C and Veronica Atkins Endowed Chair in Childhood Obesity and Diabetes. Dr Goran also serves as Co-Director of the USC Diabetes and Obesity Research Institute. Dr Goran is a native of Glasgow, Scotland, and received his Ph.D. from the University of Manchester, UK (1986). Dr Goran's research has focused on the causes of consequences of childhood obesity for over 25 years. His work is focused on understanding the metabolic and nutritional factors linking obesity to increased disease risk during growth and development and using this information as a basis for developing interventions for prevention and risk reduction. Dr. Goran has published over 300 professional peer-reviewed articles and reviews, and is co-editor of the "Handbook of Pediatric Obesity" and "Dietary Sugar and Health" to be published in 2014, and is Editor-in-Chief for Pediatric Obesity. He has been the recipient of a number of awards including: The Nutrition Society Medal for Research (1996), The Lilly Award for Scientific Achievement in Obesity (2006), the Bar-Or Award for Excellence in Pediatric Obesity Research, (2009), and the TOPS award for contributions to obesity research from The Obesity Society (2014). Full details at www.GoranLab.com

Julie Menella

Dr. Julie A. Mennella obtained a Ph.D. from the Department of Behavioral Sciences at The University of Chicago in Chicago, IL. She joined the faculty at the Monell Chemical Senses Center in Philadelphia, PA 1990 where she is now a Member. Her major research interests include investigating the timing of sensitive periods in human flavor learning during breastfeeding and formula feeding; uncovering how children are living in different taste worlds than adults and their vulnerabilities to the current food environment; and understanding role of genetics, culture and experience on food choice and habits. Dr. Mennella is the recipient of grants from the National Institute of Deafness and Other Communication Disorders and the Eunice Kennedy Shriver National Institute of Child Health and Human Development.

Matthias Tschöp

Dr. Matthias H. Tschöp received his M.D. from Ludwig-Maximilians Universität in Munich. In 1999 he accepted an invitation for a postdoctoral fellowship at the Eli Lilly Research Laboratories in Indianapolis, USA. From 2002-2003 he established his independent research laboratory at the German Institute of Human Nutrition Potsdam-Rehbrücke and then returned to the United States, where he was a tenured Professor of Endocrinology and Diabetes at the Metabolic Diseases Institute of the University of Cincinnati. Until 2011 he was the Arthur Russell Morgan Endowed Chair of Medicine and Research Director of the University of Cincinnati's Metabolism Center of Excellence for Diabetes and Obesity. Since 2011 Prof. Tschöp has been the Research Director of the Helmholtz Diabetes Center and the Director of the Institute for Diabetes and Obesity at Helmholtz Zentrum München. In addition, he holds the Chair of the Division of Metabolic Diseases at Technische Universität München. Prof. Tschöp is the first German physician to receive the Alexander von Humboldt Professorship, the highest-endowed German research award (2012). In 2013 he became an elected member of the German National Academy of Science (Leopoldina).

Jonathan Purnell

Dr. Purnell completed his medical school training at Oregon Health & Science University (OHSU), his Internal Medicine residency at the University of Vermont, and Fellowship training in Endocrinology at the University of Washington. He is a Professor of Medicine with a joint appointment in the Knight Cardiovascular Institute and the Division of Endocrinology at the Oregon Health & Science University in Portland, OR, and sees patients with obesity, diabetes, and lipid disorders. His work as a physician scientist focuses on understanding the causes and consequences of obesity, metabolic syndrome, dyslipidemia, and diabetes in humans.

Serge Ahmed

Serge Ahmed is a CNRS Research Director at the University of Bordeaux, France. He has an internationally recognized expertise in experimental and computational models of addiction, including sugar addiction. He is best known for his work on animal models of escalation of substance use and transition to addiction. He has published several dozen articles, some of them in the most prestigious peer-reviewed scientific journals, including Science, Nature, Nature Neuroscience, and PNAS. He also serves as an expert reviewer for tens of journals and for several research agencies worldwide, including the French Agency for Food, Environmental and Occupational Health & Safety.

Remy Burcelin

I have been the head of my laboratory for the last 15 years leading research to decipher the molecular mechanism responsible for metabolic diseases. My laboratory is composed of 15-20 people including 6 post docs and several students/technicians. I further hired clinicians to allow the transfer from basic science to clinic. I have been coordinator of a European consortium on the gut microbiota to hepatic steatosis crosstalk including 9 partners. I have numerous international and national collaborations and numerous publications in the area of links between the gut microbiome and metabolic diseases of direct relevance to the current proposal. This work includes my involvement with some seminal work in this area that for example established the role of "metabolic endotoxemia" in the link between gut microbiota and metabolic dysfunction related to liver disease and type 2 diabetes risk. My current work at Vaiomer related to assessment of fecal and plasma metagenomic profile and links to metabolic diseases is ideally suited to serve as a consultant on this proposal.

Kimber Stanhope

Kimber Stanhope received an M.S. and Ph.D. in Nutrition from the University of California, Davis, where she is an Associate Nutritional Biologist Researcher in the Department of Molecular Biosciences. She conducts well-controlled clinical diet intervention studies to determine the effects of sugar consumption on the development of metabolic disease. The results from these investigations have attracted much attention from the scientific community (2009 Journal of Clinical Investigation publication cited 700+ times), the lay media (interviews with CBS 60 Minutes, CBC Fifth Estate) and health advocacy groups. She has first-authored ten review articles/book chapters on the metabolic effects of sugar consumption.

Kristina Rother

I completed medical school in Freiburg, Germany, and residency training in Pediatrics at Mayo Clinic, Rochester, Minnesota. Pediatric endocrine training took place at the Children's Hospital, Zurich, Switzerland, Massachusetts General Hospital, Boston, and again at Mayo Clinic. I spent years in basic research investigating insulin signaling, initially at the Institute for Molecular Biology II, Zurich, Switzerland, then at NIH. In 2011, I became section chief (Pediatric Diabetes & Metabolism, NIDDK, NIH). My current clinical research projects focus on metabolic effects of artificial sweeteners, side effects of oral hypoglycemic agents, and endocrine features of autoinflammatory diseases associated with lipodystrophy.

Richard Mattes

Dr. Mattes is a Distinguished Professor of Nutrition Science at Purdue University, Adjunct Associate Professor of Medicine at the Indiana University School of Medicine and Affiliated Scientist at the Monell Chemical Senses Center. Dr. Mattes is the Director of Purdue's Public Health Program and the Ingestive Behavior Research Center. He is on several editorial boards and an associate editor of the American Journal of Clinical Nutrition. His research focuses on the areas of hunger and satiety, regulation of food intake in humans, food preferences, human cephalic phase responses and the chemical senses. He has authored over 240 publications.

SPEAKER ABSTRACTS

Global consumption patterns, policies, taxes and other issues

Barry Popkin, University of North Carolina, USA

While sugar has long been a critical part of the global economy and is omnipresent in our food supply, the postWWII period has been a major surge in its use in processed foods and beverages and overall global consumption. Today sugar is found in much of the globes packaged and processed foods and beverages and represents a key source of calories in many countries. It is much clearer how much sugar is added to beverages due to measurement complexities; nevertheless close to 75% of US packaged and processed foods contain added caloric sweeteners, including large increases in fruit juice concentrate use. As a beverage, intake has peaked and is declining in selected high income countries but globally SSB intake is growing rapidly. After reviewing global and selected country patterns and trends of beverage intake, I focus on the total food supply and the role of added sugar in foods and beverages and then US intake patterns and the critical distributional patterns of added sugar consumption. Intake in the US and many other countries is highly skewed and it is important to understand the impact of added sugar of the top 40% of consumers is very high. It is expected that this skewed distribution might exist globally though little research has been done on this. Countries and smaller geographic units have begun to aggressively tax and regulate SSB's in particular but to date we have essentially ignored another emerging threat, fruit juice. Taxation impacts on consumption are reported when available.

Sugars, reward, addiction

Serge Ahmed, Université de Bordeaux, France

I will review research that tests the validity of the analogy between addictive drugs, like cocaine and heroin, and palatable foods, notably those high in added sugar (i.e., sucrose). Food desires are by far the most frequent and intense desires in human daily life. Current evidence, though still scant, shows that sugar and sweetness can induce reward and craving that are comparable in magnitude to those induced by addictive drugs. This evidence is now supported by recent experimental research on sugar and sweet reward in laboratory rats. Overall, this research has revealed that sugar and sweet reward can not only substitute to addictive drugs, like cocaine, but can even be more rewarding and attractive. At the neurobiological level, the neural substrates of sugar and vsweet reward appear to be more robust than those of cocaine (i.e., more resistant to functional failures), possibly reflecting past selective evolutionary pressures for seeking and taking foods high in sugar and calories. The biological robustness in the neural substrates of sugar and sweet reward may be sufficient to explain why many people can have difficultly to control the consumption of foods high in sugar when continuously exposed to them.

Effects of Sugars on diabetes risk and fatty liver

Luc Tappy, University of Lausanne, Switzerland

Suspicion that fructose-containing caloric sweeteners (FCCS) may cause metabolic diseases has gained wide attention in the scientific and in the lay press. It rests mainly from prospective cohort studies indicating that FCCS, and more specifically sugar-sweetened beverages (SSB), consumption is associated with weight gain over time.

Fructose is metabolized independant of insulin secretion by a specific set of enzymes expressed in splanchnic organs (gut, liver, kidney). Acute fructose ingestion elicits minor glycemic response compared to glucose or starch, and improves blood glucose profile and HbA1C in subjects with type 2 diabetes. A high fructose diet however increases liver VLDL-TG secretion, which rises the concern that it may in the long term stimulate ectopic lipid deposition and cause insulin resistance. Short term studies documented that a high fructose diet impairs hepatic, but not muscle insulin sensitivity, however.

More than total body fat, omental and intrahepatic fat depots are closely associated with insulin resistance. Whether specific nutrients, such as saturated fat, trans-fat, and sugars are involved in the development of omental and intrahepatic fat depots independently of excess energy intake remains an unsolved question. In healthy humans, intrahepatic fat concentrations increase to a similar extent with excess fat, fructose or glucose, while increasing dietary protein intake partially prevents this effect.

The large consumption of FCCS in the population, makes it one target for the prevention of metabolic diseases. Further intervention trials will however be needed to evaluate the effects of FCCS reduction in selected group of subjects

Effects of sugars on obesity during development and early life

Michael Goran, University of Southern California, USA

Current evidence suggests that high maternal consumption of fat promotes obesity and increased metabolic risk in offspring, but less is known about the effects of other potential nutrient obesogens. Widespread increase in dietary fructose consumption over the past 30 years is associated with chronic metabolic and endocrine disorders and alterations in feeding behaviour that promote obesity. Emerging studies indicate that consumptions of sugars and sugar sweetened beverages is beginning to occur early in life and this early life exposure is associated with increased risk of obesity by early childhood.

In addition, I will review the evidence linking dietary intakes of sugars, especially fructose with altered metabolism and early obesity in animal models and limited human studies. I will review the evidence suggesting that high fructose exposure during critical periods of development of the fetus, neonate and infant can act as an obesogen by affecting lifelong neuroendocrine function, appetite control, feeding behaviour, adipogenesis, fat distribution and metabolic systems. These changes ultimately favour the long-term development of obesity and associated metabolic risk.

Development of sweet taste preferences

Julie Menella, Monel Center, USA

The liking for all that taste sweet is universal and evident among children around the world. At birth, the ability to detect sweet tastes is functioning and interacting with systems controlling affect. The inborn preferences for sweet tastes attract them to mother's milk and even act as an analgesic. Children continue to prefer higher levels of sweet than adults, with preferences not declining to adult levels until mid-adolescence, which coincides with the cessation of physical growth. The level of sweetness most preferred by children has remained heightened relative to adults for nearly a decade, despite reductions in sugar, both consumed and in the food environment. Evolution has shaped the response to sweets during development and our sensory systems evolved to detect and prefer the once rare, calorie-rich foods that taste sweet. In this talk, I will summarize the convergence of scientific evidence that suggests that the preference for sweet tastes is largely a reflection of biology which in turn makes children especially vulnerable to our current food environment which provides a rich supply of foods and beverages high in added sugars. This review serves as a foundation for a discussion on motivational properties of sugars since beginning early in life, sensory experiences has far-reaching effects on behaviors including food preferences and reward.

Diet-induced obesity and the brain: Role of dietary sugars

Matthias Tschöp, Helmholtz Zentrum München, Germany

All metabolic processes, from single cell substrate oxidation to complex behaviors, are under the control of specific CNS circuits, aiming to maintain homeostasis. Afferent signals include gut hormones, adipokines and nutrient components, while efferent information primarily originates from the hypothalamic nuclei and involves components of the autonomic nervous system as well as the classic endocrine axes. We recently observed that diet-induced metabolic diseases, such as obesity and type 2 diabetes, are associated with (and preceded by) pathological processes in these hypothalamic control centers. Such pathophysiology concerns the hypothalamic cell matrix beyond key neuronal populations and includes astrocytosis, microgliosis, hypervascularisation as well as increased presence of pro-inflammatory cytokines. Specific targeting of such "hypothalamic inflammation" using novel gut-peptide based delivery of glucocorticoids to key metabolic disease regions improved both local pathophysiology and systemic metabolic health. Such a novel unimolecular dual agonism and steroid delivery approach may not only offer superior therapeutic option for at least some patient subpopulations, but also suggests a pathogenetic relevance for this novel hypothalamic syndrome.

Effects of sugars on the brain

Jonathan Purnell, Oregon Health & Science University, USA

In addition to acting as the primary fuel source for the brain, glucose and its metabolites play several key roles in the maintenance of neuronal integrity as well as whole body homeostasis. Following direct uptake into both neuronal and glial cells, glucose metabolism provides energy and ensures maintenance of dendritic shuttles that clear the synaptic cleft, readying the neuron for the next activation. Glucose levels can also be 'sensed' by the brain, triggering or inhibiting specialized neurons responding to changes in blood glucose levels that, in turn, effect changes in the regulation of glucose levels, feeding behavior, and energy expenditure. Probably the best characterized are the central nervous system responses during hypoglycemia that result in counter-regulatory stimulation of adrenergic and glucocorticoid systems. Alterations in neuronal cellular energetics are also thought to play a major role in central nervous system 'nutrient sensing' that governs food intake and body weight in response to changes in dietary intake of fats, protein, and sugars. Data from animal studies and a limited number of studies in humans suggest that greater exposure to the sugar fructose, compared to glucose, alters the normal brain sensing responses so as to lead to increases in food intake and body weight, lending credence to epidemiological evidence linking added sugars in food with population-based gains in body weight.

Effects of Sugars on CV Risk

Kimber Stanhope, UC Davis, USA

The impact of sugar consumption on the development of cardiovascular disease (CVD) continues to be a controversial topic. There is considerable epidemiological evidence suggesting that increased intake of added sugars, sucrose and/or high fructose corn syrup (HFCS), or sugar-sweetened

beverages is associated with dyslipidemia, metabolic syndrome, type 2 diabetes and CVD . Recent evidence from National Health and Nutrition Examination Survey III suggests that the higher the intake of added sugar, the greater the risk, and that the average level of added sugar consumption in the U.S. is associated with an 18% increase CVD mortality risk. To prove that this relationship between sugar and CVD is cause and effect, we need plausible mechanisms and evidence from diet intervention studies. The objectives of this presentation are to:

1. Present the plausible mechanisms by which fructose-containing sugars can promote the development of CVD.
2. Present the results from our recently-completed study in which young adults consuming beverages containing 0, 10, 17.5 and 25% HFCS exhibited dose response increases in risk factor for CVD.
3. Discuss the limitations of the direct experimental evidence.
4. Discuss the contradictive results from an industry-funded investigation with a similar study design.

There are plausible mechanisms by which fructose-containing sugars can promote the development of CVD. There is much evidence from diet intervention studies that sugar consumption increases risk factors for CVD. However, the limitations of these studies preclude definitive conclusions and allow the controversy to continue. The controversy is further fueled by the contradictive evidence from the industry-funded studies

Dietary sugars and gut health

Remy Burcelin, University of Toulouse, France

The last decade demonstrated the crucial role of gut microbiota on metabolic diseases. The major causal link was related to the impact of gut microbiota on the triggering of inflammation through bacterial determinants such as LPS and peptidoglycans as well as full tissue microbiota. The origin of the gut microbiota dysbiosis could be linked to changes in dietary habits. Recent data show evidence related to the important impact of dietary sugars on the control of gut microbiota. The consecutive impact of gut dysbiosis could be similarly linked to the triggering of inflammation leading to obesity and metabolic diseases. Blood and gut biomarkers related to changes in feeding habits can be identified and hence the impact of dietary sugars on gut dysbiosis can be evaluated to better prevent the occurrence of metabolic disease. Lactose and more recently sweeteners have been incriminated. Sugar impact on intestinal immune cells or the enteric nervous system could impact the overall energy metabolism through mechanisms requiring changes in gut microbiota. The precise mechanisms still remain to be identified but numerous efforts are ongoing and should benefit the overall food and pharmacology industries

Overview of existing non-caloric sweeteners

Kristina Rother, National Institute of Health, USA

Artificial sweeteners (herein called non-nutritive sweeteners, NNS) have been associated with multiple adverse health outcomes, such as obesity, metabolic syndrome and type 2 diabetes. Most of the evidence is derived from epidemiologic studies, which cannot establish causality. More recently, mechanistic studies have reported plausible reasons for the apparent paradox of "fewer calories and more obesity". These explanations include behavioral and cognitive mechanisms such as greater energy intake after 'saved' calories due to choosing artificial sweeteners and a disruption of the learned relationship between sweet taste perception and caloric intake. Furthermore, NNS have been shown to induce less of a central reward response compared to caloric sugars, potentially leading to continued seeking of palatable food. Furthermore, alterations of the gut microbiome have been reported after NNS exposure, promoting greater energy harvest. Based on in vitro studies, it has also been suggested that artificial sweeteners may up-regulate adipogenesis.

Another plausible explanation for the link between NNS consumption and obesity is stimulation of insulin secretion in response to binding of NNS to sweet taste receptors (T1R2/T1R3) on pancreatic beta-cells. This has been documented in vitro and is supported by findings in humans. Extra-oral sweet taste receptors are also found in the intestine, where they modulate various gut hormone responses, including glucagon-like-peptide 1 (GLP-1) secreted from enteroendocrine L-cells and gastric inhibitory peptide (GIP) secreted from enteroendocrine K-cells. In this presentation, we report the results of a randomized same-subject crossover study testing the effects of NNS on glycemia, insulin, and incretin responses in healthy adults.

Interventions involving sugars reduction including use of non-caloric sweeteners

Richard Mattes, Purdue University, USA

High intensity sweeteners (HIS) were introduced into the food supply more than a century ago. Their use has grown markedly over the last four decades, primarily in beverages, but increasingly in foods. Their primary intended function is to help moderate sugar and energy intake. Despite intensive study and a long history of use, questions about their safety and efficacy persist and have recently intensified. This is attributable to novel findings from rodents that HIS consumption paradoxically, reportedly, stimulates food intake and increases bodyweight. Additionally, there is new evidence that receptors mediating sweetness on the tongue are also present on intestinal enteroendocrine cells and may hold previously unsuspected effects on appetite, carbohydrate metabolism and chronic disease risk. A critical assessment of the recent rodent and receptor data will be presented. It is argued that the premise that feeding regulation hinges on sweet conditioning is questionable since, unlike the other macronutrients, there are no essential nutrient supplied by carbohydrate, it is not the most energy dense macronutrient and its intake was not high over much of human evolutionary time. Further, the experimental conditions employed to document the effects are of uncertain relevance to human exposures. A consideration of the evolving receptor literature indicates early findings in cell culture and animal models have not translated to humans under physiological conditions. Recent intervention trials confirm earlier conclusions from multiple reviews that HIS can aid weight management if used as a substitute for other energy sources that would ordinarily have been ingested.

ORAL PRESENTATION ABSTRACTS

DAY 1

Impact of Liquid Sugar Reduction on Behavioural and Brain Responses to Food Viewing

Camille Crézé[1], Marie-Laure Bielser[2], Jean-François Knebel[2], Vanessa Campos[1], Luc Tappy[1,3], Micah Murray[2,4], Ulrike Toepel[2]

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Obesity is increasingly considered as a brain disease in which homeostatic mechanisms regulating food intake are overridden by hedonic drives towards consumption of foods high in fat and sugar. How consumption of added sugars from sugar-sweetened beverages (SSBs) impact hedonic drives to foods remains unknown.

We studied 14 high SSB consumers (8 men, 6 women) before and after 3 months of SSB replacement by artificially sweetened equivalents. Participants performed behavioural pleasantness ratings on a 5-point scale when presented with colour images of solid foods differing in fat content (high- vs. low-fat) and taste quality (savory vs. sweet). Moreover, the spatio-temporal brain dynamics while viewing the food types were assessed using high-density electroencephalography (EEG) and electrical neuroimaging analyses.

Behavioural results show a significantly greater appreciation of savory foods, but decreased appreciation of sweet foods post-SSB replacement. The 3-month SSB replacement modified the initial stages of visual processing as well, in particular in response to low-fat foods. Therein, the neural activity when viewing low-fat/savory foods was reduced in the right inferior temporal gyrus and insula, i.e. brain areas involved in food categorization and valuation. Responses to high-fat foods seem less susceptible to (dietary) modulations, likely due to their greater salience and reward valuation.

Our results show that the replacement of liquid sugars by artificially sweetened equivalents differentially influences responsiveness to solid calories differing in taste quality, likely explaining changes in behaviour towards food intake. Whether these effects are due to sugar reduction or to artificial sweeteners per se remains to be investigated.

Including fruit juice concentrate as an added sugar substantially changes estimates of US added sugar purchasing; a case study of beverages in 2007-2008

Including fruit juice concentrate as an added sugar substantially changes estimates of US added sugar purchasing; a case study of beverages in 2007-2008

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Background: Globally, fruit juice concentrate (FJC) with its 'natural' connotations is globally uncounted in estimates of added sugars, despite being a caloric sweetener. Food industries have pushed to remove FJC from the definition of added sugars proposed in the US FDA Nutrition Facts Panel (NFP) revision. We examine the contribution of FJC in US beverages containing added sugars.

Methods: This analysis uses the 2007-08 Nielsen Homescan Consumer Purchase data on US household CPG purchasing, plus NFP and ingredient data from a number of sources. A linear programming model to estimate added sugar amounts has been applied to a subset of sweetened beverages (2007 n= 9,773, 2008 n=9,463). Amounts of FJC and total added sugars within unique products, and purchased by households were analyzed.

Results: Of the sweetened beverages examined in 2007-2008, 14% contained FJC each year. The mean amount of FJC in beverages varied by category, from <1g/100g of product to 4g/100g. In 2008, households with kids (<19y) purchased 12,292 kcal (34 kcal/day) in FJC, while households without kids purchased 6,526 kcal (18 kcal/day). Households with higher education heads and higher income also purchased fewer calories in FJC.

Conclusion: Globally FJC has been ignored as a major added sugar, despite increasing use. We showed that omission of FJC from the definition of added sugars for NFP labelling would omit a significant amount of calories purchased. By extension, this study suggests FJC deserves attention from the global public health community given the adverse health effects of added sugar, particularly in beverages.

No differential effect of beverages sweetened with fructose, high-fructose corn syrup, or glucose on systemic inflammation in healthy, normal weight or obese individuals: a randomized controlled trial

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Consumption of sugar-sweetened beverages (SSBs) is associated with type 2 diabetes and cardiovascular disease, both independently and through increased body weight. Chronic, low-grade inflammation is associated with these diseases and with obesity. Based on observational data suggesting SSBs promote inflammation, we conducted a randomized, controlled, double-blind cross-over intervention to determine whether fructose vs. glucose vs. high-fructose corn syrup (HFCS)-sweetened beverages differentially influence biomarkers of systemic inflammation. Based on rodent data showing that fructose triggers intestinal permeability, we also assessed whether SSBs influence biomarkers of gut barrier function. Subjects (24 healthy adults) consumed a standardized diet ad libitum for three 8-day dietary periods while drinking four servings per day of beverages sweetened with fructose, glucose, or HFCS (55% fructose, 45% glucose), comprising 25% of estimated calorie requirements (consumption mandatory). On average, subjects consumed 116% of their estimated calorie requirement while drinking these beverages, with no difference in total energy intake between groups. C-reactive protein (CRP) concentrations did not change significantly while subjects consumed the beverages ($p=0.353$). At the end of the fructose, HFCS, and glucose phases, neither CRP ($p=0.403$) nor interleukin-6 ($p=0.933$) differed from each other significantly. The lactulose-mannitol ratio suggested that intestinal permeability decreased following the HFCS period compared to the glucose and fructose periods ($p < 0.001$), while no difference was seen in zonulin ($p=0.366$) or lipopolysaccharide-binding protein ($p=0.387$), a measure of lipopolysaccharide exposure. This well-controlled intervention demonstrates that medium-term consumption of fructose- vs. HFCS- vs. glucose-sweetened beverages does not differentially affect biomarkers of systemic inflammation or intestinal permeability.

Are all sugars alike? Case of low-caloric and non-cariogenic sugars

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Public Health recommendations advise to reduce added sugars, defined as "all mono- and disaccharides added to foods during processing" in order to decrease non-communicable diseases and improve dental health. Our purpose was to evaluate whether decreasing the consumption of all mono- and disaccharides is relevant to health. The caloric content and in-vitro digestibility of >30 mono-and disaccharides were evaluated from literature search. Those with a caloric content of 4 kcal/g were automatically excluded from further evaluation, due to their obvious contribution to energy intake. Further literature evaluation of in-vivo digestibility, cariogenicity and other health effects was performed on the short list. Nine monosacharides and fifteen disaccharides of caloric content <4 kcal/g were evaluated. Their main sources included natural origin such as milk, cereals and fruits. Of particular interest, the monosaccharides allulose and tagatose had a caloric content <1.4 kcal/g estimated from in-vivo studies. They were shown to have a beneficial effect on tooth demineralisation, and reduction of post-prandial glycemia. Of the disaccharides, 13 had beta-linkages and were not digested but fermented, and hence had a default caloric value of 2 kcal/g. Limited in vitro data indicated that 3 disaccharides were cariogenic while 3 were not. In conclusion, in vivo data on low-caloric sugars is scarce. More knowledge is required in order to position these sugars as part of global sugar reduction strategies. However, some low caloric and/or not cariogenic mono- and disaccharides were identified. This raises the question whether they should be included in the "added sugars" definition or not.

Association for the Study of Obesity and National Alliance against Noncommunicable Diseases in Germany (German NCD-Alliance): actions to reduce sugar consumption

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Since more than a decade the Association for the Study of Obesity in Germany (DAG e.V.) is communicating the need for health-in-all-policies to shape healthier informational, nutritional and physically activating environments supporting healthier food preferences and lifestyles. In 2004, DAG joined the national platform on nutrition and physical activity. In 2007, DAG developed an Action Plan against Overweight, that later was partly implemented as the "national initiative IN FORM". Since 2008 DAG is advocating for the curbing of marketing of obesogenic food and beverages to children and the need for a colour-coded nutrition labelling front of pack. To enhance awareness and lobbying, in 2011, DAG joined the National Alliance against Noncommunicable Diseases (German NCD-Alliance) in the run up to the first UN-Summit on NCDs. DAG participated in WHO open consultations for the Global Action Plan on NCDS (2013-2020) and is advocating for a Global Convention to Protect and Promote Healthy Diets. Today the German NCD-Alliance comprises 17 health orientated NGOS and is supporting a sugar-tax, amongst other measures. In 2014, the German Federal Council recommended the implementation of a federal prevention law, a national diabetes plan and additional legislative measures to reduce the sugar content in food for younger children. However, a prevention law recently was drafted half-heartedly, with a focus on self-responsibility.

DAY 2

Ingestion of Sucralose and Acesulfame-Potassium Augments Glucose-stimulated Insulin and GLP-1 Secretion in Healthy Adults

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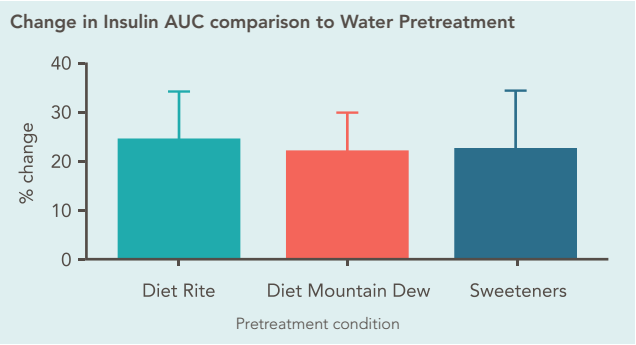
Aim: To test the acute metabolic effects of the non-nutritive sweeteners (NNS) sucralose and acesulfame potassium, specifically on incretin secretion and insulin response.

Methods: Thirty-one healthy adults aged 18-45 years participated in a four-period, same-subject cross-over study. Subjects consumed 12 ounces of commercially-available caffeine-free Diet Rite Cola™ (68 mg sucralose, 41 mg acesulfame-potassium), caffeine-free Diet Mountain Dew™ (18 mg sucralose, 18 mg acesulfame-potassium, and 57 mg aspartame) or 68 mg sucralose and 41 mg acesulfame-potassium in seltzer water (n=31) prior to 75g glucose. The order of pre-treatments was randomly assigned. Blood samples were collected prior to ingestion of the glucose load and serially for 120 minutes following ingestion.

Results: The combination of sucralose with acesulfame-potassium augmented insulin secretion (peak, Diet Rite Cola™ vs. seltzer water p=0.039; area under the curve (AUC) > 20% in all sweetener containing conditions vs. seltzer water) without altering glucose concentrations. Active GLP-1 was also elevated (Diet Rite Cola™ vs. seltzer water, AUC p=0.039; Diet Mountain Dew™ vs. seltzer water AUC p=0.07). No difference in glucose levels was observed following administration of diet soda nor sucralose and acesulfame-potassium in seltzer water, compared to the carbonated water control.

Conclusions: Sucralose and acesulfame-potassium in diet soda and in seltzer water augmented insulin and incretin response. We speculate that this may reflect a state of mild insulin resistance, and may ultimately promote food intake and weight gain if maintained with chronic NNS ingestion. Well-designed trials are required to elucidate the metabolic effects of prolonged NNS consumption.



Percent change in insulin area-under-the-curve is shown following ingestion of a preload of Diet Rite Cola™, Diet Mountain Dew™, or sucralose and acesulfame-potassium in seltzer water, when compared to a plain, unsweetened carbonated water control (100%). All preloads (diet sodas, sweeteners, or carbonated water control) were administered ten minutes prior to a glucose load and insulin was measured serially for two hours following glucose ingestion.

Effects of Roux-en-Y gastric bypass on sugar-induced hypertriglyceridemia

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Oral fructose increases postprandial blood triglyceride (TG) concentrations, possibly by stimulating intestinal de novo lipogenesis (iDNL) and decreasing extrasplanchnic TG clearance. We hypothesized that these effects of fructose may be abolished by Roux-en-Y-gastric bypass (RYGB) due to bypass of the proximal small bowel. To evaluate this hypothesis, we studied 8 patients 12-18 months after RYGB, and 8 age-, weight- and gender- matched controls (C). Each participant was studied on two occasions, over 6-hours after ingestion of 1) a protein/ lipid meal (PL), and 2) a protein/ lipid/ glucose/ ^{13}C-fructose meal (PLGF). Chylomicrons-triglyceride secretion and clearance were assessed from postprandial incremental areas under the curve for TG and apoB48. Sugar-related hyperlipemia was assessed from the difference between chylomicron-TG iAUC after PLGF and after PL. iDNL was assessed from postprandial 13C-chylomicrons palmitate AUC.

Postprandial chylomicrons-TG iAUCs were, in RYGB: 16.8 ± 7.0 mmol/L*360min after PL vs 6.4 ± 5.7 after PLGF (NS), and in C: 28.4 ± 12.7 after PL vs 67.9 ± 23.0 after PLGF (p = 0.025). Sugar-related hypertriglyceridemia was 2.1 ± 10.6 mmol/L*360min in RYGB vs 46.7 ± 15.0 mmol/L*360min in C (p = 0.03). ^{13}C-chylomicrons palmitate AUCs were similar in both groups. ApoB48 peaked earlier in RYGB than in C, but post-prandial iAUCs for apoB48 were not different in the two groups.

These results indicate that sugar induced hypertriglyceridemia was abolished after RYGB. This is not related to decreased iDNL, and suggests that postprandial chylomicron-TG clearance is increased after RYGB.

Isocaloric Fructose Restriction for 10 Days Improves Glucose Metabolism and Insulin Sensitivity in Obese Latino and African American Children

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Background: Some studies have shown that high sugar (specifically fructose) consumption is associated with increased risk for insulin resistance, but other studies did not show an association. The current study was designed to determine whether short-term fructose- but not calorie- restriction could improve parameters of glucose metabolism.

Design/Methods: Obese Latino and African American children (ages 9-18; BMI z-score 2.4±0.3; 47.0±4.9% fat [DXA]), who were high dietary sugar consumers at baseline (average fructose intake >50 g/day), had all meals provided for 10 days with the same energy and macronutrient composition as their standard diet, but with other carbohydrate substituted for fructose. Subjects were instructed to consume all food provided. Subjects were weighed daily and diets adjusted to maintain baseline weight. A 2-hour oral glucose tolerance test was performed on Days 0 and 10.

Results: Paired data are available in 43 participants (Latino 14F, 13M; African American 13F, 3M). Fasting glucose (-5.4%), insulin (-30.7%), HOMA-IR (-34.2%), and lactate (-29.8%) decreased significantly, as did glucose (-7.2%), insulin (-32%), and lactate (-19.4%) AUCs, with 10 days of fructose restriction (all P<0.001 [paired t-test]). All of these effects remained statistically significant after adjusting by ANCOVA for minor weight loss (0.9±0.3 kg, P<0.001).

	Day 0	Day 10
Fasting glucose (mmol/L)	5.4±0.5	5.1±0.4
Fasting insulin (pmol/L)	226±133	157±73
HOMA-IR	7.9±4.8	5.2±2.6
Glucose AUC/h	15.2±2.2	14.1±2.2
Insulin AUC/h	2,542±1,567	1,728±1,028
Fasting lactate (mmol/L)	1.21±0.38	0.85±0.26
Lactate AUC/h	2.67±0.58	2.15±0.58

Mean±SD

Conclusions: Isocaloric dietary fructose restriction for 10 days improved both fasting glucose and its clearance and the insulin response to a glucose challenge in Latino and African American children irrespective of weight loss.

Metabolic fate of a fructose load ingested before or after exercise

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Exercise prevents fructose-induced hypertriglyceridemia. To assess how exercise impacts fructose metabolism, we compared the metabolic fates of an oral 13C-labelled fructose load (OFL) consumed a) in resting, non-exercising condition, b) before and c) after an aerobic exercise.

8 healthy males were studied on 3 occasions after 4 days on a weight-maintenance, high-fructose diet. On the 5th day, they ingested an OFL (0.75g/kg) either a) in resting condition (C), b) with an aerobic exercise (FruEx; 60 min cycling at 100W) starting 90 min after fructose ingestion, or c) with the same exercise starting 75 min before ingestion (ExFru).

On each occasion, fructose oxidation (13CO2), non-oxidative fructose disposal (NOFD: fructose ingestion – 13C-fructose oxidation, corresponding to net glycogen storage), conversion of fructose into plasma glucose and plasma lactate concentrations were measured over 7 hours after fructose loading.

Fructose oxidation was significantly higher in FruEx than in C (80 ± 3% vs 49 ± 1% OFL; p<0.001), but was unaltered in ExFru (46 ± 1% OFL; p=0.14 vs C). Consequently, NOFD was significantly lower in FruEx than in C (20 ± 3% vs 51 ± 1% OFL; p<0.001) and in ExFru (54 ± 1% OFL, p<0.001 vs FruEx). Fructose conversion into glucose as well as plasma lactate concentrations remained unchanged in FruEx and ExFru, as compared to C.

We conclude that exercise performed immediately after fructose ingestion enhances fructose oxidation, while exercise performed before fructose ingestion does not significantly alter its metabolic fate. This work was supported by the Swiss National Science Foundation.

Children's liking for the taste of sucrose and nonnutritive sweeteners

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Carbohydrates are a source of energy often equated with sweetness, the detection of which is associated with powerful hedonic appeal, especially among children. Intakes of nonnutritive sweeteners (NNS), which provide sweetness with few or no calories, have risen consistently over the past two decades. Despite their prevalence in the food supply, there is a paucity of pediatric research on the behavioral and psychophysical evaluation of these ingredients. To this end, we used three methods (e.g. 3-point and 5-point hedonic face scales, and the general Labeled Magnitude Scale) to assess liking of varying concentrations of nutritive (sucrose) and nonnutritive (sucralose, aspartame, acesulfame potassium, and stevia) sweeteners in solution among 6- to 14-year-old children (N=48) and their mothers. Methods were identical for both age groups: subjects tasted each solution individually and in randomized order, indicated their hedonic rating, rinsed their mouth twice with water, and waited one minute before tasting the next solution. Analyses revealed that compared to adults, children more frequently liked the taste of both sucrose and NNS; however, both groups had essentially the same pattern of preferences for the sweeteners. In general, sweetener type was a much better predictor of its hedonic rating than was subject age. Findings set the stage for continued efforts aimed at better understanding the implications of NNS on dietary behavior of pediatric populations.

Increased Sugary Beverage Consumption is Related to Alterations in the Systemic Microbiome in Overweight and Obese Hispanic Children

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The human gut microbiome performs essential functions such as maintaining the intestinal barrier. Increased dietary sugar intake, especially fructose, can significantly alter the gut microbiota, which results in decreased epithelial tight junction integrity and bacterial translocation into the systemic circulation. This endotoxemia is thought to contribute to increased inflammation and insulin resistance. We examined interrelationships between dietary sugar intake, sugar sweetened beverage and juice (SSB/J) consumption, and the serum microbiome.

In sixty-seven Hispanics aged 8-15 years (BMI percentile: 95.4±9.5), dietary intake and SSB/J consumption was assessed using 24-hour dietary recalls. Serum microbiome was characterized using Illumina-based 16S rRNA sequencing. Microbial associations were examined using the non-parametric Kendall's tau test with p-values corrected by the Benjamini–Hochberg procedure for multiple testing.

Average SSB/J consumption was 2.0±1.5 servings/day. SSB/J consumption was associated with decreased microbial diversity at the phyla level (Shannon Diversity; $p=0.01$; Spearman $r=-0.39$) and increased Proteobacteria relative abundance ($p<0.05$; $r=0.30$). At the OTU level, there was a positive association ($p<0.006$; FDR-corrected $p=0.07$; $r=0.32$) between SSB/J consumption and an OTU that classifies to genus Sphingomonas within the Alphaproteobacteria. There were no significant associations at a 10% false discovery rate threshold between microbial diversity and other bacteria at the level of phyla or class or OTUs with other dietary sugars.

Increased SSB/J consumption may negatively alter the gut microbiome and/or gut bacterial translocation. These changes may decrease systemic microbial diversity and increase Proteobacteria, potentially contributing to obesity and metabolic disease. We are currently validating these intriguing results in a separate cohort.

POSTER PRESENTATIONS

All poster presenters are required to put their posters up on Monday 29th June during the first morning coffee break.

POSTER NUMBER	ABSTRACT TITLE	NAME
A66	Metabolism of Fructose in 3-dimensional brain cell cultures	Alexandra Broyer
A4	Association of sweet food and beverage intake with common mental disorder in Whitehall	Anika Knueppel
A64	Do young children already eat too much sugar?	Annett Hilbig
A8	The cost of policy inaction on sugar sweetened beverage consumption: Implications for obesity in South Africa	Aviva Tugendhaft
A13	A Policy Analysis of Product Reformulation Efforts to Reduce Sugar Consumption in the United States	Courtney Scott
A14	Sugar Industry Efforts to Undermine Evidence Linking Sucrose Consumption with Coronary Heart Disease	Cristin Kearns
A15	L-arginine supplementation improves insulin resistance in experimental model of obesity.	Damian Skrypnik
A70	Consumption of free sugars and micronutrient adequacy in the Netherlands	Diewertje Sluik
A16	Effectiveness of interventions to reduce sugar-sweetened beverage intake in children and adolescents.	Elisa Joan Vargas-Garcia
A65	Has school food legislation influenced socio-economic differences in sugar intake in children in Scotland?	Geraldine McNeill
A19	Is change in added sugar consumption associated with change in body mass index in older Australians?	Hanieh Moshtaghian
A21	An Exploratory Study on Dietary Intake among Adolescents in Malaysia: Outcome from the Malaysian Health and Adolescents Longitudinal Research Team Study (the MyHeART study)	Hazreen Abdul Majid
A22	Sweetness preference and its association with socio-demographic characteristics in Hong Kong children	Ho Sy
A26	Leptin suppresses development of glp-1 innervation to the hypothalamus	Jessica Biddinger
A27	Metabolic effects of aloe vera gel complex in obese prediabetes and early non-treated diabetic patients: Randomized controlled trial	Jin Ho Park
A58	Do beverages with low glyceamic index positively influence parameters of arterial stiffness in healthy men?	Judith Karschin
A57	Do low glycemic-sugar-sweetened beverages have a favorable effect on glucose metabolism and metabolic flexibility in healthy men?	Julia Kahlhöfer
A28	Prevalence and trend of metabolic risk factors and the metabolically healthy phenotypes among adults in Germany	Julia Truthmann
A69	Are South Indians heading towards an obesity epidemic? – Is it the Rice burden.	Lalitha Shivaprakash
A54	Beverage consumption and long-term association with body weight status in German adolescents - Results from the DONALD Study	Lars Libuda
A33	Fructose, glucose and sucrose profiles in sweetened beverages – focus on added sugars	Małgorzata Grembecka
A56	Consumption of sugar-containing soft drinks and change in anthropometric measures among adults in Germany	Marjolein Haftenberger
A34	Consumption of juice and carbonated drinks in relation to Body Mass Index among Kuwaiti children aged 2-5 years.	Maryam Al-Hilal

POSTER NUMBER	ABSTRACT TITLE	NAME
A35	Fructose, but not glucose, impairs insulin signaling in the three major insulin-sensitive tissues.	Miguel Baena
A38	High dietary fructose load affects fatty acid oxidation and lipogenesis without increasing lipid deposition in the liver of Wistar rat	Natasa Velickovic
A53	Prospective Association between Beverage Consumption and Body Weight Development among School Children: A Secondary Analysis of a Water Intervention Study	Rebecca Muckelbauer
A42	Lifestyle correlates of preference for sweet food in Hong Kong children	Ruolin RUAN
A44	Consumption of commercial energy drinks by children and adolescents: a systematic review of consumer attitudes and associations with health, behavioural and social outcomes	Shelina Visram
A45	Energy drinks: hype or hyper? A qualitative exploratory study involving children, parents and teachers from schools in North East England	Shelina Visram
A48	Effects of soft drinks and artificially sweetened drinks on hepatic fat content in overweight subjects	Vanessa Campos

POSTER PRESENTATION ABSTRACTS

Metabolism of Fructose in 3-dimensional brain cell cultures

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Whether fructose at physiological concentrations is metabolized in the brain, and hence may exert direct effects on brain function remain unknown. We specifically hypothesized that fructose may be converted into lactate and glucose, which are the main energy substrates for neurons. To assess this hypothesis, we incubated 3-dimensional brain cell cultures containing neurons and glial cells for 3 hours in a physiological buffer containing either 5 mM glucose, or 5 mM 13C-labelled fructose, or 1 mM glucose + 5 mM 13C-fructose, or 5 mM glucose + 1mM 13Cfructose, and monitored 13C-lactate and 13C-glucose released in the medium. Results are summarized below:

GC-MS analysis detected the presence of $^{13}C_3$-lactate and $^{13}C_3$-glucose, but not $^{13}C_2$ or $^{13}C_6$- glucose.

We conclude that fructose is converted into lactate and glucose in brain tridimensional cell cultures. Labelling pattern indicates that glucose is synthesized from trioses-phosphate rather than from pyruvate or lactate. We hypothesize that fructose conversion into lactate and glucose takes place in astrocytes, since these cells are known to express gluconeogenic enzymes and glucose-6-phosphatase. The functional significance of fructose metabolism in brain cells remains to be explored.

	Lactate concentration (mM)	^{13}C-Lactate IE (%)	^{13}C-Lactate concentration (µM)	^{13}C-Glucose IE (%)	^{13}C-Glucose concentration (µM)
5mM Glc	2.29±0.23	0.00	0.00	0.00	0.00
5mM Fru	0.28±0.07	2.01±0.68	5.46±1.49	ND	ND
5mM Glc + 1mM Fru	2.32±0.44	0.01±0.00	0.31±0.11	0.42±0.15	19.67±7.34
1mM Glc + 5mM Fru	1.26±0.16	0.47±0.12	5.83±1.45	27.28±6.10	87.90±30.30

ND: not detectable

Association of sweet food and beverage intake with common mental disorder in Whitehall II

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Background: Dietary patterns have been linked with depressive symptoms in several populations, but the active ingredients are unknown. In an exploratory cross-sectional analysis of links between diet and common mental disorder (CMD; symptoms of depression and anxiety) we found associations with consumption of sweet foods and beverages.

Aim: To investigate systematically the cross-sectional and prospective associations between sweet food and beverage intake and CMD.

Methods: We analysed repeat measures data from the Whitehall II cohort study. In a cross-sectional analysis 18,710 person-observations were included, 12,864 in prospective analysis. Common mental disorder was measured with the 30-item General Health Questionnaire (GHQ). Dietary assessment by food frequency questionnaire was available at three phases. Random effects logistic regressions were performed to estimate cross-sectional and prospective associations of sugar intake from sweet food/beverages and GHQ caseness. Both models were adjusted for sex, age and ethnicity.

Results: The cross-sectional analysis showed that intake of over 50g sugar per day from sweet food/beverages was associated with a 14% higher odds of being a GHQ case (OR: 1.139, 95%-CI: 1.005; 1.292), compared to consuming less than 25g per day. In prospective analysis, after exclusion of baseline cases, and with the same exposure contrast, the odds of incident GHQ caseness 5 years later was increased by 21% (OR: 1.210, 95%-CI: 1.036; 1.414).

Conclusions: The cross-sectional and prospective associations observed suggest there may be an adverse effect of sweet food/beverage intake with long-term psychological health. Weight gain may play an intermediate role in this diet-health association.

Do young children already eat too much sugar?

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Objectives: Recently, new population based guidance on the consumption of free sugar (added + naturally occurring sugar in fruit juices and concentrates) set an upper limit of 10% of total energy (E%). A further reduction to below 5% was suggested as conditional recommendation (WHO 2014). The aim of this analysis was to describe the intake of free and added sugar and to specify the relevant food sources in German infants and toddlers.

Methods: Data from the ongoing Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) Study and the cross-sectional German Representative Study of Toddler Alimentation (GRETA) were used.

2312 3-day-weighed-dietary-records from DONALD (2004-2014) and 525 7-day-estimated-records from GRETA (2008) from participants aged 3-36 (DONALD) and 10-36 months (GRETA) were analyzed.

The intake of added sugar (mono-, disaccharides) and the percentage of food sources were determined, stratified by age. In the DONALD Study sample, the intake of free sugar was specified, conforming to WHO.

Results: The Overall mean intake of added sugar ranged between 10-12 E% in GRETA and increased with age from 0.5 to 1.8 E% in DONALD. Also, the intake of free sugar increased from 0.5 to 16.9 E% in DONALD. The main food contributors to added sugars were milk(products) (49-29%), confectionary and cakes (37-55%) and soft drinks (6-9%).

Conclusion: With the introduction of family food around the age of 10-18 months, the intake of free sugar exceeds the maximum WHO limit of 10 E%. The conditional limit of 5 E% is by far exceeded even by added sugar alone.

The cost of policy inaction on sugar sweetened beverage consumption: Implications for obesity in South Africa Abstract

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Background: Over the past decade the prevalence of obesity has increased in South Africa, as have the sales and availability of sugar sweetened beverages (SSBs). Excess sugar consumption is associated with weight gain and increased risk for non---communicable diseases (NCDs). Soft drink sales in South Africa are projected to grow between 2012 and 2017 at an annual compounded growth rate of 2·4% in the absence of regulations.

Aim: We estimated the effect of increased SSB consumption on future adult obesity prevalence in South Africa in the absence of any regulatory measures.

Methods: A model was constructed to simulate the effect of a 2··4% annual increase in SSB consumption on the obesity prevalence. The model computes the change in energy intake assuming a compounding increase in SSB consumption. The population distribution of body mass index by age and gender was modelled by fitting measured data from the 2012 South African National Income Dynamics Survey to the lognormal distribution and shifting the mean values.

Results: A 2··4% annual growth in SSB sales alongside population growth and aging will result in an additional 1287000 obese adults in South Africa by 2017, 22% of which will be due to increased SSBconsumption.

Conclusion: In the absence of preventive measures South Africa will face a much greater challenge in the future in addressing obesity and related NCDs.

Keywords Obesity; NCDs; Policy; Nutrition; Sugar sweetened beverages; South Africa

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A Policy Analysis of Product Reformulation Efforts to Reduce Sugar Consumption in the United States

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Product reformulation, the reduction or removal of nutrients such as fat, sugar, and salt from foods and beverages, is a topic of increasing prominence in nutrition policy. While there are success stories with reformulation, particularly for salt in the UK and Finland, efforts around sugar have been variable and limited. Product reformulation has been praised as a rare example of a "win-win" for the food and beverage industry and public health efforts to reduce obesity and non-communicable diseases (NCDs), however reformulation initiatives in the US have also been for criticized for being largely voluntary and self-regulated.

To better understand the political context of sugar reformulation, and inform on-going public health efforts to reduce NCDs, we examined rhetoric, narratives and discourse in responses (n=65) to a United States Department of Agriculture consultation on product reformulation (Dietary Guidelines Advisory Committee consultation 2.1).

A consistent narrative and discourse is identifiable in food and beverage industry responses: that reformulation is "part of the solution" to NCDs, even though the product or industry sector is not a large contributor to NCDs, and that progress has been made in the face of significant limitations and risks of reformulating well-known products, particularly for sugar reduction. The responses from other stakeholders did not contain a unified narrative. The food and beverage industry's consistent use of narratives and discourse around product reformulation may indicate a political strategy to avoid mandatory regulation of their products. The potential influence of these narratives on the nutrition policy landscape requires further research.

Sugar Industry Efforts to Undermine Evidence Linking Sucrose Consumption with Coronary Heart Disease

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Background: Epidemiological and biological evidence that sucrose consumption elevates serum lipid biomarkers for coronary heart disease (CHD) began accumulating in the 1950s, but the role of added sugars in CHD remains controversial.

Methods and Results: We reviewed 350 internal Sugar Research Foundation (SRF) documents from 1959-1971 for relevance to heart disease research and policy. SRF adopted a strategy to counter research demonstrating that when starch replaced sugar in the diet of rats and man, serum cholesterol, serum triglyceride, and liver fat levels decreased. SRF tactics included: using consultants to evaluate heart disease research, monitoring scientists producing unfavorable results, cultivating relationships with government and American Heart Association advisors, and funding research. Between 1966 and 1971, SRF allocated $80,000 ($577,000 in 2015 dollars) to 8 heart disease research projects. Sixteen publications were produced that have been cited 558 times between 1968 and 2014. SRF findings were used to support its position that reductions in serum lipid levels achieved when starch replaced sugar were insignificant compared to those obtained by decreased saturated fat intake.

Conclusions: Between 1959 and 1971, SRF developed a heart disease research program for the purpose of protecting industry interests, designed to influence policymakers evaluating the body of evidence on diet and heart disease. Despite the 2015 U.S. Dietary Guidelines Advisory Committee conclusion that

moderate evidence indicates that higher intake of added sugars is associated with increased risk of hypertension, stroke, elevated serum triglycerides and CHD, the sugar industry continues to advocate that added sugars are not associated with CHD.

L-arginine supplementation improves insulin resistance in experimental model of obesity

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Aim: To determine the impact of L-arginine supplementation, the substrate to nitric oxide production, on adiponectin concentration in rats on a high-fat diet. The connection between intake of L-arginine and insulin resistance was evaluated.

Materials and Methods: 36 Wistar rats were divided into three groups: group 1 was fed standard diet, group 2 high-fat diet, group 3 high-fat diet supplemented with L-arginine. After 42 days serum lipid profiles, glucose, insulin, adiponectin and NO concentration were measured, insulin resistance (IR) was estimated using homeostasis model assessment (HOMA).

Results: At both the beginning and the end of the study, body mass was equal in all 3 groups. In group 2, visceral fat was greater after 42 days. In group 3, there was a downward tendency for visceral fat levels. Increases in triglicerydes, cholesterol, insulin concentration and HOMA–IR, decreases in NO and adiponectin concentration were seen in group 2, while in group 3, L-arginine ameliorated these disturbances.

Conclusions: Our study demonstrated that supplementation of L-arginine to high-fat diet in rats results in improvement of insulin sensitivity. Our findings suggest that the underlying mechanism could be associated with increased concentration of adiponectin.

Key words: obesity, insulin resistance, high-fat diet, nitric oxide, L-arginine supplementation

Consumption of free sugars and micronutrient adequacy in the Netherlands

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Aim: A high (free) sugar consumption has been associated with micronutrient dilution of the diet, especially in children and persons with a low energy intake. We estimated free sugar intake and micronutrient adequacy in a representative sample of the Dutch population.

Methods: A total of 3,817 persons (7-69 years) from the Dutch National Food Consumption Survey 2007-2010 were included. Diet was assessed with two 24-hour recalls. Free sugars are all sugars added to foods by the manufacturer, cook, or consumer, and sugars naturally present in honey, syrups, fruit juices, and fruit concentrates. Micronutrient dietary reference intakes (DRI) from the Dutch Health Council were used.

Results: Median free sugar intake was 89-113 g/d (18-21 en%) in children (7-18 years) and 53-85 g/d (11-14 en%) in adults (19-69 years). In children, 0% had a free sugar intake <5 en% and 5% had an intake <10 en%. In adults, 4% had a free sugar intake <5 en% and 29-33% an intake <10 en%. In general, micronutrient intake was highest with a free sugar intake between 10-20 en%. Adults with a free sugar intake >25 en% showed lowest adherence to micronutrient DRI. In children, micronutrient adequacy was lowest with a free sugar intake <10 en%.

Conclusions: Adherence to the WHO guideline of <10 and <5 en% free sugars was generally low. No large differences in micronutrient adequacy were observed across levels of free sugar intake. Moreover, no consistent trends in adequacy were shown for the different micronutrients, and across age and sex categories.

Keywords: free sugars, micronutrient intake, micronutrient adequacy, the Netherlands.

Effectiveness of interventions to reduce sugar-sweetened beverage intake in children and adolescents

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Evidence is growing linking higher intakes of sugar-sweetened beverages (SSBs) with increased risks of obesity, type 2 diabetes and cardiovascular disease. As a result, there has been much interest in targeting SSBs across public health interventions. Emphasis has been placed on childhood, as it represents a

critical period where dietary habits are developed, reinforced and permanently established. Consequently, a systematic review and meta-analysis has been undertaken on the impact that initiatives to reduce SSBs have had on consumption in childhood, and other age groups. A search strategy was developed and executed in 6 databases so that studies published after 1990 in any language, that had a control group available and that reported changes in daily intake of SSBs were retrieved, screened and analysed for internal and external validity. Quality appraisal was assessed in duplicate following Cochrane's principles. From a total of 5461 original records, 201 full papers were obtained, and 34 met the inclusion criteria. Only 16 studies, involving 10,110 participants, had complete data for meta-analysis on SSB intakes in children (< 18 years old). Overall, interventions significantly decreased consumption of SSBs by **74 millilitres/day (95% CI: -132, -16 millilitres/day) compared to controls.** Heterogeneity was high across studies (I^2 96%), and this was explained after subgroup analyses were conducted on potential confounders (setting of interventions, randomisation and use of certain behaviour change techniques as rationale). Results here highlight the need to support initiatives targeting SSBs in younger populations, as one way of addressing a modifiable dietary behaviour linked with obesity.

Has school food legislation influenced socio-economic differences in sugar intake in children in Scotland?

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In the UK the overall prevalence of child obesity has decreased a little but the disparity between socio-economic groups is increasing. Two national surveys are used here to investigate socio-economic differences in children's diet in Scotland before and after the introduction of legislation on sale of HFSS foods and drinks in schools.

Children aged 3-17y across Scotland were identified through national child welfare records. Intake of specific food groups and of energy and non-milk extrinsic sugars (equivalent to added sugars) was assessed by a food frequency questionnaire. Socio-economic differences were assessed by comparison across quintiles of the Scottish Index of Multiple Deprivation.

1,700 (68%) children took part in 2006 and 1,906 (63%) in 2010. Intake of energy and non-milk extrinsic sugars (% energy) was higher among children living in more deprived areas in both years. The differences in energy intake increased but differences in non-milk extrinsic sugars intake (% energy) decreased between 2006 and 2010 with a greater reduction in the intake of sugar-sweetened soft drinks, confectionery and crisps and savoury snacks and a greater increase in vegetable intake among children in more deprived areas. This could reflect an impact of the change in the food retail environment in schools.

Is change in added sugar consumption associated with change in body mass index in older Australians?

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Aim: To assess the association between changes in added sugar (AS) intake and body mass index (BMI) in a cohort of older Australians during 15 years of follow-up.

Methods: Dietary intake and BMI data were collected from participants of the Blue Mountains Eye Study (aged ≥49 years), in five-year intervals between baseline (1992-1994) and follow-up (2007-2009). A 145-item food frequency questionnaire (FFQ) was used for dietary data collection and AS content of FFQ items were determined using a stepwise method (e.g. recipes, food labels and other estimation methods). Complete data at all-time points were available from 358 women and 259 men. A Generalized Estimating Equations model was used to investigate the association between change in AS intake and in BMI, adjusted for baseline age, BMI, and changes in energy, fat and natural sugar intake.

Results: Mean (SD) AS intake for women at baseline and each successive follow-up were 40.0 (29.0), 41.7 (26.9), 42.0 (25.4) and 43.3 (26.1) grams (P for trend <0.01), and for men were 55.4 (33.5), 53.9 (32.3), 53.9 (30.6) and 50.8 (30.4) grams (P for trend=0.318), respectively. Baseline mean (SD) BMI in women and men were 26.1 (4.3) and 26.0 (3.3) kg/m^2, respectively. In both groups, mean BMI at each successive follow-up was higher than baseline (P for trend <0.05). There were no significant associations between changes in AS intake and BMI in women (ß=0.003, P=0.379) and in men (ß=0.002, P=0.658).

Conclusion: During 15 years of follow-up, no association was observed between changes in AS consumption and BMI in participants of this Australian cohort.

An Exploratory Study on Dietary Intake among Adolescents in Malaysia: Outcome from the Malaysian Health and Adolescents Longitudinal Research Team Study (the MyHeART study)

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Background: Optimum nutrition is essential for healthy growth in adolescents.

Aim: The study aims to investigate the baseline dietary intakes of the Malaysian adolescent cohort.

Methods: The study population consisted of schoolchildren who were attending one of the 15 public secondary schools from the central and northern region of the Peninsular Malaysia. Qualified dietitians conducted a 7-d diet history of habitual food intake in 837 adolescents. Detailed information on the portion sizes and meal contents were recorded and facilitated by flipcharts and household measurement tools. Nutritionist Pro™ Diet Analysis software was used to analyze the diet record.

Results: Mean age of adolescents was 13 y. Mean energy intake was 1670.8 ± 329.6 kcal/d. Males had significantly higher energy intake than females (1791.1 ± 329.2 vs 1598.5 ± 308.3 kcal/d). Adolescents in rural schools consumed more energy, fat and cholesterol compared to adolescents in urban schools (1718.9 ± 356.1 kcal/d, 56.6 ± 15.9 g/d and 246.8 ± 98.0 g/d, respectively). Sugar intake of overweight/obese adolescents (36.4 ± 18.7 g/d) was significantly higher ($P < 0.001$) compared to normal weight adolescents (31.9 ± 17.1 g/d).

Conclusions: Adolescents in rural schools had unhealthy dietary intake compared to adolescents in urban schools with higher energy intake of fat and cholesterol. Overweight/ obese groups consumed higher sugar compared to the normal weight adolescents. A structured, tailored intervention should be implemented to improve adolescent dietary intake and narrow the nutritional gap between adolescents in the urban and rural areas.

Sweetness preference and its association with socio-demographic characteristics in Hong Kong children

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Aim: Sweetness preference has been associated with socio-demographic characteristics, although the results were inconsistent. We assessed the prevalence of sweetness preference in Hong Kong children and identified its socio-demographic correlates.

Methods: Socio-demographic and lifestyle profiles of 106127 Grade 4 students (mean age 9.9±0.7, 50.9% boys) were recorded by the Student Health Service in 1998-2000. Sweetness preference was assessed by a 4-option questionnaire item "My attitude towards sweet food is:". A binary logistic regression model was fitted to investigate the association of sweetness preference with 8 socio-demographic factors.

Results: The prevalence of the 4 options was: "I like them very much" (denoting sweetness preference, 37.5%), "they are acceptable" (49.5%), "I'll try a little" (8.7%) and "I dislike them" (4.3%). Younger students and girls (adjusted odds ratio 1.03, 95% CI 1.00-1.06) were more likely to have a sweetness preference. Fathers with primary or below education (vs tertiary; 1.14, 1.08-1.21), and with occupations of clerk/service industry (vs manager/professional; 1.21, 1.12-1.31), and mothers with occupations of manual work (vs manager/professional; 1.18, 1.10-1.27) were associated with sweetness preference. No significant association between maternal education and sweetness preference was observed. Public or temporary housing (vs private housing; 1.11, 1.08-1.14) also predicted sweetness preference. However, sweetness preference was associated with living in districts with higher median income (highest vs lowest quartile; 1.06, 1.02-1.09).

Conclusions: Younger age, being girls, and lower parental socioeconomic status in general were associated with sweetness preference in Hong Kong children.

Leptin suppresses development of GLP-1 innervation to the hypothalamus

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Childhood obesity has increased in the United States and internationally, which sets up young people for a lifetime of devastating and costly health problems. Neural networks that regulate metabolic homeostasis must develop appropriately early in life in order to function properly. Neural circuits that develop under abnormal environmental conditions can become disrupted and no longer function normally, which can lead to obesity and diabetes. The nucleus of the solitary tract (NTS) receives and integrates visceral nutritional status, and primarily sends this information to the paraventricular nucleus of the hypothalamus (PVH), which coordinates neuroendocrine responses like glucose regulation. Leptin receptors (LepRb) in NTS neurons have been shown to impact food intake and glycemic state. In addition to leptin's role in ingestive behavior, it functions as a neurotrophic factor in development and is required for axon outgrowth within the hypothalamus. Because leptin receptors are also located in NTS neurons (including a large proportion of GLP-1 neurons that regulate glucose homeostasis), the requirement of leptin for development of GLP-1 innervation to the PVH was determined. Using genetically targeted labels and immunohistochemistry, we determined 100% of GLP-1 neurons in the NTS were responsive to leptin during development. GLP-1 neurons in the NTS and their projections in the PVH were quantified in obese leptin-deficient $Lep^{ob/ob}$ mice in development and adulthood. $Lep^{ob/ob}$ mice showed increased GLP-1 innervation in the PVH compared with wt mice, with no resulting changes in GLP-1 neuron number. These results suggest leptin's normal function is to suppress GLP-1 innervation from the NTS.

Metabolic effects of aloe vera gel complex in obese prediabetes and early non-treated diabetic patients: Randomized controlled trial

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Objective: The metabolic effects of an aloe vera gel complex (Aloe QDM complex) on people with prediabetes or early diabetes mellitus (DM) are unknown. The goal of this study was to determine the effects of Aloe QDM complex on body weight, body fat mass (BFM), fasting blood glucose (FBG), fasting serum insulin, and Homeostasis Model of Assessment - Insulin Resistance (HOMA-IR) in obese individuals with prediabetes or early DM who were not on diabetes medications.

Methods: Participants (n = 136) were randomly assigned to an intervention or a control group and evaluated at baseline and at 4 and 8 wk.

Results: The study lost six participants in the control group and eight in the intervention group. At 8wk, body weight (P = 0.02) and BFM (P = 0.03) were significantly lower in the intervention group. At 4 wk, serum insulin level (P = 0.04) and HOMA-IR (P = 0.047) were lower in the intervention group; they also were lower at 8 wk but with borderline significance (P = 0.09; P = 0.08, respectively). At 8 wk, FBG tended to decrease in the intervention group (P = 0.02), but the between-group difference was not significant (P = 0.16).

Conclusion: In obese individuals with prediabetes or early untreated DM, Aloe QDM complex reduced body weight, BFM, and insulin resistance.

Do beverages with low glyceamic index positively influence parameters of arterial stiffness in healthy men?

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Objective: Epidemiological studies have shown a relationship between sugar-sweetened beverage consumption and cardiovascular risk. When compared to high glyceamic index (GI) sugars, low-GI isomaltulose may have favorable effects on arterial stiffness.

Methods: In a 2x2wk controlled cross-over dietary intervention (55% CHO, 30% fat, 15% protein), 13 healthy physically active men (age: 24±2y, BMI: 23.6±2.0kg/m2) were investigated after 1wk of habitual physical activity (steps/d) followed by 1wk of reduced physical activity (steps/d) with either low or high GI beverages (isomaltulose vs. maltodextrin-sucrose, 20% energy requirement, adapted for sweetness). Measures of arterial stiffness were assessed fasting and 3h postprandially (meal test: 37g isomaltulose vs. 28g maltodextrin+9g sucrose): central pulse pressure (cPP), augmentation index (AIx) and carotid-femoral pulse wave velocity (PWV_{cf}).

Results: In fasting conditions, no differences in all parameters of arterial stiffness were found between GI-groups (all $p>0.05$). Postprandially, prolonged arterial relaxation by AIx was observed in response to isomaltulose compared to maltodextrin-sucrose after the inactive phase (isomaltulose: Δ60min: -2.6 ±3.3% and Δ180min: -3.5 ±2.7% both $p<0.05$ vs. baseline; maltodextrin@ sucrose: Δ60min: -3.7 ±3.2%; Δ180min: -1.7 ±3.6%, only Δ60min $p<0.05$ vs. baseline). No inactivity-induced changes in parameters of arterial stiffness were observed. However, higher step reduction was associated with an increase in PWV_{cf} ($r=-0.58$) and cPP ($r=-0.60$), as well as a decrease in diastolic blood pressure ($r=0.73$) during maltodextrin-sucrose beverage intake (all $p<0.05$).

Conclusion: 7-days of reduced physical activity with high-GI sugar-sweetened beverages does not impair basal vascular function in healthy young men. However, low-GI isomaltulose-sweetened beverages lead to prolonged postprandial arterial relaxation.

Do low glycemic-sugar-sweetened beverages have a favorable effect on glucose metabolism and metabolic flexibility in healthy men?

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Objective: Sugar-sweetened-beverages are the principle source of added sugar intake and have been shown to impair insulin sensitivity and promote weight gain. Beverages sweetened with low glycemic index (GI) sugars may favorably affect glucose metabolism and facilitate maintenance of metabolic flexibility (ability to switch between fat and carbohydrate oxidation).

Methods: In a controlled cross-over dietary intervention 13 healthy men (age: 23.7±2.2y, BMI: 23.6±1.9kg/m²) consumed low-GI (isomaltulose) or high-GI (maltodextrin-sucrose) sugar sweetened beverages providing 20% of energy requirement for 7 days. Participant´s habitual high physical activity (11,375±3124 steps/d) was reduced during study period (2,363±900 steps/d). The provided *ad libitum* diet comprised 55% CHO, 30% fat and 15% protein. Daylong glycemia was assessed over 7-d as area under the interstitial glucose curve. Measures of Insulin sensitivity (basal: HOMA-IR, postprandial: Matsuda-ISI) are available in a subgroup (n=5) yet. Metabolic flexibility was assessed (i) basal as fasting respiratory quotient and (ii) postprandial as capacity to increase CHO oxidation (incremental area under the RQ-curve) during oral glucose tolerance test (OGTT) and during meal test (37g isomaltulose vs. 28g maltodextrin+9g sucrose, adapted for sweetness).

Results: Daylong glycemia was lower with low-GI compared to high-GI sugar (-8%, $p<0.05$). Insulin sensitivity tended to be better with low-GI sugar (ΔHOMA-IR: 27%, $p=0.11$; ΔMatsuda-ISI: 21%, $p=0.29$). No difference between high-GI and low-GI sugar was observed in fasting ($p=0.69$) and postprandial metabolic flexibility ($p=0.79$ for OGTT and $p=0.64$ for meal tests).

Conclusion: In healthy men 7-day consumption of low-GI versus high-GI beverages during physical inactivity did not favourably affect metabolic flexibility.

Prevalence and trend of metabolic risk factors and the metabolically healthy phenotypes among adults in Germany

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Objective: To identify the metabolically healthy (MH) phenotypes in Germany and to investigate the prevalence and trend from 1997-99 to 2008-11.

Design and Methods: Data from 6,100 participants of the first national health survey GNHIES98 and 6,818 participants of the subsequent DEGS1 survey (2008-11) aged 18-79 years were analysed. MH was defined as none of the following criteria of the metabolic syndrome (MetS): serum glucose≥5.6mmol/l, blood pressure≥120/80mmHg, triglycerides≥1.7mmol/l, HDL≤1.03/1.30mmol/l (women/men). Previous diagnosis of diabetes, hypertension or dyslipidaemia or medication use was counted as fulfilled criterion. MH phenotypes were defined using abdominal obesity (waist circumference, WC): WC<80/94cm, 80/94<WC≤88/102cm, WC≥88/102cm (women/men).

Results: The most frequent exceeded MetS parameter was blood pressure. The prevalence of elevated blood pressure decreased from 60% to 54% among women and elevated triglycerides decreased from 51% to 49% among men (both $p<0.001$). The prevalence of low HDL cholesterol increased among women (40% to 45%; $p=0.028$) and decreased among men (44% to 42%; $p=0.046$). The prevalence of high serum glucose was unchanged. The prevalence of MH was higher among women compared to men (GNHIES98: 25% vs. 12%; DEGS1: 28% vs. 13%). The prevalence of MH was unchanged, except for women with WC<80cm the prevalence increased from 41% to 47% ($p=<0.001$).

Conclusions: Only one of four women and one of eight men is metabolically healthy. During the last decade, the prevalence of MH among women with abdominal obesity and men was unchanged. These findings have high public health implications as the majority of the population is still under metabolic risk.

Are South Indians heading towards an obesity epidemic? – Is it the Rice burden

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Aims and objectives: Purpose of the present study was evaluating the nutritional burden specifically the white rice leading to the very high prevalence of obesity and abdominal obesity in the diabetic population.

Methods: This study was held at Karnataka Institute of Diabetology, Bengaluru, during the period of 2013-2015. A total of 23417 diabetic adults were studied. Body weight, height, waist and hip circumference were measured and recorded.

Diet dairy for 3740 people was recorded and the total amount of rice and other cereals,number of sevings of fruits and vegetables were recorded. The average consumptions of proteins in the form of meat or vegetarian source was recorded.

Results: The prevalence of obesity according to the BMI, 2, 13, 31& 54 % women and 3, 23, 48, & 26% men were lean, normal, overweight and obese respectively i.e. 85% of women are overweight and 74% of men are overweight. Abdominal obesity prevalence is 65% in men and 89% in women according to the waist circumference. We found that the total calorie requirements were derived mainly from white rice by 68% of the study group. Less than 76% of the study group was getting 10% of the protein requirement from their diet. More than79% of them derived their energy from fat. Clearly there is gross protein malnourishment with high carbohydrate derived rice diet that has contributed to the high prevalence of obesity among diabetics and also high incidence of diabetes in this population.

Beverage consumption and long-term association with body weight status in German adolescents - Results from the DONALD Study

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Aim: This study investigates the relationship between the consumption of beverage groups and body weight status in 5 years of study participation in German adolescents.

Methods: Anthropometric and dietary data from 3-day weighed records of 244 subjects between 9 and 18 years of age participating in the Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) Study between 1985 and 2005 were examined. Only subjects with at least 4 out of 6 possible weighed dietary records were considered. A repeated-measures regression model (PROC MIXED) was used to analyse the effect of beverage consumption on body weight status with BMI standard deviation scores (BMI-SDS) and body fat percentage (%BF) as the dependent variables.

Results: In boys, energetic beverage consumption was not associated with BMI-SDS or %BF, neither cross-sectionally nor prospectively. In girls, baseline consumption of energetic beverages did not predict baseline BMI-SDS, baseline %BF, or change in either variable over the study period. However, an increase in energetic beverage consumption over the study period was associated with an increase in BMI-SDS (+0.070 SDS / MJ increase in energetic beverages consumption, p=0.01). Separate consideration of regular soft drinks and fruit juices revealed that, in girls, BMI-SDS increased with increased fruit juice consumption (+0.096 SDS / MJ increase in fruit juice consumption, p=0.01), and to a lesser extent with regular soft drink consumption (+0.055 SDS / MJ increase in regular soft drink consumption, p=0.08).

Conclusion: These results suggest that increasing consumption of energetic beverage may result in weight gain, at least in adolescent girls.

Fructose, glucose and sucrose profiles in sweetened beverages – focus on added sugars

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Human body response is identical, independently of sugars origin, i.e. natural or added, but natural ones are usually accompanied by essential nutrients and minerals, whereas those added are only supplying calories without extra nutritional value. Over-consumption of both, naturally occurring sugars and added sugars, especially in the form of sweetened beverages, increase the risk of development of dental caries as well as of becoming overweight. The aim was to estimate sweetened beverages composition in view of fructose, glucose and sucrose levels and with an emphasis on added sugars content.

Analysis was performed on 85 commercially available non-alcoholic sweetened beverages by HPLC coupled to corona aerosol discharge (CAD). The newly developed method was validated and characterized by wide concentration range (1 – 150 µg/mL), sensitivity and good accuracy (94.9–103%). The method offers excellent linearity with regression coefficient $R^2 > 0.999$ and good repeatability (RSD <5%).

The results showed great variation in sugars concentration in energy, sports, soft and fruit drinks, fruit nectars and juices. The highest levels of sugars were found in energy drinks (14.2 g/100 mL), followed by fruit nectars (13.7 g/100 mL) and soft drinks (12.7 g/100 mL). Soft drinks characterized with the highest average fructose content, i.e. 5.77 g/100 mL, whereas sports drinks contained the lowest amounts of this monosaccharide. The total sugar content within each group varied from information provided by manufacturer. High and regular intake of energy drinks as well as soft drinks might lead to over-consumption of added sugars in view of recommended standards.

Consumption of sugar-containing soft drinks and change in anthropometric measures among adults in Germany

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Objectives: The association between the intake of sugar-containing soft drinks (SCS) and change in body weight (BW) and waist circumference (WC) between 1998 and 2008-2011 was analyzed among adults in Germany.

Methods: In the longitudinal part of the German Health Interview and Examination Survey for Adults (DEGS) participants aged 18 to 79 years in 1998 were reexamined in 2008-2011. Anthropometric measurements and food frequency information were available for 1395 men and 1502 women at both examinations. Frequency of SCS-intake was classified as: low (less than once a week), intermediate (once or several times a week) or high (almost daily and more). The association of baseline and change in SCS-intake with change in anthropometric measures was studied by analysis of variance, considering relevant confounders.

Results: During the observation period, men with high baseline SCS-intake gained on average +1.8 kg more BW as well as +1.7 cm more WC than men with a low SCS-intake (p= 0.01), while differences were not significant among women (BW: +1.2 kg, WC: +1.9 cm). Participants with a high consumption of SCS at both examinations had the highest weight gain (men: +6.1 kg; women +5.1 kg). Mean BW-gain of women, who increased their SCS-intake over time, was twice as much BW compared to women with a consistently low SCS-intake (+4.1 kg versus +2.0 kg, p< 0.05). The association between change in SCS-intake and change in WC was similar but, however, not significant.

Conclusions: Intake of sugar-containing soft drinks seems to affect body weight gain among German adults.

Consumption of juice and carbonated drinks in relation to Body Mass Index among Kuwaiti children aged 2-5 years

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Background: Consumption of sugar-sweetened beverages is a key contributor to the childhood obesity. We investigated the association between the consumption of juice or carbonated drinks and current Body Mass Index (BMI) among 2-5 years old children who participated in the Kuwait National Surveillance System 2013 (KNSS 2013).

Method: Data were collected through KNSS 2013, which selected children (n= 1074) from nurseries and vaccination clinics in health centres in all governorates of Kuwait. Data on consumption of sugar-sweetened beverages were collected through personal interview with mothers as a proxy source of information. Anthropometric measurements were made using standardized protocol.

Results: Approximately 42% consumed 100% fruit juice daily while 62% and 37% consumed juice as a nectar (20-30% fruit juice) or consumed drinks (without fruit juice, e.g. Sunkist and Vimto), respectively, while 21% consumed carbonated drinks on daily basis. There was no significant difference in BMI between those who consumed 100% fruit juice and those who did not, mean (SD) was 16.11(2.04) and 16.03 (2.13), p=0.593. Similarly, no difference in BMI was found between those who consumed carbonated drinks and those who did not, 16.14 (2.35) and 16.09 (2.02), p=0.797. The findings remained unchanged even after stratification by age.

Conclusion: Consumption of carbonated drinks, juice and drinks among 2-5 years old was not associated with increase in BMI, which may be due to inaccurate data collection tools. Efforts should be made to develop and test locally better dietary assessment technique for children in KNSS.

Fructose, but not glucose, impairs insulin signaling in the three major insulin-sensitive tissues

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Objective: Studies in humans relate high consumption of fructose, especially as sugar-sweetened beverages, and fatty liver, hypercholesterolemia, and diabetes. We aimed to

determine in a rodent model whether supplementation of liquid fructose (F) or glucose (G) is able to promote molecular changes resulting in insulin resistance in liver, muscle and adipose tissue.

Methods: Female Sprague-Dawley rats were fed during 56 days with normal chow (control) or normal chow plus *ad libitum* 10% F or G in equicaloric conditions. At the end of the study, a glucose tolerance test (GTT) was performed. Then, 12-h fasted animals were anesthetized and administered with intraperitoneal insulin (0.15 units/g). 15 minutes later, blood was obtained from the hepatic portal vein and liver, adipose tissue and quadriceps were excised and frozen.

Results: Only F rats displayed increased plasma glucose excursions at all time-points in the GTT, whereas insulin levels were significantly increased in both carbohydrate-supplemented rats. In F animals, liver IRS-2 levels were reduced. Further, insulin failed to increase p-Akt in the liver and adipose tissue, and to reduce lipolysis only in F rats. In skeletal muscle, despite insulin increased p-Akt, levels of p-AS160 were unchanged only in F animals.

Conclusions: Under equicaloric conditions F but not G impairs insulin signal transduction in peripheral insulin-sensitive tissues, resulting in an altered GTT.

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High dietary fructose load affects fatty acid oxidation and lipogenesis without increasing lipid deposition in the liver of Wistar rat

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Fructose overconsumption is commonly linked to alterations of lipid metabolism in the liver, however the real lipogenic nature of fructose has recently been questioned. The homeostasis of hepatic lipids is mainly determined by the interplay of lipogenesis and fatty acid βoxidation. In this study we hypothesized that high-fructose feeding affects hepatic lipid metabolism by disturbing the balance between these processes. To test this hypothesis, we analyzed the effects of 9-week consumption of 10% fructose solution on glycemia, lipid status, liver histology and expression of key proteins of fatty acid oxidation and de novo lipogenesis. The results showed that 10% fructose-fed rats were normoglycemic and hypertriglyceridemic, had increased plasma free fatty acid (FFA) levels, but did not show any signs of visceral adiposity or obesity. Western blot analyses revealed that high-fructose diet led to joined increase in the level of nuclear peroxisome proliferatoractivated receptor γ coactivator 1α (PGC 1α) and lipin1, as well as of carnitine palmitoyltransferase 1 (CPT1), implying stimulation of fatty acid oxidation in response to increased FFA influx. On the other hand, the gene expression of hepatic lipogenic enzyme fatty acid synthase (FAS) was increased, which was not accompanied with a comparable change in the sterol regulatory element binding protein 1c (SREBP 1c), nor with accumulation of lipids in the liver.

Our results suggest that 10% fructose diet stimulates hepatic fatty acid oxidation, but simultaneously leads to increased lipogenesis without concomitant lipid deposition. These metabolic alterations may represent a mechanism of adaptation to high-fructose diet aimed at postponing hepatosteatosis.

Prospective Association between Beverage Consumption and Body Weight Development among School Children: A Secondary Analysis of a Water Intervention Study

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Aim: The replacement of sugar-containing beverages by water is a suggested dietary behavioral change to prevent overweight and obesity in children. We investigated if increased water consumption changed the consumption of sugar-containing beverages and if change in water and sugar-containing beverage consumption was associated with changes in body mass index.

Methods: We performed a secondary analysis of a controlled intervention study aiming on increased water consumption. In total, 3220 school children of 32 elementary schools in Germany were enrolled. Body weight and height were measured and beverage consumption was self-reported by a 24-recall questionnaire at the beginning and end of the school year 2006/2007. The effect of a change in water consumption on sugar-containing beverages (soft drinks and juices) consumption and on change in BMI (kg/m^2) was analyzed using linear regressions adjusting for baseline BMI, consumption of other beverages, age, sex, migrational background, study arm, and for clustering by school.

Results: Children (n=1987, mean age=8.3 years) with complete data were analyzed. Mean follow-up was 250 days. After follow-up, increased water consumption by 1 glass/day was

independently associated with a reduced consumption of sugar-containing beverages by 0.12 glasses/day ($p<0.001$). Increased consumption of sugar-containing beverage consumption by 1 glass/day was associated with an increased BMI by 0.02 kg/m^2 ($p=0.011$). In contrast, increased water consumption was not associated with a change in BMI ($ß=0.00$, $p=0.63$).

Conclusion: Water could replace the consumption of sugar-containing beverages which are associated with weight gain. This replacement might be one pathway how water consumption could prevent overweight.

Lifestyle correlates of preference for sweet food in Hong Kong children

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Aim: Sweetness preference is an important predictor of dietary practices and has been linked to unhealthy eating habits in adults. To identify lifestyle factors associated with sweetness preference in Hong Kong children.

Methods: A lifestyle questionnaire was completed by 105,910 Grade 4 students (mean age 10.0±0.7; 50.7% boys)at Student Health Service centres in 1998-2000.Sweetness preference was assessed using the item" My attitude towards sweet food is:" with 4 response options. Students who chose "I like them very much" rather than "they are acceptable", "I'll try a little" or "I dislike them" were categorised as having a sweetness preference. Logistic regression was used to calculate adjusted odds ratios (AORs) for the association of sweetness preference with 9 lifestyle factors, adjusting for sex, age, body mass index and socioeconomic indicators.

Results: Sweetness preference, reported by 37.6% of students, was significantly associated with not having breakfast or lunch at home, and less frequently having dinner at home. More junk food intake(everyday vs never; AOR 1.22,95% CI 1.09-1.38), television watching (≥4h/day vs <1h/day; 1.60,1.52-1.68) and video/computer game playing(sometimes vs never; 1.38, 1.31-1.45) were related to sweetness preference. Less frequent (i) vegetable/fruit intake (<once/week vs ≥3 times/day; 1.38, 1.30-1.47), (ii) physical activities (never vs ≥ 3 times/week; 1.72,1.63-1.81) and (iii) teeth brushing(never vs ≥2/day; 1.47, 1.37-1.58) were also associated with sweetness preference.

Conclusions: Sweetness preference was associated with unhealthy diet, physical inactivity and poor oral hygiene behaviours in Hong Kong children.

Consumption of commercial energy drinks by children and adolescents: a systematic review of consumer attitudes and associations with health, behavioural and social outcomes

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Purpose: To examine patterns of energy drink consumption by children and adolescents and any evidence of adverse effects. Growing numbers of young people report regular consumption of energy drinks, which typically contain high levels of sugar and caffeine in combination with other stimulants.

Methods: A systematic review was conducted. Data sources included nine bibliographic databases, reference lists of relevant studies, and searches of the internet. Two independent reviewers assessed the methodological quality of the studies and abstracted data, which were then descriptively summarized in a narrative synthesis.

Results: A total of 262 studies were located, with 37 meeting the inclusion criteria – 31 quantitative, four qualitative, and two literature reviews. The majority involved subjects aged 11-18 years. Studies were largely conducted in North America or Europe, yet all of the qualitative studies were from Australia or New Zealand. None were from the UK. Two-thirds were cross-sectional surveys exploring i) consumption patterns, attitudes and reasons for energy drink use, or ii) associations with health-related outcomes, including susceptibility to smoking, sleep problems, and diminished executive functions. A number of key themes emerged from the qualitative studies: the role of branding and advertising; taste as a motivating factor; peer influence; and perceived (negative and positive) physiological effects.

Conclusions: Energy drinks have no known therapeutic benefit and evidence suggests that they may put some children at risk of adverse health effects. However, taste and youth-aimed marketing combine to ensure their popularity with young consumers. More research is needed to explore the short- and long-term impacts.

Energy drinks: hype or hyper? A qualitative exploratory study involving children, parents and teachers from schools in North East England

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Purpose: This study aims to investigate what motivates children and young people to consume commercial energy drinks and what effects they are perceived to produce, in order to inform the development of tailored resources and interventions.

Methods: Focus groups are underway with pupils (10-11 and 13-14 years) from schools in North East England. Semi-structured interviews are being undertaken with staff and parents. All data are being transcribed verbatim and analysed using the constant comparative approach. Preliminary analyses and intervention options will be discussed with stakeholders, including children and families, at one or more participatory workshops.

Findings: Six focus groups with pupils (n= 27) and eight interviews with staff have been conducted. Emerging themes include the role of branding and marketing on young people's choices, in addition to the influence of parents, siblings and peers. The data highlight similarities and differences between the young people's views and those of the adult participants, as well as gender and age differences amongst the young people. There is heterogeneity in the motivations, perceived benefits and risks, and the effects associated with energy drink consumption. Suggestions have been put forward by participants to address these issues, and subsequent discussions will consider how these might work, and for whom.

Conclusions: Although data collection is ongoing, a number of important issues have already begun to emerge. Given that this is the first in-depth UK-based study on this topic, we are confident that it will continue to generate findings of interest to diverse academic, practitioner and lay audiences.

Effects of soft drinks and artificially sweetened drinks on hepatic fat content in overweight subjects

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Overweight/obese subjects with a high usual consumption of sugar-sweetened beverages (SSB, ≥2 cans/day) were enrolled and randomized to consume either SSB or artificially sweetened beverages (ASB), while the diet was otherwise left unchanged (spontaneous food intake). Fasting metabolic parameters, visceral adipose tissue volume (VAT) and intrahepatocellular lipids (IHCL) were monitored with magnetic resonance after 4-week run-in and after 12-week intervention.

Consumption of SSB was similar in both groups at inclusion. IHCL were positively correlated with VAT and BMI, but not with sugar consumption. IHCL were reduced by 25.9 ±10.7 % of initial values with ASB ($p<0.01$) but did not change in ASB. ASB, however, did not significantly change body weight (-1.4±1.6 vs +0.8±0.6 kg in C), VAT (-98.4 ±78.4 vs -11.8 ±73.4 cc in C) or fasting glucose, insulin, uric acid, ASAT, ALAT, TG, cholesterol or HDL-cholesterol concentrations.

15 participants (8 in the ASB group, 7 in the C group) had hepatic steatosis as defined by IHCL > 60 mmol/L. They had significantly higher body weight, VAT, TG, uric acid, ASAT, ALAT, and lower HDL-cholesterol than 12 participants without hepatic steatosis. In this subgroup, IHCL was reduced to 56.9 ±12.6%, ALAT to 80.3 ±7.8% and ASAT to 90 ±4.0% with ASB ($p<0.05$).

Our results indicate that, in high SSB consumers 1) body weight and visceral fat volume are major determinants of hepatic steatosis, 2) replacing SSB with ASB reduces intrahepatic fat, most likely due to decreased total energy intake.

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