Obesity and Environmental Contaminants

- 2nd International Workshop
Uppsala, 8-9 October 2015
Dear Colleagues, welcome to Uppsala!

It is a great pleasure to welcome you to the “2nd Obesity and Environmental Contaminants Workshop” in Uppsala.

In 2010 we arranged "The first Obesity and Environmental Contaminants Workshop" in Uppsala. At that time, the idea that environmental contaminants could induce obesity and diabetes had just began to spread to the scientific world due to the pioneering studies by Duk-Hee Lee and Bruce Blumberg, among others, in 2006.

During the 5 years that has passed since the first workshop, these ideas have continued to spread to fields of science other than those dealing specifically with environmental contaminants. Nowadays, for example, there are sessions on environmental contaminants and diabetes on most major international diabetes meetings. Data on environmental contaminants and atherosclerosis and heart disease have been published in leading cardiovascular journals. Projects on environmental contaminants and obesity have been completed within the EU framework OBELIX.

Furthermore, analytical chemists have been able to reduce the amount of blood needed for chemical analysis of environmental contaminants, facilitating the measurement of environmental contaminants in large-scale epidemiological studies. The development in a variety of -omics technologies during the very last years has also improved the ability to further understand the mechanisms whereby environmental contaminants affect health.

In addition, through results from new experimental studies, it is becoming clear that exposure to low, environmentally relevant, doses of environmental contaminants during development can lead to disease later in life. To some extent the authorities has adjusted the threshold values, but research has shown biological effects at levels way below these thresholds.

Thus, taken together, a big progress in the field of research regarding environmental contaminants, obesity and related disorders has taken place during this 5 years period, a fact that will make this meeting an interesting meeting point for an update on the state of the art and a meeting point for discussions amongst the many disciplines involved in this research area!

We hope that you will enjoy the workshop and have a pleasant stay here in Uppsala.

Margareta Halin Lejonklou & Monica Lind
SCIENTIFIC PROGRAMME

THURSDAY 8 OCTOBER

8:00 – 8:40 REGISTRATION AND COFFEE (Registration open until 10:45).

8:40 – 8:50 WELCOME ADDRESS: Dr. Eva Tiensuu Janson, Dean of the Medical Faculty, Uppsala University, Sweden

8:50 – 9:00 WELCOME ADDRESS: Dr. Monica Lind, Occupational and Environmental Medicine, Uppsala University

9:00 – 9:45 OPENING SESSION: Dr. Linda Birnbaum, Director, National Institute of Environmental Health Sciences (NIEHS), U.S.A.: Developmental Origins of Health and Disease (DOHaD): A good start lasts a lifetime

9:45 – 10:15 Dr. Laura Vandenberg, University of Amherst, Massachusetts, U.S.A.: Low doses of environmental contaminants and non-monotonic dose response curves, with special reference to current risk assessment practices

10:15 – 10:45 BREAK: Organic fruit and coffee

10:45 – 11:30 Dr. Jerry Heindel, Scientific program administrator, NIEHS, U.S.A.: Metabolic Disruptors and Metabolic Disruption

11:30 – 12:00 Dr. Åke Bergman, Director SWETOX, and Stockholm University, Sweden: The significance of EDC exposure via dust intake/inhalation

12:00 – 13:00 ORGANIC LUNCH

13:00 – 13:30 Dr. Angel Nadal, Miguel Hernandez University, Elche, Alicante, Spain: Is Bisphenol A a Diabesogen? Timing of Exposure as a Predictor of Diabesity

13:30 – 14:00 Dr. Richard P. Phipps, University of Rochester School of Medicine and Dentistry, Rochester, NY, U.S.A.: Environmental Obesogens: Mechanism of action through Thy1 (CD90)

14:00 – 14:40 Hong Kyu Lee, Eulji University Hospital, Daejeon, South Korea; and Yongmi Kim Pak, Kyung Hee University, Seoul, South Korea: Endocrine disrupting chemicals associated with serum AhR binding and mitochondrial activity inhibition in diabetic subjects

14:40 – 15:15 BREAK: Organic fruit and coffee
15:15 – 15:45 Dr. Erik Lampa, Uppsala University, Sweden: Mixture effects of multiple environmental contaminants on the metabolic syndrome in a human population-based sample

15:45 – 16:15 Dr. Carlos Guerrero-Bosagna, Linköping University, Sweden: Epigenetics and disease etiology: linking past environmental exposures to current disease trends

16:15 – 16:45 Dr. Daniel Zalko, French National Institute for Agricultural Research (INRA), Toxalim research center in food toxicology, Toulouse, France: Exposure routes, metabolism and targets of bisphenol A and its analogues

16:45 – 17:15 Dr. Margareta Halin Lejonklou, Uppsala University, Sweden: Effects of developmental low dose exposure to Bisphenol A with special reference to metabolic and bone disturbances

17:15 – 17:30 CONCLUSIONS OF THE DAY. Dr. Mattias Öberg, SWETOX and Institute of Environmental Medicine, Karolinska Institutet

19:00 DINNER FOR ALL PARTICIPANTS AT NORRLANDS NATION, Västra Ågatan 14, Uppsala

FRIDAY 9 OCTOBER

8:30 – 9:00 Dr. Juliette Legler, VU University Amsterdam, The Netherlands: Integration of experimental and epidemiological approaches to developmental obesogenicity – Results from the EU-project OBELIX

9:00 – 9:30 Dr. Marlene Ågerstrand, Stockholm University, Sweden: Risk assessment and regulation of endocrine disruptors from a global perspective

9:30 – 10:15 Dr. Bruce Blumberg, University of California, Irvine, U.S.A.: Transgenerational effects of endocrine disrupting compounds with focus on adipose tissue and bone disturbances

10:15 – 10:45 BREAK: Organic fruit and coffee

10:45 – 11:15 Dr. Lars Lind, Uppsala University, Sweden: Using "omics"-technologies to discover links between environmental pollutants and human disease

11:15 – 12:00 MODERATED DISCUSSION: HOW CAN WE MOVE THE FIELD FORWARD?

- Why is the importance of EDCs in obesity, diabetes and lipid disorders not accepted by the general public, clinicians and basic scientist? What data are needed to have our field moved into more mainstream thinking on obesity and how to cure and prevent it?
- How can we better integrate epidemiology studies and experimental studies to get the greatest impact?
- How can we move into mixture studies?

12:00 – 12:30 HIGHLIGHTS OF THE MEETING, Dr. Linda Birnbaum, NIEHS
Metabolic Disruptors and Metabolic Disruption: Moving the Field Forward

Jerrold J. Heindel, Ph.D. Division of Extramural Research and Training, National Institute of Environmental Health Sciences, RTP, North Carolina, USA, heindelj@niehs.nih.gov

The obesogen hypothesis that posits that there are specific environmental chemicals that can increase the susceptibility to obesity across the lifespan, especially when exposure occurs during development is now about 10 years old. During this time, which could be called phase I, numerous “Obesogens” have been identified and their effects on weight gain and adipose tissue have been characterized.

There is still much work to be done to identify and characterize the effects of new obesogens, as well as to define dose responses and tissue targets of known obesogens. Nonetheless, the field is moving from the descriptive phase I to a more mechanistic phase II. The first major shift is that the field is moving from a focus on obesogens to a more general characterization of metabolic disruptors due to the data indicating that many obesogens also affect pancreas and liver function. The field is also moving to better define critical windows of sensitivity to metabolic disruption including preconception and transgenerational windows. It is also now apparent that metabolic disruptors do not cause metabolic disruption per se but only increase the sensitivity or set-point for metabolic disruption and that many of the effects are sexually dimorphic. These are important areas where more data are needed.

While there has been an increased focus on mechanism, much more needs to be done including focus on brain pathways, importance of stem cells as sites of action, epigenetics and other pathways as mechanisms of persistent effects as well as a focus on the importance of basic pathways such as sirtuins, MTor, oxidative stress and mitochondrial dysfunction and inflammation. Finally there is improved integration of animal and human data and endpoints. The move to phase II will hopefully have an impact on the acceptance of the metabolic disruptor field by the basic science, clinical and general communities which could lead to important advances in prevention of metabolic disruption.
Are Endocrine Disruptors “Diabetogens”? Timing of Exposure as a Predictor of Diabetes Mellitus

Paloma Alonso-Magdalena, Ph.D. Esther Fuentes, Ph.D. Cristina Ripoll, Ph.D. Ivan Quesada, Ph.D. and Angel Nadal, Ph.D. Institute of Bioengineering and CIBERDEM, Universidad Miguel Hernández de Elche, Alicante, Spain, nadal@umh.es

Type 2 diabetes (T2D) and obesity are two of the world's greatest health care issues. The etiology of T2D is multifactorial and not completely understood. T2D development is normally associated with genetic predisposition as well as with environmental factors such as obesity, aging, and lack of exercise.

Endocrine disrupting chemicals (EDCs) exposure is also a recognized risk factor for T2D. It has been proven to induce insulin resistance in cellular and animal models, leading to the “diabetogen hypothesis”. This hypothesis proposes that: “every EDC circulating in plasma able to produce insulin resistance, independently of its obesogenic potential and its accumulation in adipocytes, may be considered a risk factor for metabolic syndrome and type-2 diabetes”. Epidemiological studies associate exposure to EDCs with T2D in humans, particularly POPs such as DDT and its metabolites, PCBs and dioxins as well as other EDCs including Biphenol-A, phthalates and arsenic. Causality has been demonstrated in animal and cellular models.

White adipose tissue and pancreatic beta cells are disrupted by EDCs, altering insulin secretion and insulin signaling and causing inflammation. In adult rodents, some EDCs cause insulin resistance and dysfunction of pancreatic beta cells inducing a pre-diabetic like state. Perinatal exposure represents a risk factor for offspring later in life. Notably, offspring final phenotype is dose, gender and age dependent. Recently, published data demonstrate that pregnancy is also a sensitive period for the mother.
Environmental obesogens: Mechanism of action through Thy1 (CD90)

Richard P. Phipps, Ph.D. and Collynn Woeller, Ph.D. University of Rochester School of Medicine and Dentistry Rochester, NY USA, Richard_Phipps@urmc.rochester.edu

Obesity has risen dramatically over the last 30 years. In the U.S. alone, 60 million people are defined as clinically obese. Especially concerning is the nearly epidemic rate of childhood obesity. Obesity occurs through excessive adipogenesis (formation of adipocytes) or through increases in established adipocyte size. Environmental toxicants termed “obesogens” disrupt the endocrine system and can increase adipogenesis. Tributyltin (TBT), dichlorodiphenyl-dichloroethylene (DDE), bisphenol-A diglycidyl ether (BADGE), and tetrabromobisphenol-A (TBBPA), have all been reported to increase obesity.

The mechanism(s) by which xenobiotic obesogens function to disrupt the endocrine system and increase rates of obesity requires active investigation. Our studies focus on the surface glycoprotein, Thy1 (CD90). We discovered that Thy1 has a direct role in inhibiting adipogenesis and that cells with no/low Thy1 are more prone to become fat cells. Furthermore, Thy1 knockout mice gain more weight when put on a high fat diet than control mice. We also discovered that Thy1 expression is reduced in both human and mouse multipotent stromal cells (MSCs) after exposure to the obesogens TBBPA, BADGE and TBT. Importantly, one mechanism of action of these obesogens is to increase expression of microRNA 103, a miRNA that is upregulated in obesity and type 2 Diabetes. We now show that Thy1 is indeed a target of miR103, and miR103 decreases Thy1 levels. These findings provide a molecular link between environmental endocrine disruptors, Thy1 and obesity.
Endocrine disrupting chemicals associated with serum AhR binding and mitochondria inhibiting activities in diabetic subjects

Hong-Kyu Lee, MD and Youngmi Kim Pak, PhD. Department of Internal Medicine, College of Medicine, Eulji-University and Department of Physiology, College of Medicine, Kyung Hee University, Seoul, Korea, hkleemd@eulji.ac.kr

Some Persistent organic pollutants (POPs) and endocrine disrupting chemicals (EDCs) are ligands of aryl hydrocarbon receptor (AhR), which is a ligand-activated nuclear receptor. POPs have emerged as the important causal factor of insulin resistance and metabolic syndrome. Based on the previously recognized role of abnormal mitochondrial function in diabetic pathogenesis, POPs were suspected to disrupt mitochondrial activities. We have developed a highly sensitive cell-based AhR-ligand binding assay, and found serum level of AhR ligands is elevated in subjects with diabetes and metabolic syndrome. Furthermore we found serum AhR binding activity predicts future development of diabetes. Mitochondrial dysfunction is closely associated with insulin resistance and biochemical abnormalities underlying metabolic syndrome. As intracellular ATP contents of cultured cells treated with human serum were measured, diabetic serum decreased ATP contents more than normal serum, indicating higher mitochondria inhibiting activity. Surprisingly ATP content correlated with AhR binding.

Recently, we compared the AhR binding activity and ATP contents with the levels of chemically measured plasma total toxic equivalence (TEQ) of POPs mixture and individual POPs using 1,016 sera from the Prospective Investigation on the Vasculature in Uppsala Seniors (PIVUS) study. AhR binding activity was linearly related to TEQ to 12 of 16 measured PCBs, independently of dioxin-like or non-dioxin-like properties. OCDD was not related to AhR binding, but BDE47 was. ATP content was negatively related to TEQ and TEQplanar, but not to TEQortho. Paradoxically, only 4 PCBs were related to ATP. Significance of these findings will be discussed.
Mixture effects of multiple environmental contaminants on the metabolic syndrome in a human population-based sample

Erik Lampa, Ph.D. Uppsala University, Uppsala, Sweden, erik.lampa@ucr.uu.se

The metabolic syndrome is a collection of five risk factors for cardiovascular disease that usually run together. Several environmental contaminants have been associated to the prevalence of the metabolic syndrome but prospective studies and studies assessing mixture effects are lacking. In this study, we investigated the associations between a large number of environmental contaminants measured in blood in 452 individuals, free of the metabolic syndrome, aged 70 years and 10-year incidence of the metabolic syndrome. Associations were investigated using boosted Classification and Regression Trees (CARTs). A single CART is easily interpreted but lacks predictive power. Combining, boosting, several CARTs leads to superior predictive performance and automatically handles non-linear and non-additive effects. The bootstrap was used to quantify uncertainties in predicted probabilities.

92 individuals (20%) developed the metabolic syndrome during follow-up. The strongest predictors for incident metabolic syndrome were in order; baseline HDL cholesterol, baseline triglyceride levels, baseline waist circumference, PCB 126, energy intake, PCB 118 baseline fasting blood glucose, Cobalt, Hexachlorobenzene (HCB) and baseline systolic blood pressure. No interactions between any contaminants could be seen.

PCB 126, PCB 118, HCB and to a lesser extent Cobalt predicted incident metabolic syndrome independent of baseline risk factors. The strengths of association were on par with baseline fasting blood glucose and baseline systolic blood pressure.
Epigenetics and disease etiology: Linking past environmental exposures to current disease trends

Carlos Guerrero-Bosagna, Ph.D. Avian Group, IFM Biology, Linköping University, Linköping, Sweden, carbo@ifm.liu.se

Early exposures to environmental toxicants during fetal development are fundamental to explain reproductive impairments and metabolic diseases recently observed in human populations. Some environmental exposures, which include daily practices, occupational exposures and contact with contaminants, are currently known to produce epigenetic changes related to conditions in humans. Interestingly, experiments in animal models have shown that exposure to environmental toxicants can, in addition, induce transgenerational inheritance of some disease phenotypes.

The mechanism of transgenerational epigenetic inheritance involves exposure of the germ line to drastic environmental conditions during critical developmental periods. Such exposure generates germ line epigenetic alterations that can be transmitted to future generations and associate with altered phenotypes in the unexposed individuals of subsequent generations. Exposures to environmental toxicants such as fungicides, pesticides or plastic compounds have been shown in rodents to produce abnormal reproductive or metabolic phenotypes that are transgenerationally transmitted. Environmentally-induced and transgenerationally transmitted phenotypes observed in animal models include non-communicable diseases of increasing incidence in human populations such as of obesity, polycystic ovary syndrome (PCOS), pregnancy defects or fertility impairments.

This presentation summarizes recent findings showing that early developmental exposures to a variety of environmental toxicants can induce epigenetic transgenerational inheritance of phenotypes associated with non-communicable diseases of common incidence.
Exposure routes, metabolism and targets of bisphenol A and its analogues

Daniel Zalko, Ph.D. French National Institute for Agricultural Research (INRA), Toxalim, Toulouse, daniel.zalko@toulouse.inra.fr

Bisphenol A (BPA) is a model xeno-estrogen produced in large quantities, with broad and acknowledged effects on the reproductive system as well as general homeostasis. BPA’s use is already regulated in several countries, and emerging candidate “replacement” bisphenols (BPS, BPF…) are of growing concern for toxicologists.

Other analogues, namely halogenated bisphenols, are used to manufacture flame-proofed epoxy resins, with recent results strongly hinting for previously unexpected toxicological effects. Bisphenols are readily conjugated into the corresponding glucuronidated and sulfated metabolites, which are their major metabolites. Since BPA-glucuronide was 15 years ago demonstrated not to be estrogenic, it is classically considered that bisphenols biotransformation results in efficient and almost complete detoxification. However, in vivo and in vitro studies of bisphenols fate have demonstrated that this assumption is incorrect. Among others, was demonstrated the ability of BPA-glucuronide to deconjugate back into the parent compound at the level of target tissues, and BPA itself reaches the developing fetus even in primates.

Moreover, although phase II (conjugative) metabolic pathways predominate for all bisphenols, phase I (oxidative) metabolic routes exist, and qualitatively depend on the presence of a central asymmetric carbon and on its degree of substitution. Phase I reactions may play a major role in the toxicity of halogenated bisphenols. The latter bisphenols (but also some of their metabolites) target distinct nuclear receptors, further suggesting a direct impact on energy metabolism. This lecture will give an overview of the current knowledge about bisphenols routes of exposure, about their fate and its links with toxicological issues.
Effects of developmental low dose exposure to Bisphenol A with special reference to metabolic and bone disturbances

Margareta H Lejonklou, Ph.D. Linda Dunder, MSc. Emelie Bladin, MSc. Monica Rönn, Ph.D. Håkan Melhus, Ph.D. Annica Rasmusson, Ph.D. Thomas Lind, Ph.D. Sune Larsson, Ph.D. Lars Lind, Ph.D. Tomas B. Waldén, Ph.D. P. Monica Lind, Ph.D.

Effects of Bisphenol A (BPA), a monomer used in for example polycarbonate plastic, have been extensively studied. Controversies remain regarding whether BPA exerts effects at low doses (corresponding to those of human exposure, 0.1 – 1.5 µg BPA/kg bw/day, or in traditional toxicology studies defined as below a LOAEL of 50 mg BPA/kg bw/day), despite the fact that endogenous hormones act at minute concentrations.

EFSA has lowered their TDI in two steps: From 50 (the current FDA TDI) to 5 µg BPA/kg bw/day in January 2014, and in January 2015 to a new preliminary TDI of 4 µg BPA/kg bw/day. EFSA expresses concern regarding effects on sexual differentiation, behavior, and the prostate.

Several studies have pointed to BPA as a potential metabolic disruptor. Low dose exposure to BPA during development has shown somewhat contradictory results regarding metabolic effects, possibly due to different experimental setups (such as species and strain, exposure route, exposure window, diet). Many studies have shown weight gain and blood lipid imbalances, while in other studies developmental BPA exposure led to weight loss. Osteoporosis related bone fractures as well as metabolic disturbances have reached epidemic proportions during the last decades, and bone quality has been shown to be affected by several endocrine disruptors.

We exposed pregnant and lactating Fischer F344 rats, an estrogen sensitive strain, to low doses of BPA (0.5 and 50 µg/kg bw/day) from GD 3.5 to PND22 via their drinking water, without additional stimuli. The offspring was sacrificed at five and 52 weeks of age. We found gender specific blood lipid and fatty acid imbalances, increased cell density in the inguinal fat depot, as well as altered adipose tissue gene expression in the five-week-old offspring. Five-week-old males exhibited a weaker bone phenotype, determined using pQCT and biomechanics. Additionally, we observed a significant weight increase in the 52-week-old female offspring developmentally exposed to 50 µg BPA/kg bw/day. Our present study shows that developmental exposure to a BPA-dose 8 times lower than the current EFSA TDI significantly alters several parameters.
Integration of experimental and epidemiological approaches to developmental obesogenicity – results from the EU-project OBELIX – and beyond

Juliette Legler, Ph.D. Institute for Environmental Studies, VU University Amsterdam, The Netherlands, juliette.legler@vu.nl

OBELIX ("OBesogenic Endocrine disrupting chemicals: LInking prenatal eXposure to the development of obesity later in life") was a large pan-European study researching the obesogen hypothesis. This multidisciplinary study project focused on assessing the exposure to and obesity related effects of major classes of EDCs found in food including dioxins and polychlorinated biphenyls, brominated flame retardants, organochlorine pesticides, phthalates and perfluorinated alkyl acids, as well as bisphenol A (BPA).

A series of five long term animal studies were performed, in which mice were exposed to EDCs through the diet during gestation and lactation. Offspring were monitored up to adulthood. Doses in animal experiments reflected human exposure and levels were below low adverse effect levels for developmental toxicity. Epidemiological studies involved mother-child cohorts from 4 European countries in which perinatal EDC exposure was determined in cord blood and milk samples in the same EDCs as in the animal studies, and weight trajectories were followed up to 8 years. Gene expression and DNA methylation analyses were performed to identify mechanisms underlying the potential programming effects of chemical exposure, using human, animal and in vitro adipocyte models.

The results of OBELIX show that early life exposure to EDCs can alter metabolic pathways that play an essential role in energy metabolism and weight regulation, underlining the importance of preventing chemical exposures during sensitive periods of life. Current research in my lab is focused on unravelling mechanisms by which EDCs can perturb metabolic pathways and adipogenesis, including disruption of circadian rhythms and lipid metabolism.
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Painting of Linné: Svenska Linnesällskapet/Gustaf Lundberg