doi: 10.1111/obr.12259

Poster Abstracts

7th Fribourg Obesity Research Conference (FORC-2013)

'Pathways from dieting to weight regain, to obesity and to the metabolic syndrome'

P1

The mouse metabolic evaluation facility (MEF) of UniL/CHUV

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The MEF proposes a vast repertoire of metabolic analyses to assess the phenotype of murine models of obesity & diabetes, & to test the effectiveness of new therapeutic pharmacological agents. For this purpose, MEF

- Provides services to researchers from Academia and Industry,
- Ensures the development of new investigation techniques,
- Teaches some techniques,
- Supports internal metabolic research at the Center for Integrative Genomics, thus ensuring a constant updating of equipment and expertise.

The MEF, first facility of its kind in Switzerland since 2006, offers a comprehensive and constantly evolving panel of analyses in areas of Energy metabolism, Glucose homeostasis, and the relationship between brain and periphery. Excerpts of services provided by the MEF include:

- The non-invasive and accurate analysis of body composition by MRS
- The analysis of metabolism by indirect calorimetry,
- Analysis of metabolism during exercise (treadmill) by indirect calorimetry,
- Measurement of energy content in biological samples by bomb calorimetry,
- Real-time, online measurement of food intake (cumulative & patterns, food preference),
- The real-time, telemetric measurement of body temp. & locomotor activity,
- The assessment of glucose homeostasis by insulinemic (eu-, hyper-, hypoglycemic) clamps
- Intracerebral (ICV/IHP) injections of metabolites/drugs/peptides/viruses.
- Blood chemistry (Hitachi robot) and multiplex assays of cytokines / hormones (Luminex).

The MEF, which has already been very well received by academic researchers and industrial customers alike, strives at provide customized, affordable services to the broader scientific community in a constant search for excellence and standardization.

P2

Implication of ketone bodies in brain control of energy homeostasis and food intake

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Although glucose is the main energy substrate of the brain, alternative substrates can also be used under specific circumstances. Thus, during fasting, ketone bodies (KB) production increases to fulfil energy needs of brain cells. However, their level also increases in type I diabetes or high fat diet-induced obesity. Moreover, monocarboxylate transporters (MCTs) (which are also KB carriers) exhibit increased expression in the brain of obese hyperketonemic mice. A recent study demonstrated in vitro that KB stimulation increases the mRNA expression level of the orexigenic neuropeptide AgRP. In the present study, we aimed to determine in vivo the involvement of KB in the central control of food intake and energy homeostasis. A carotid KB infusion was used to stimulate specifically brain areas. After the infusion, the hypothalamus (involved in food intake regulation) was removed to determine the mRNA levels of the orexigenic and anorexigenic neuropeptides. MCT protein expression was also determined by western blot. Results obtained show that brain stimulation by KB increased food intake of mice within 6 h, which persisted until 24 h. This food intake increase was associated with increased levels of the orexigenic neuropetides NPY and AgRP in the hypothalamus while no effect was detected on the anorexigenic ones (POMC and CART), or on MCTs. These results show that KB represent an orexigenic signal for the hypothalamus by stimulating the orexigenic neurons involved in food intake and energy homeostasis regulation.

P3

Body weight influences the interplay of brain dynamics to food viewing and gastric hormone secretion

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Endocrine factors (i.e. digestive hormones) convey information about energetic needs to brain areas implicated in homeostatic maintenance, but also hedonic valuation processes. So far, it is yet not well determined in how far digestive hormones interact with cognitive and sensory processes during food viewing while motivation towards ingestion varies, and whether these processes are modulated as a function of body weight. Our work investigated the influence of body mass index (BMI) on gut hormone secretion in women with BMIs ranging from 19 to 36 kg/m^2, as well as on the associations of BMI and hormone secretion with spatio-temporal brain dynamics when viewing foods in fasted vs. fed nutrition state.

Results showed that the increased secretion of hormonal satiety signals active on short- vs. long-term time scales (i.e. GIP and leptin) is associated with decreased activity in BMI-modulated "food evaluation" brain regions when fasted, but also decreased activity in BMI-modulated "food intake control" regions when fed. Our study indicates that an elevated BMI likely leads to discrepancies between the evaluation of homeostatic needs, reward attribution and food intake control, in extension promoting increased food intake and weight gain.

P4

The ADIFIT program: an intensive multi-disciplinary, cognitive behavioral treatment based approach for weight management

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Aim: The ADIFIT program is an intensive 12-week multidisciplinary treatment program which promotes weight reduction among obese. During the first six weeks behavior change, physical exercise, and nutritional counseling is offered three times a half a day per week, thereafter once a week half a day. The aim was to evaluate the efficacy of the ADIFIT program in weight reduction, and its effect on eating behavior, and body image.

Methods: Patients were evaluated at baseline (T0) and after 3 month (T1). BMI was assessed. Psychometric instruments such as the German versions of the Eating Behavior Questionnaire (FEV), and of the Body Image Questionnaire (BIQ-20, FKB-20) were used at both time points. Paired-sample *t*-test and Wilcoxon Signed Rank test were used for statistical analyses.

Results: Fifty patients participated, 86% were female. BMI was significantly reduced from T0 to T1 (mean ± SD: 41.1 ± 6.6 vs. 39.8 ± 6.6; $t = 7.68$, $p < 0.000$). Results discerned significant improvements from T0 to T1 in FKB scale 1 (negative attitudes towards one's own body) (34.6 ± 6.0 vs. 32.2 ± 6.1; $t = -3.68$, $p < 0.000$), and scale 2 (restricted body dynamics) (28.2 ± 7.0 vs. 31.2 ± 6.3; $t = -3.86$, $p < 0.000$). On the other hand, FEV scale 1 (cognitive restraint of eating) improved significantly from T0 to T1 (8 vs. 15, $z = -5.72$, $r = -0.57$, $p < 0.000$). FEV scale 2 (disinhibition) was significantly lower at T1 (11 vs. 6, $z = -5.84$, $r = -0.58$, $p < 0.000$) as was FEV scale 3 score (hunger) (8 vs. 4; $z = -4.98$, $r = -0.49$, $p < 0.000$).

Conclusions: After 12 weeks patients showed a significant reduction in weight, as well as improvements in body image and eating behavior.

P5

Effect of neonatal nutritional status on the association between polymorphism rs9939609 of the FTO gene and obesity in Chilean children of Amerindian origin

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Aim: The aim of this study was to investigate the effect of neonatal nutritional status on the association between the polymorphism rs9939609 of FTO gene and obesity in a cohort of Chilean children of Amerindian origin.

Method: In 238 obese and 136 eutrophic children between 6 and 11 years of age, the neonatal ponderal index (NPI) was calculated from their birth records. The children were classified as eutrophic or obese according to the percentiles adjusted for age and gender proposed by the CDC. The FTO polymorphism was determined by real-time high-resolution melting PCR.

Results: Carrying the FTO A-allele was associated with an increased risk of obesity (OR 1.873 CI 95% 1.149–3.062 p value = 0.0076). The risk of obesity from carrying the A-allele considerably increases in subjects with NPI < p10 compared to those with NPI > p10 (OR 5.65 vs. OR 1.69). A multiple logistic regression model demonstrated the risk of obesity from carrying the FTO A-allele; using neonatal nutrition and gender as control variables, the magnitude of the effect increases (OR 2.534 CI 95% 1.23–5.23 p value = 0.012).

Conclusion: Our results confirm the potentiating effect of neonatal malnutrition in the link between the A-allele of FTO SNP rs9939609 and childhood obesity, as has been communicated by other authors in other ethnic groups and with other SNPs.

P6

Cardiovascular responses to ingestion of sugary drinks using a randomised cross-over study design: does glucose attenuate the blood pressure-elevating effect of fructose?

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Overconsumption of sugar-sweetened beverages has been implicated in the pathogenesis of cardiovascular diseases. Our objective was to elucidate acute hemodynamic and microcirculatory responses to sugary drinks made from sucrose, glucose or fructose at concentrations similar to those often found in commercial soft-drinks. In a randomized cross-over study design, 12 young healthy humans (7 males, 5 females) ingested 500 mL tap water in which was dissolved 60 g of either sucrose, glucose or fructose, or an amount of fructose equivalent to that in sucrose (i.e. 30 g fructose). Continuous cardiovascular monitoring was conducted for 30 min before and 60 min after the sugar drinks, and measurements included beat-to-beat blood pressure and impedance cardiography. Additionally, microvascular endothelial function testing was performed after iontophoresis of acetylcholine and sodium nitroprusside using laser Doppler flowmetry. Ingestion of fructose (60 g or 30 g) increased diastolic and mean blood pressure to a greater extent than 60 g of glucose or sucrose ($p < 0.05$). Sucrose and glucose increased cardiac output ($p < 0.05$), index of contractility ($p < 0.05$), and stroke volume ($p < 0.05$), but also reduced total peripheral resistance ($p < 0.05$), which contrasts with the tendency of fructose (60 g and 30 g) to increase resistance. Microvascular endothelial function did not differ in response to the various sugar drinks. In conclusion, sucrose is comprised of glucose and fructose but its hemodynamic actions are more related to glucose than to fructose. Our data suggest that the blood pressure-elevating effects of fructose are dampened in the presence of glucose.

P7

Presence of brown adipocytes in white adipose tissue and its role in obesity resistance and insulin sensitivity

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Background and aims: Brown adipose tissue (BAT), characterized by uncoupling protein 1 (UCP1), has been described as metabolically active in humans. It induces an increase in energy expenditure (EE) by increasing thermogenesis. Lou/C rats which originate from the Wistar strain are resistant to obesity induced by either age or a high fat diet. In inguinal white adipose tissue (WATi) of Lou/C rats, we previously demonstrated the presence of ectopically expressed UCP1, as well as of an overexpression of the β3-adrenoreceptor which controls UCP1 expression. The aim of the present study was to investigate the role of UCP1 in the metabolic regulation of Lou/C rats, including the control of body weight gain and the improvement of insulin sensitivity.
Materials and methods: β3 agonist treatment (CL-316243, s.c, 1 mg/kg/day) was administered in 3 month-old Wistar and Lou/C rats for 2 weeks. Glucose and cold tolerance, fat mass repartition, overall & tissue-specific insulin sensitivity, and EE were measured.
Results: The treatment induced lower food efficiency, a decreased fat volume (Wistar control = 19.08 cm^3, Wistar treated = 7.91 Lou/C control = 4.83, Lou/C treated = 2.27, $p < 0.001$) and enhanced EE in both strains (W = 7.09 kcal/h/$kg^{0.75}$, Wt = 9.04, L = 8.68, Lt = 11.23, $p < 0.001$). It also increased overall insulin sensitivity (GIR: W = 22.3, Wt = 32.6, L = 31.8 Lt = 40.6, $p < 0.001$), as determined using euglycemic hyperinsulinemic clamp technique. However, muscle glucose uptake, measured with labelled 2-deoxy-glucose, was unaltered by the treatment in both groups. On the contrary, insulin-stimulated glucose uptake in different white adipose tissue depots was highly increased by the β3 adrenoceptor treatment in the Lou/C group only (L = 1.6 ng gluc/mg tissues, Lt = 19.7, $p < 0.001$), an increase that correlated with the expression of UCP1 in these tissues. Regarding this gene, the treatment induced its overexpression in the Lou/C group only, and observed in WATi (164-fold increase, $p < 0.01$) and even more marked in epididymal fat (WATe) (28 864-fold increase, $p < 0.01$). These results were confirmed by Western blot analysis. In BAT, UCP1 increased to a similar extent in Wistar and Lou/C rats in response to the treatment (threefold increase in both groups, $p < 0.001$).
Conclusion: A β3 adrenoceptor treatment has similar beneficial effects in Wistar and Lou/C rats on food efficiency through a stimulation of EE and overall insulin sensitivity. However, the treatment-induced increase in white adipose tissue insulin sensitivity is more marked in Lou/C than in Wistar rats, as a likely result of UCP1 overexpression in this tissue. This could be one of the mechanisms responsible for the maintenance of a lean phenotype in Lou/C rats.
Grant acknowledgement: Swiss National Science Foundation (31003A_134919/1), European Union FP7 (DIABAT).

P8

The effect of western-style high fat-high fructose diet on skeletal muscle mitochondrial energetics in sedentary rats

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Introduction: We have recently found that administration of a low fat diet rich in fructose to adult rats, that develop obesity and insulin resistance, is able to increase mitochondrial energetic efficiency, both in liver and skeletal muscle. The purpose of this study was therefore to investigate the effects of isocaloric administration of either a high fat or a high fat-high fructose diet, a typical unhealthy Western diet, on mitochondrial energetics in skeletal muscle of adult rats and the possible links with insulin resistance.
Methods: Adult rats were isocalorically fed with high fat or high fat-high fructose diet for 2 weeks. Body and skeletal muscle composition, energy balance and plasma lipid profile were measured. Mitochondrial mass, respiratory activity and energetic efficiency, together with indexes of oxidative stress and antioxidant defence of these organelles were also assessed. Whole-body insulin sensitivity was determined after glucose load, while skeletal muscle insulin responsiveness was evaluated through the determination of levels of phosphorylated Akt (p-Akt), a distal effector of insulin in this tissue.
Results: Rats fed high fat-high fructose diet exhibited significantly higher plasma triglycerides and non-esterified fatty acids, together with a significantly higher plasma glucose and insulin response to

glucose load. Skeletal muscle triglycerides and ceramide were significantly higher in rats fed high fat or high fat-high fructose diet, while p-Akt normalised to plasma insulin was significantly lower, in rats fed high fat-high fructose diet. Skeletal muscle mitochondrial energetic efficiency and uncoupling protein 3 protein content were significantly higher, while adenine nucleotide translocase content was significantly lower in rats fed high fat or high fat-high fructose diet.
Conclusion: The results suggest that Western diets even without hyperphagia are able to increase lipid flow to skeletal muscle and mitochondrial energetic efficiency, that could have two detrimental effects: (a) energy sparing that contributes to the early onset of obesity, (b) reduced oxidation of fatty acids and lipid accumulation in skeletal muscle, which could generate insulin resistance.

P9
Does dieting affect brain metabolism and cognitive functions?

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Improvement in socioeconomics over the past century has drastically affected our lifestyle. The concomitant increase in food availability and sedentary nature of life has drastically affected health. Brain, although it represents only 2% of the total body weight, consumes about 25% of the total glucose body utilization at rest. Therefore, it is more than likely that nutrition and/or physical activity may affect brain metabolism, and especially neuroenergetics, but also cognitive functions. However, the precise molecular mechanisms induced by food and/or physical activities on neuroenergetics and on cognitive functions are not fully understood. In the present study, food restriction was performed on 12-weeks-old C57Bl/6J males for 5 weeks to assess whether dieting would affect neuroenergetics and subsequently cognitive functions. Two different food restriction diet protocols were tested in parallel: the caloric restriction diet (CR) and the intermittent fasting diet (IF). Mice were then tested in different behavioral paradigms to evaluate cognitive performances and finally the expression levels of genes involved in neuroenergetics and in synaptic plasticity were measured to assess the impact of dieting on brain functions. Our preliminary results suggest that both food restriction diets differently affected mice behavior, and especially spatial memory, but also neuorenergetics and synaptic plasticity. Mice maintained on CR, but not on IF diet, exhibited enhanced cognitive performances. Further, an increase in the expression level of genes involved in neuroenergetics, such as the moncarboxylate transporters and the lactate dehydrogenases, but also in synaptic plasticity, were observed in animals maintained on dieting compared to animals maintained on ad libitum diet. In conclusion, dieting induced early neuroenergetics and synaptic adaptations, resulting in neuronal remodeling and in enhanced brain metabolism in order to better couple neuroenergetics to cognitive processes.

P10
Cortical circuits matching body metabolic signals and behavior

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The insular cortex (IC) monitors the homeostatic state of the body and responds to peripheral metabolic challenges, such as fasting. Studies in humans and animal models have shown that it also plays a key role in complex behaviors, such as decision making and emotions. However, the neuronal circuits involved in these integrative functions of IC are poorly understood. We aim to investigate the cellular mechanisms implicated in the monitoring of body metabolic states by IC and to identify the cortical microcircuits that link these mechanisms with complexes behaviors. Whole-cell electrophysiological recordings from acute slices of mouse IC combined with histological techniques enabled us to characterize the biophysical, morphological, and molecular identity of IC neurons and their patterns of responses to metabolic signals. A subpopulation of IC neurons was sensitive to changes in extracellular glucose concentrations with either a glucose-inhibited or a glucose-excited phenotype. We further showed that glucose responsiveness is an intrinsic property of some of these cells. We are now looking at the cellular mechanisms involved in these responses.

P11
Calogenetic balance, an educational program for long-term weight control based on measured resting metabolic rate and intake of favorite foods, promotes motivation and success rate in lean, overweight and obese participants

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Introduction: Recommendations for daily caloric intake are usually based on calculated mean values, which can markedly and unpredictably differ from the real ones. This discrepancy can create confusion and facilitate undesirable weight gain even eating healthy food. Calogenetic Balance was designed to improve these conditions by using individually measured resting metabolic rate (RMR) to assess personal daily caloric needs.
Methods: Lean, overweight or obese participants (180 women, 27 men) were referred by physicians or recruited via media/seminars. The program included seminars on energy balance and regular nutritional counseling. RMR was measured via indirect calorimetry. Daily nutritional schedules included individual food preferences, adapted if needed to standard nutritional rules. Daily caloric deficit was kept below 700 kcal.
Results: Initial BW (kg) were: 63.6 ± 4.7 (n = 21) lean; 77.5 ± 6.6 (n = 81) overweight and 94.7 ± 11.3 (n = 105) obese. After 6-months BW was reduced by 2.7 ± 1.6 (lean), 4.7 ± 2.2 (overweight) and 6.4 ± 3.3 (obese). In a subgroup of obese patients followed for 12 months BW was reduced by 9.0 ± 3.7 (n = 32).
Conclusions: The most relevant subjective statement by all participants was that the familiarity with the own real metabolic capacity gives security, removes confusion and promotes motivation and adherence. The observed consistent reduction in BW indicates, that an educational program like Calogenetic Balance will contribute to prevent and cure obesity by maintaining any local and individual nutritional habits.

P12
Red wine increases activity of nitric oxyde synthase in experimental animals

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Background and aims: There is a growing scientific concern on possible use of bioactive compounds in the prevention and treatment of cardiovascular and metabolic diseases. Red wine is among foodstuffs widely distributed in the human diet known as a source of plant bioactive compounds such as polyphenols. To date several studies have proposed the mechanisms by which red wine could exert its beneficial effects toward cardiovascular disorders. These mechanisms mainly involve increased vasorelaxation and blood pressure reduction by enhancing nitric oxide synthase (NOS) activity and nitric oxide production. The objective of this study was to investigate the effects of this red wine extract on nitric oxide synthase (NOS) activity and blood pressure development and in two groups of experimental animals, spontaneously hypertensive rats (SHR) and normotensive Wistar Kyoto rats (WKY).
Methods: In experimental animals plasma catechin levels as a principal polyphenolic compound were measured. Young 6-week-old male SHR and WKY rats were treated with red wine extract (24.2 mg/kg/day) for 3 weeks. Systolic blood pressure was measured by tail-cuff plethysmography. Total NOS activity was determined in the heart, aorta and kidney.
Results: SHR and WKY animals treated with the red wine extract show the concentrations catechin in plasma up to 4.7 mg/L, whereas untreated control rats have in their plasma very low concentrations of catechins or below the detection limit either. There were no significant differences in the plasma concentrations of catechins between treated SHR or WKY rats. The red wine extract increased NOS activity in the heart, aorta and kidney of SHR, while it did not change NOS activity in WKY rats. At the same time, wine extract treatment had no significant effect on blood pressure development in WKY and SHR.
Conclusions: This study has confirmed that polyphenols from red wine enters the blood circulation and stimulate NOS activity.

P13
Energy gap assessment in the aetiology of the obesity epidemic in the UK and USA: from multiple assumptions to flawed conclusions

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Objective: To critically re-evaluate recent studies assessing, by the Food Balance Sheet (FBS) method, the magnitude of the « energy gap » in the context of the obesity epidemic and their conclusions that the rise in food supply was more than sufficient to explain the epidemic of obesity in UK women and in US men and women.
Methods: Two studies were revisited: Scarborough et al. (Br J Nutr, 2010, 105: 1–6) and Swinburn et al. (Am J Clin Nutr 2009; 90: 1453–6). Both studies used a semi-epidemiological, semi-physiological and semi-nutritional approach to predict the average weight gain in two populations (UK and the USA, respectively).
Results: Based on our re-analysis, the increased body weight in the UK may be due to both an increase in energy intake and a reduction in physical activity in women and men, rather than only in men as Scarborough et al. have reported. Furthermore, when the data of Swinburn et al. in the US population were revisited using food intakes data generated in the same sample of subjects rather than based on US Food Balance Sheet, the rise in food intake alone was not sufficient to explain the US epidemic of obesity, thereby suggesting that increased sedentarity also played a concomitant role.
Conclusions: Major flaws in study findings can arise when conclusions are based on data pooled from different heterogeneous sources, when food intake is crudely estimated (FBS) and when most of the variables are roughly estimated, rather than measured in the same population.

P14
Heterogeneity in the energy cost of standing posture maintenance: are energy-savers a minority or the majority?

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With the health risks associated with sedentary behavior now firmly established and the search underway for a panacea to the surging prevalence of lifestyle-related disease, low-level physical activity has become a burgeoning area of research. Such research is often focused on ways of decreasing time spent sitting or lying in order to increase overall energy expenditure. The key dogma of this work is the notion that different postures (i.e. sitting, standing) vary in terms of energetic cost, and therefore simply altering posture allocation will modify overall energy expenditure (EE) and impact on body weight regulation and health. However direct evidence in support of this notion is equivocal. Recently, we have demonstrated heterogeneity in the energetic cost of standing versus sitting in a group of healthy, young European adults (1). Here we expand on this work to demonstrate that the vast majority of males (n = 21/27) in a student population of mixed ethnicity show little/no increase in EE during standing relative to sitting, or only an acute increase followed by a rapid return to baseline, sitting EE values. The mechanism by which the majority of individuals appear to maintain standing posture at the same energetic cost to sitting remains to be elucidated but is of immense importance to our understanding of the spontaneous physical activity compartment of energy expenditure and its potential as a target for weight regulation.
1. Miles-Chan et al. 2013 *PLoS ONE* **8**(*5*): e65827.

P15
Whole-body vibration in obese women improves body composition and aerobic fitness and decreases cardio-metabolic risk factors

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Introduction: Substantial decrease in body fat and increase in aerobic fitness is difficult to observe in obese subjects due to poor compliance to exercise. Whole-body vibration (WBV) training has positive effects on both body composition and VO2max but the application of this technique to obese patients remains mitigated.

Methods: 29 obese women were randomly allocated to 2 groups: WBV (n = 16) and non-exercising control group (C, n = 13). On the vibration platform, dynamic exercises were performed under supervision 3 times per week (session duration ~30 min.) for 12 weeks. Total and regional body composition was assessed by whole body and segmental BIA at baseline and after training.
Results: In the WBV group, body weight and total body fat decreased by 2.9 ± 0.2 kg & 2.2 ± 0.1 kg respectively ($p < 0.05$). Waist circumference decreased by 8.0 cm ± 1.6 ($p < 0.001$). in WBV & VO2max increased by 14.5% in WBV (17.9 ± 2.1 to 20.5 ± 2.7 mLO_2kg^{-1} min^{-1}, $p > 0.01$). Blood pressure dropped by 13.4 ± 8.7 and 9.4 ± 6.0 mmHg ($p > 0.01$) for systolic vs. diastolic, respectively. In the C group, no significant changes were observed for all above variables.
Conclusion: WBV training constitutes a promising type of physical activity for obese women accompanied with a substantial reduction in cardiovascular and metabolic risk factors, and improvement in cardio–respiratory fitness level. Additional studies over a longer duration, with more obese subjects of various obesity phenotypes (including both gender) are required to confirm the excellent s and compliance observed with WBV training.

P16
Are resting energy expenditure and fat oxidation increased in response to water drinking? A sham-controlled study in young men

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Drinking large amounts of water is often recommended for weight control – a notion which is supported by reports that drinking 500 mL of water increases resting energy expenditure (REE) by 20–30% on average during 30–90 min post-ingestion. These findings are, however, inconsistent with other human studies reporting marginal or no significant thermogenic response to similar (or even greater) water load. Moreover, there is also controversy about whether water drinking might stimulate fat oxidation, assessed by a decrease in respiratory quotient (RQ) over time. Whether the RQ response is a time effect or a direct effect of water load is the focus of this study. One explanation for these conflicting results may reside in the fact that studies reporting substantial water-induced thermogenesis and fat oxidation utilized tap water or bottled mineral water, both of which contain ingredients (minerals, salts, pollutants, etc) which may be contributing to the thermogenic and fat oxidizing effects of water (H20). In experiments reported here in overnight fasted young men in whom REE was measured by ventilated hood indirect calorimetry before and for 2 h after sham drink or water ingestion (21°C), we found that drinking 500 mL of distilled water did not result in a significant increase in REE (1.4% on average; n = 17), but in a significant decrease in RQ (–0.031 on average, $p < 0.001$; n = 17). In a second study, we observed a small increase in REE (<3% on average) after both sham and distilled water drink. RQ diminished to the same extend over time in response to both water drink and sham drink ($p < 0.001$). Our results suggest that drinking 500 mL of purified water has marginal or no effect on REE (<3% increase). The results with sham drink suggest that the decrease in RQ as a time effect and not a water effect. Whether the presence of minerals/salts and/or sensorial aspects of tap or mineral water brands utilized in the other studies may explain differential thermogenic and fat oxidizing responses to 'water' remains to be investigated.

P17
Design and preliminary evaluation of a mobile application for obesity expert and children teams

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Childhood obesity is one of the major disease patterns of the twenty-first century. Due to the need for multi-professional therapies requiring intensive personnel and financial resources, IT-supported interventions promise help. Meta analyses, however, show their limited impact on health outcomes up till now. The current work aims therefore to design and evaluate a mobile application that increases the cooperation between obesity experts and children. For that purpose, four IT experts, five therapists, nine obese children 10 to 14 years old and their parents adopted a structured design-science methodology. Perceived characteristics of the application and direct effects on cooperation of therapists and children were evaluated. The resulting application provides recipe recommendations based on ingredients available at home and desired by children. It further allows to document groceries and meals via a photo functionality. All interactions with the application were recorded to document screen time and utilization for efficient shopping and healthy meals. First feedback from seven therapists, six children and their parents indicates that the application is perceived useful, easy and fun to use. With regard to direct effects on the cooperation between obesity expert and children teams, there is evidence that the application supports shared understanding and cross understanding. Future work will incorporate further components of therapy programs, such as physical activity or relaxation, but will also investigate in a longitudinal field study how the use of this application within a therapy program influences health condition of obese children.

P18
Mapping the food entrainable oscillator of mice

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Resetting the circadian timing system by food cues has given great importance to understand mechanistic relationship between food cues and clock genes at molecular level. The mealtime prediction in animals is revealed by food seeking activity (food-anticipatory activity ;FAA), body temperature, plasma corticosterone level. Under restricted feeding (RF) conditions, the increased locomotor activity 2–3 h preceding to food availability is called as FAA; and is behavioral manifestation of food entrainable oscillator (FEO). The food anticipatory activity analysis in clock mutant animals has revealed the relevance of clock genes to FEO. The FAA study in Per2Brdm1 has emerged with promising results. Lack of food anticipatory rhythms in Per2mutant mice has suggested Per2 as critical component in food entraining signals. On the complex background of FEO, the current project is aimed to site the FEOs and study food entrainable rhythms at molecular level. The FAA study in Per2Brdm1 led our attention to examine FAA in conditional KO mice, which might explore the sites for FEO. We examined FAA in Per2Δflx/Δflx (total KO) in restricted feeding condition. The Per2Δflx/Δflx was lack of FAA. In order to address long lasting mystery about the nature of FEO, the FAA will be examined in the FAA in Per2 liver specific (Lcre Per2–/–) and brain (neuron) specific (Nestin cre Per2–/–) KO mice.