Background/Aims: Insulin is one of the most potent anabolic agents involved in the macronutrients synthesis and storage. It plays a pivotal role in the development of obesity while within the central nervous system acts as a growth factor in synaptogenesis and nerve growth. Therefore the lack of insulin or presence of insulin resistance (IR) could potentially lead to cognitive decline and impaired learning. This review focuses on animal and human studies that assess the relationship between the IR and regulation of cognitive function and mood.
Methods: Literature searches were conducted on electronic databases examining the association between insulin resistance, glucose regulation, obesity, cognitive function and mood.
Results: In animal models, the central focus is on effects to the hypothalamic-pituitary-adrenal axis (HPA-axis), an area identified as the most likely area to influence mood and stress. Damage to the insulin receptors in this region was found to be associated with the increase in food intake and occurrence of adiposity. In humans, these areas play a central role in motivation and decision making. Furthermore, IR and major depressive illness share several pathologies, including disorders of the HPA-axis, the autonomic nervous system, platelets and endothelial function.
Conclusions: There is increasing evidence suggesting a close association between the obesity and mood disorders such as depression, anxiety, panic and bipolar disorders. However, there is no clear evidence of individual aspects that can be ascribed as pathological drivers of the problem.
Funding source(s): N/A.

IMPAIRED CEREBROVASCULAR RESPONSIVENESS TO A WORKING MEMORY TASK IN OLDER ADULTS WITH TYPE 2 DIABETES MELLITUS (T2DM)

R.S. Nealon, P.R.C. Howe, L. Jansen, M.L. Garg, R.H.X. Wong. *Clinical Nutrition Research Centre, School of Biomedical Sciences and Pharmacy, University of Newcastle, NSW, Australia*
E-mail address: rhenan.nealon@uon.edu.au (R.S. Nealon)

Background/Aims: Impairments in specific cognitive domains in T2DM may be partly attributable to stiffness in cerebral arteries, resulting in poor cerebral perfusion. This cross-sectional study investigated whether impairments in the ability of the cerebrovasculature to supply blood in response to a battery of cognitive tests could predict poorer cognition in T2DM.
Methods: Forty nine T2DM and 28 non-T2DM adults underwent transcranial Doppler ultrasound measurements of basal mean cerebral blood flow velocity (MBFV) and pulsatility index, a measure of arterial stiffness, in the left and right middle cerebral arteries (MCA). A battery of cognitive tasks assessing domains of working memory, executive function and information processing speed was then administered whilst MBFV was recorded. Cerebrovascular responsiveness (CVR) to cognitive tasks was calculated as a percentage increase in MBFV from the basal level.
Results: Using *t*-tests, we found no differences in basal MBFV; however, cerebral vessels were 14 percent stiffer in T2DM ($p < 0.05$). As expected, T2DM performed poorer in tasks relating to working memory (i.e. N-back, Digit-Symbol Coding, Symbol-Digit Coding), executive function (Concept Shifting Task) and information processing speed. Importantly, CVR to the N-back task was reduced by 53 percent in T2DM ($p < 0.05$) but was independent of task performance.
Conclusions: We have shown for the first time that impaired cerebral perfusion during a working memory task is accompanied by poor task performance. We plan to evaluate the ability of selected *vasoactive* nutrients to enhance cerebrovascular function and see whether this improves cognition in at-risk populations.
Funding source(s): N/A.

INCIDENCE AND RISK FACTORS OF TYPE 2 DIABETES: RESULTS FROM THE THAI COHORT STUDY

K. Papier [1], S. Jordan [2], C. D'est [1], C. Banwell [1], C. Bain [2], V. Yiengprugsawan [1], S. Seubsman [3], A. Sleigh [1]. [1] *National Centre for Epidemiology and Population Health, Australian National Uni., ACT, Australia;* [2] *Population Health, Queensland Institute of Medical Research QLD, Australia;* [3] *School of Human Ecology, Sukhothai Thammathirat Open Uni., Nonthaburi, Thailand*
E-mail address: keren.papier@anu.edu.au (K. Papier)

Background/Aims: The global prevalence of type 2 diabetes mellitus (T2DM) is high and increasing rapidly in countries undergoing a nutrition transition like Thailand. This study aimed to assess the relationship between T2DM and factors associated with the nutrition transition among Thai adults.
Methods: Data were from Thai Cohort Study participants surveyed in 2005, 2009 and 2013 ($n = 39{,}519$). Cumulative incidence of diabetes was calculated and multivariable analyses were conducted using logistic regression.
Results: T2DM incidence (per 1000) was higher in males (24.9 *vs.* 11.9). The factors most strongly associated with T2DM in both sexes were increasing age and BMI but, amongst males, smoking [Odds Ratio (OR) =1.70, 95%CI: 1.29-2.24] and alcohol intake (OR = 1.67, 95%CI: 1.00-2.82) were also associated with increased risk. Infrequent gardening, low vegetable intake, and urban childhood residence were also related to T2DM risk however these associations attenuated after adjusting for BMI. Among females, high income was associated with T2DM (OR = 1.72, 95%CI: 1.03-2.89). Urban childhood residence and education were also associated with T2DM however these associations were attenuated after adjusting for BMI.
Conclusions: The factors associated with T2DM risk in our study are consistent with findings from previous studies conducted in countries undergoing a nutrition transition. With the prevalence of these factors projected to increase it is likely that the incidence of T2DM will keep rising. This may be of particular concern for Thai men who appear to be in the earlier stages of the nutrition transition. Our study suggests that females are at a more advanced stage of the nutrition transition.
Funding source(s): NHMRC.

LAURIC ACID DIFFERS FROM OTHER SATURATED FATTY ACIDS IN METABOLIC SYNDROME IN RATS

S. Shafie [1,2], L. Brown [1]. [1] *School of Health & Wellbeing, Faculty of Health, Engineering & Science, Uni. of Southern Queensland, QLD, Australia;* [2] *Dept. of Nutrition & Dietetics, Faculty of Medicine & Health Sciences, Universiti Putra Malaysia, Malaysia*
E-mail address: sitiraihanah.shafie@usq.edu.au (S. Shafie)

Background/Aims: The aim of this study is to evaluate different saturated fatty acids on cardio-vascular, liver and metabolic responses in rats.
Methods: Rats were fed 20% lauric (HLA), myristic (HMA), palmitic (HPA) or stearic (HSA) acids or beef tallow (HCHF) for 16 weeks with increased fructose and condensed milk. Control rats were fed a corn starch (C) diet. Food and water consumption, body weight, body composition, heart stiffness, blood pressure, blood glucose, lipid profiles and liver function of rats were measured.
Results: Final body weight ranked HLA < C < HMA = HPA = HSA < HCHF rats with HLA rats showing 11.1% decrease. Total fat mass reflected changes in body weight with HLA (54.4 ± 3.2 g) < C (79.5 ± 11.0 g) < HPA (122.7 ± 12.9 g) = HSA (122.9 ± 9.5 g) = HMA (132.4 ± 9.9 g) < HCHF rats (207.7 ± 27.2 g). Left ventricular diastolic stiffness (κ) was similar in control (22.0 ± 0.5) and HLA rats (21.8 ± 1.0) but less than HMA (25.3 ± 0.7), HPA (26.6 ± 0.7), HSA (27.0 ± 0.4) and HCHF rats (28.2 ± 0.5). Systolic blood pressure increased with C (127.7 ± 1.1 mmHg) < HLA (136.2 ± 5.3 mmHg) = HMA (141.7 ± 1.2 mmHg) < HPA (150.4 ± 2.5 mmHg) = HSA (152.9 ± 3.5 mmHg) = HCHF rats (157.8 ± 2.8 mmHg). HLA rats showed improved glucose tolerance, insulin sensitivity and attenuated dyslipidaemia compared to HMA, HPA, HSA and HCHF rats. Plasma liver enzymes increased in HLA, HMA, HPA, HSA and HCHF rats compared to C rats.
Conclusions: For most parameters, lauric acid produced less pathophysiological changes than other saturated fatty acids in this model of diet-induced metabolic syndrome.
Funding source(s): University of Southern Queensland.

HIGHER VITAMIN D STATUS IS INVERSELY ASSOCIATED WITH THE METABOLIC SYNDROME AND RISK OF T2DM IN VICTORIAN ADULTS

P.K. Pannu [1], Y. Zhao [2], M.J. Soares [1], L.S. Piers [3], Z. Ansari [3]. [1] *Directorate of Nutrition, Dietetics & Food Science, School of Public Health, Curtin University, WA, Australia;* [2] *Directorate of Epidemiology & Biostatistics, School of Public Health, Curtin University, WA, Australia;* [3] *Health Intelligence Unit, System Intelligence and Analytics Branch, Dept. of Health, VIC, Australia*
E-mail address: m.soares@curtin.edu.au (M.J. Soares)

Background/Aims: Vitamin D may have a protective role in the development of many chronic diseases, including T2DM. The aim of this study was to investigate the association between serum 25OHD, the risk of T2DM and presence of the metabolic syndrome (MetS).
Methods: We analysed the 2009-2010 Victorian Health Monitor survey where information on 25OHD, HBA1c, fasting plasma glucose and presence of the metabolic syndrome were available. Logistic regression analyses were used to evaluate the association between tertiles of serum 25OHD and HbA1c (< 5.7% *vs.* ≥ 5.7%), fasting plasma glucose (< 5.6 mmol/L *vs.* ≥ 5.6 mmol/L) and MetS (yes/no). Confounders tested were age, gender, country of birth, income, education, physical activity, smoking status, season, weight, calcium, magnesium, energy intake, television viewing time, sitting time, waist circumference, BMI, and intakes of alcohol, total fat, carbohydrate, protein, retinol, fibre and caffeine.
Results: A total of 3434 Australian adults excluding those with T1DM and T2DM entered the analysis. Greater 25OHD had a lower OR of being associated with the presence of MetS [OR 0.36 (95% CI; 0.27, 0.48), $p = 0.005$], lower OR of a higher HbA1c [OR 0.62 (95% CI; 0.49, 0.77), $p = 0.004$], and a lower OR of higher fasting plasma glucose [OR 0.55 (95% CI; 0.39, 0.77), $p = 0.004$].
Conclusions: Higher 25OHD may have a beneficial effect on MetS and risk of T2DM.
Funding source(s): N/A.

THE EFFECT OF VITAMIN D ON CHRONIC PLAQUE PSORIASIS: A RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED SUPPLEMENTATION TRIAL

M.A. Ingram [1], B. Jones [1], W. Stonehouse [2], P. Jarrett [3,4], R. Scragg [3], P. von Hurst [1]. [1] *Massey University, New Zealand;* [2] *CSIRO, Adelaide, SA, Australia;* [3] *University of Auckland, New Zealand;* [4] *Dept. of Dermatology, Middlemore Hospital, Auckland, New Zealand*
E-mail address: welma.stonehouse@csiro.au (W. Stonehouse)

Background/Aims: Vitamin D has anti-proliferative, pro-differentiative and immune-modulating effects. We aimed to determine whether raising serum 25(OH)D through vitamin D3 supplementation improves psoriasis.
Methods: In a randomised (2:1), double-blind, placebo-controlled trial, 101 participants > 18 y with plaque psoriasis took 200,000 International Units (IU) of cholecalciferol at baseline then 100,000 IU/month for 11 months ($n = 67$), or placebo ($n = 34$). Psoriasis Area and Severity Index (PASI) and serum 25(OH)D concentration were assessed at baseline, 3m, 6m, 9m and 12m. Primary outcomes were a) difference in PASI between groups over time, and b) the relationship between PASI and 25(OH)D over time, assessed by linear mixed models adjusted for confounding and/or individual factors.
Results: There was a significant inverse relation-ship between 25(OH)D and PASI. Elevating serum 25(OH)D by increments from 25 − 125nmol/L was associated with mild decreases in PASI (estimated range of decrease 0 − 2.6; $p = 0.002$). PASI did not differ by group ($p = 0.62$, group*time $p = 0.54$), and an improvement in PASI of 50% or higher was achieved by 11.9% of treatment and 11.8% of placebo. However, mean 25(OH)D significantly increased from baseline at 3m for treatment [$b = 33$ (95% CI 28 − 38) nmol/L, $p < 0.001$] and 6m for placebo [$b = 24$ (95% CI 17 − 30) nmol/L, $p < 0.001$], possibly confounding these results.
Conclusions: At a population level, elevating serum 25(OH)D is associated with improved psoriasis. Estimated improvements were mild at 25(OH)D concentrations in this study and may not be clinically significant; higher concentrations could have greater benefit.
Funding source(s): Lottery Health Research, Massey University.

ACUTE EFFECT OF RED MEAT AND DAIRY ON GLYCEMIC RESPONSE

K.M. Turner, J.B. Keogh, P.M. Clifton. *University of South Australia, SA, Australia*
E-mail address: kirsty.turner@unisa.edu.au (K.M. Turner)

Background/Aims: In contrast to some epidemiological evidence our previous research found that a 4-week diet high in dairy reduced insulin sensitivity compared to one high in red meat. Our aim was to investigate whether a dairy meal produced lower glucose compared with a carbohydrate-matched red meat meal.
Methods: A total of 19 men and 24 women (age 50.8 ± 16.0, BMI 30.0 ± 3.5 kg/m^2) completed the randomized crossover study. Twenty-two participants had normal glucose tolerance; 21 had impaired fasting glucose and/or impaired glucose tolerance. One meal contained lean red meat, bread and orange juice and the other milk, yoghurt, cheese and bread. Meals were isoenergetic, equal in macronutrient profile and consumed one week apart. Glucose and insulin were measured before and 30, 60, 90, 120, 150 and 180 minutes after consuming the meal. Difference between meals was tested using repeated measures ANOVA and paired sample T-tests.
Results: The red meat meal resulted in a higher glucose response at T_{30} ($p < 0.001$) however total glucose AUC was not different between meals ($p =$ NS). Incremental AUC for glucose was significantly higher after the dairy meal (2.23 ± 3.19 mmol/L *vs.* 0.88 ± 3.73 mmol/L, $p = 0.004$). Total and incremental insulin AUC was not different between meals (iAUC 159.65 mU/L for red meat, *vs.* 167.49 mU/L for dairy, $p =$ NS).
Conclusions: Lean red meat and dairy meals produced a similar glycemic response. The higher glucose response 30 minutes after the red meat meal is likely attributable to differences in glycemic load between orange juice and milk or yoghurt. An insulinotropic effect of dairy was not observed.
Funding source(s): University of South Australia.

POTENTIAL OF DIETARY PROPOLIS IN PROTECTING BOVINE MAMMARY EPITHELIAL CELLS AGAINST MASTITIS PATHOGENS USING *IN VITRO* MODELS

K. Wang, X.L. Jin, J. Liu, F. Hu. *College of Animal Sciences, Zhejiang University, Hangzhou, China*
E-mail address: kaiwang628@gmail.com (K. Wang)

Background/Aims: Inflammation of the mammary gland (mastitis) caused by invading pathogens is common among lactating dairy cows and causes great economic losses. We recently demonstrated that Chinese propolis (CP), a resinous substance collected by honeybees, can positively influence anti-inflammatory processes and antioxidant defense systems *in vivo* but little is known about how propolis treatment of mastitis in dairy cows may influence these activities.
Methods: We investigated the potentially protective effects of CP on aspects of mastitis by using an *in vitro* model of mastitis-induced cell damage involving cultured bovine mammary epithelial cells (MAC-T) which received a range of mastitis pathogen-related insults. Cell viability was measured by cell counting kit (CCK)-8 cell viability assay and expression of inflammatory or antioxidant genes were measured by qPCR. Using a cell-based reporter assay system, we evaluated CP and its main constituents' on NF-κB and Nrf2-ARE transcription activity.
Results: Treatment of cells with cell wall components lipopolysaccharide (LPS), heat inactivated *E. coli* and *S. aureus*, but not TNF-α or lipo-teichoic acid (LTA), significantly decreased cell viability ($p < 0.01$). CP pretreatment (15 μg/mL) significantly ($p < 0.05$) protected cell viability losses. CP pretreatment of MAC-T cells resulted in less impact on IL-6 and TNF-α mRNA expressions but increased expression of antioxidant genes *HO-1*, *Txnrd-1* and *GCLM*. CP and its polyphenolic active components (mainly CAPE and quercetin) had strong inhibitive effects against NF-κB and increased the transcriptional activation of the Nrf2-ARE pathway.
Conclusions: Our findings support the usage of dietary propolis for the treatment of bovine mastitis.
Funding source(s): National Science Foundation of China.

PREDICTING BODY COMPOSITION IN BREASTFED TERM INFANTS

L.C. Ward [1], Z. Gridneva [2], A.R. Hepworth [2], C.T. Lai [2], P.E. Hartmann [2], D.T. Geddes [2]. [1] *School of Chemistry and Molecular Biosciences, University of*