insulin is the only effective treatment, but there is still no cure or interventional therapy available to inhibit progression of T1D. Successful T1D interventional therapy must protect pancreatic beta cells from autoimmunity while enhancing beta cell survival and function. Our data suggests *Cornus officinalis* (CO) *may be a candidate for* interventional therapy to protect pancreatic beta cells from autoimmune attack and increase their function. CO has been used in traditional Chinese medicine (TCM) for over 2,000 years and has shown characteristics of anti-diabetic effects in vitro and in vivo but never examined in the application of T1D. Our prior publication (*Mol. and Cell. Endo.*2019:494:110491), has shown increased proliferation and protection against Th1 cytokine attack upon CO treatment using a human pancreatic beta cell line, 1.1B4. From this, we sought to define precise molecular mechanism by employing a global and phosphorylation mass spectrometry (MS) approach. We applied CO to 1.1B4 cells for 2, 6, 12, and 24h then collected the cell lysates for MS analysis. Our global MS analysis revealed a 12-fold increase in beta cell functional regulator, IGFBP2, at multiple time points. IGFBP2 has been shown to display a T2D protective effect and regulate glucose metabolism. The ingenuity pathway analysis program (IPA) predicted an increase in insulin starting at 2h and the NRF2-mediated oxidative stress pathway at 12h and 24h. Furthermore, NRF2 is an upstream regulator of P62 which was significantly hyperphosphorylated at multiple timepoints from our MS analysis. Nrf2 is responsible for activating antioxidant enzymes upon oxidative stress, which is caused by proinflammatory cytokines in T1D. P62 aids in this pathway by targeting proteins for autophagy upon oxidative stress in order to keep cellular homeostasis within beta cells rather than cells progressing through apoptosis. Autophagy is critical for beta cell function and survival as it promotes survival under beta cell stress which would otherwise lead to cell death. The recovery and protection of autophagy in beta cells of patients in the pre-diagnosed stages of T1D could provide a beneficial interventional therapy in order to delay or inhibit the onset of T1D. Altogether, our proteomic analysis revealed an increase in IGFBP2 and predicted an increase in the NRf2-mediated oxidative stress pathway upon CO induction. Further analysis will examine the IGFBP2 and Nrf2-mediated oxidative pathway as a mechanism of CO induced protective and proliferative effects in pancreatic beta cells.

Reproductive Endocrinology

HYPERANDROGENISM

Metformin-Fish Oil Adjunct Therapy Improves apoB-Remnant Lipoprotein and Triglyceride Levels in Women with Polycystic Ovary Syndrome

Ethan Proctor, BSc[1], Olivia Weaver, BSc[1], Mahua Ghosh, Dr, PhD, FRCPC[2], Katerina Maximova, Dr, PhD[1], Spencer Proctor, Dr, PhD[2], Donna Vine, Dr, PhD[1].

[1]Univ of Alberta, Edmonton, AB, Canada, [2]University of Alberta, Edmonton, AB, Canada.

SUN-022

Background: Polycystic ovary syndrome (PCOS) is highly associated with the metabolic syndrome (MetS): obesity, insulin resistance and atherogenic dyslipidemia. Women with PCOS-MetS are at higher risk of developing ischemic cardiovascular disease (CVD) and Type-2 Diabetes. First-line intervention in PCOS-MetS includes targeting diet and lifestyle, and metformin is commonly prescribed to treat insulin resistance, however these interventions have shown limited effectiveness to improve dyslipidemia. At present there are limited safe and efficious options to target atherogenic dyslipidemia in young women with PCOS. Fish oil (FO) and Icosapentyl ethyl supplementation have been shown to reduce fasting TG, apoB and to improve ischemic CVD outcomes. The efficacy of FO or as an adjunct therapy to metformin to improve ApoB-remnant lipemia in PCOS-MetS is unknown. The aim of this pilot study was to determine the effect of metformin, FO and FO-metformin combination treatment on fasting and non-fasting plasma TG and apoB-remnant lipoprotein metabolism in patients with PCOS-MetS.

Methods: Participants diagnosed with PCOS aged 18-30yrs received dietary counselling and were randomly assigned to receive FO (n=8), metformin (n=7) or FO-metformin (n=12) treatment for 12 wks. Plasma lipids (TG and cholesterol), ApoB48 and ApoB100 lipoprotein metabolism were assessed in the fasting and non-fasting state using a standardized high-fat meal test.

Results: At baseline, the fasting plasma TG, ApoB48 and ApoB100 was 238.0 ± 21.0 mg/dL, 9.00 ± 1.12 ug/ml and 290 ± 18.00 mg/dL. FO and FO-metformin decreased fasting plasma TG by 10% and 30% compared to the metformin treatment group (7%). Fasting ApoB48 was reduced 45%, 16% and 19% in FO-metformin, FO and metformin treatment groups, respectively. Non-fasting plasma TG and apoB48 lipoprotein area under the curve were reduced by 30% in the FO-metformin treatment group.

Conclusion: These pilot findings demonstrate FO-metformin adjunct therapy may have greater efficacy to improve atherogenic apoB-dyslipidemia compared to metformin or FO alone in high-risk patients with PCOS-MetS. A larger clinical trial is warranted to determine the long term effects of FO-metformin intervention on apoB-dyslipidemia and atherosclerotic cardiovascular disease indices.

Reproductive Endocrinology

MALE REPRODUCTIVE HEALTH - FROM HORMONES TO GAMETES

Changes in Metabolic Parameters After Administration of Novel Oral Androgens with Progestational Activity for 28 Days

Fiona Yuen, MD[1], Arthi Thirumalai, MBBS[2], Ronald S. Swerdloff, MD[1], Peter Y. Liu, PHD,MBBS[1], Youngju Pak, PhD[1], Laura Hull, BS[1], Stephanie T. Page, MD, PhD[2], Christina Wang, MD[1].

[1]The Lundquist Institute, Torrance, CA, USA, [2]University of Washington, Seattle, WA, USA.

SAT-040

Background: While the metabolic effects of testosterone have been well studied, the effects of co-administration of an androgen and progestin are less established. Two novel compounds being investigated for male hormonal contraception, dimethandrolone undecanoate (DMAU)