parameters in a manner that is independent of HO-1 activity. Further studies are warranted to identify the underlying mechanisms that may reveal new molecular targets for the treatment of obesity and associated diseases.

Reference

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Invited talk: Treating diabetes and obesity using the gut microbiome involves dietary diversity

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Background: A healthy gastrointestinal microbiome is a diverse microbiome and results from a diverse diet. During the past 50 years, 75% of the world's dietary diversity has been lost. One can look for gut dysbiosis in disease and give foods to correct the dysbiosis or use rare, heirloom foods to increase microbiome diversity as two strategies to treat disease by acting on the gastrointestinal (GI) microbiome.

Methods: The microbiome in diabetes is low in short chain fatty acid (SCFA) production, has increased GI inflammation and produces excess methane. NM-504 contains inulin, beta-glucan and blueberry pomace to address the SCFA, GI inflammation and methane abnormalities, respectively. Soy pod fiber can be stimulated to make glyceollin which increases microbiome diversity.

Results: NM-504 reduced blood sugar in a clinical trial to a similar degree as sitigliptin, a DPP-4 inhibitor. NM-504 protected the GI mucosal barrier from inflammation, reduced hsCRP, reduced appetite, and had no adverse events while increasing bowel regularity. NM-504 also reduced the GI side effects associated with metformin. Young soy pods activated to make glyceollin by cutting was fed to mice with dietary obesity. The mice ate more food, but lost weight and systemic inflammation was reduced. Faecal fat was increased, but there was no oil in faeces. The activated soy contains an FXR agonist, reduces inflammation, faecal bile acids, bile acid transport and decreases microbiota making antagonists of bile acids in the gut.

Conclusion: Increasing the diversity and correcting the dysbiosis of the GI microbiome in disease can be used in the treatment of diabetes, metformin intolerance, obesity and possibly non-alcoholic fatty liver disease.

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Early weight loss responders to liraglutide 3.0 mg had greater weight loss, regression to normoglycaemia, and reduced T2D development at 3 years vs early non-responders: SCALE Obesity and Prediabetes

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Background: The SCALE Obesity and Prediabetes (NCT01272219) trial randomised adults with prediabetes and obesity (BMI $\geq$ 30 kg/m^2) or overweight with comorbidities ($\geq$27 kg/m^2; dyslipidaemia/hypertension) to liraglutide 3.0 mg (N = 1505) or placebo (N = 749) as adjunct to diet and exercise for 3 years.

Methods: This *post-hoc* analysis compared liraglutide 3.0 mg early responders (ERs; $\geq$5% weight loss [WL] at Week [W] 16) and early non-responders (ENRs; <5% WL at W16), in keeping with EMA and Australian stopping-rule criteria. Efficacy outcomes are estimated means in ERs (n = 580) and ENRs (n = 210) who completed 160 weeks' treatment. Development of T2D/regression to normoglycaemia were analysed using the full analysis set with LOCF. Safety was analysed using the safety