Basic Science E-Poster Presentation

EP_B001

NIRMATRELVIR/RITONAVIR-INDUCED HYPONATREMIA PRESENTING WITH SIADH AND GASTROINTESTINAL LOSS: A CASE REPORT

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INTRODUCTION/BACKGROUND
Nirmatrelvir/ritonavir (Paxlovid™) is an antiviral used to treat mild to moderate COVID-19 infection. Hyponatremia in COVID-19 has been documented in several case reports but none related to treatment with nirmatrelvir/ritonavir to date.

CASE
A 75-year-old female with diabetes mellitus, hypertension and bronchial asthma was treated as COVID-19 Category 2b with symptoms of fever, diarrhoea and poor oral intake. Serum sodium on admission was 138 mmol/L. She was given nirmatrelvir/ritonavir and intravenous drip due to ongoing gastrointestinal losses. Her symptoms resolved in the next two days. On day 7 of illness, she started having new-onset diarrhoea. Repeat Na was 112 mmol/L. Further hydration led to declining Na levels until she became symptomatic at 105 mmol/L. Her paired sample results suggested SIADH, with serum osmolarity 228 mOsm/kg, urine osmolarity 233 mOsm/kg and urine Na 84 mmol/L despite ongoing gastrointestinal losses and clinical signs of hypovolemia. Nirmatrelvir/ritonavir was already discontinued. She was treated with hypertonic 3% saline until her symptoms improved. Her diarrhoea resolved, and her Na levels normalized with fluid restriction and oral salt treatment.

CONCLUSION
Nirmatrelvir/ritonavir has been reported to cause elevated transaminases, hypertension and diarrhoea, apart from its known drug-drug interactions. This case highlights the importance of SIADH as one of the possible adverse effects of this new drug which is now widely used for the management of COVID-19. It is important for clinicians to be vigilant about drug-induced SIADH when investigating the cause of hyponatremia in patients with COVID-19.

EP_B002

INHIBITION OF LIPID ACCUMULATION AND UPREGULATION OF BROWNING GENES BY SIRT1 ACTIVATION IN 3T3-L1 CELLS

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INTRODUCTION
Obesity is the cause of over 4 million deaths each year according to the Global Burden of Disease study. It is characterized by an increase in adipocyte number and size, making adipogenesis an important target in the prevention and management of obesity. A possible approach is to induce adipose cells into becoming thermogenesis-competent, such as brown or beige adipose cells. In this study, the effect of activating Sirtuin1 (Sirt1) in 3T3-L1 and its role in promoting the browning of white adipose cells was investigated.

METHODOLOGY
White pre-adipocyte cell lines 3T3-L1 were treated with Sirt1 activator (SRT1720) 2.5 mM, Sirt1 inhibitor (EX527) 10 mM or rosiglitazone (positive control for adipogenesis) 50 mM throughout ten days of adipogenic differentiation period. The effect of these treatments was assessed by western blotting, oil red O lipid staining and real-time PCR.

RESULTS
Inducing Sirt1 activity affected intracellular lipid accumulation. This was based on the observation of lesser accumulation of lipid in Sirt1-activated cells stained with oil red O. In contrast, when Sirt1 activity was attenuated by Sirt1-specific inhibitor, lipid production increased. Further investigation showed that the expression of genes critical for brown adipose tissue regulation, such as PGC1a and Cidea, were found to be elevated when Sirt1 activity was induced.

CONCLUSION
This work shows that targeting Sirt1 activity in white adipose cells would instigate an underlying molecular network that modulates the adipogenesis process during cell differentiation by producing less lipid. It also promotes the cells to differentiate into brown-like adipose cells that