GUEST LECTURE SERIES

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Deactivating the Off Switch: Leveraging Energy Expenditure in the Management of Obesity-Related Disorders

The prevalence of obesity-related disorders continues to increase, but current management options remain limited. Brown adipose tissue (BAT) is rich in mitochondria and mediates non-shivering thermogenesis, playing key role in human energy expenditure. Interventions that increase BAT activity are expected to mitigate obesity. Thioesterase superfamily member 1 (Them1; synonyms acyl-CoA thioesterase 11 and StarD14) is a fatty acyl-CoA thioesterase that is highly expressed in BAT and strongly suppresses energy expenditure. Studies from our laboratory have demonstrated that genetic disruption of Them1 in mice leads to increased energy expenditure, along with resistance to diet-induced obesity, diabetes and non-alcoholic fatty liver disease (NAFLD). *These findings suggest that chemical inhibition of Them1 could be leveraged in the management of NAFLD.*