regulation of appetite and satiety, leading to increased food intake and abnormal eating behavior. For instance, abnormal central estrogen signaling (such as decreased activity of amygdala aromatase) can enhance the motivation to eat and the amount of food consumed, making it more likely for women to gain weight (Maric et al., 2025). Furthermore, irregular eating (such as eating at night) intensifies hunger signals, alter the levels of appetite-related hormones, and makes adipose tissue more inclined to store fat rather than break it down, thereby accelerating fat accumulation (Vujovic et al., 2022).

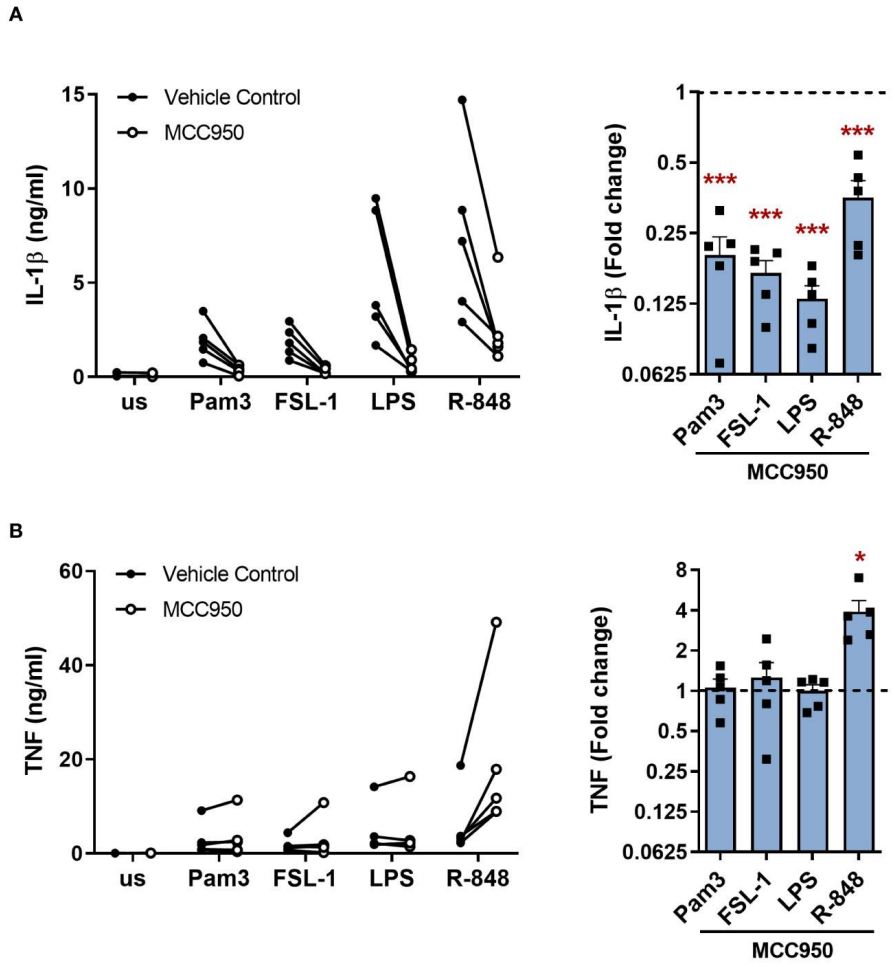

Figure 2 NLRP3 is required for TLR induced release of IL-1β in primary human monocytes (Adopted from Unterberger et al., 2023)
Image caption: Monocytes were unstimulated (us) or stimulated for 24h with 100 ng/mL Pam3, 1ng/mL FSL-1, 10 ng/mL LPS or 2 μg/mL R-848 in the presence of 10μM MCC950 or a vehicle control; Secretion of (A) IL-1β and (B) TNF were measured. Data are displayed as mean of technical triplicates (left) and as the fold change normalised to the corresponding TLR activation without MCC950 (dotted line) and pooled as the mean±SEM (right) from 5 individual donors; Significance was determined using two-tailed one sample t-test against the response without MCC950 treatment (*$p \leq 0.05$, ***$p \leq 0.001$) (Adopted from Unterberger et al., 2023)

In addition to increased intake, decreased energy expenditure and heat production capacity can also contribute to the progression of obesity. Weakened IL-6 signaling in the brain and impaired brown fat function are closely related to reduced energy expenditure and weight gain. Meanwhile, impaired central thermoregulatory pathways (such as decreased function of the cold-sensing TRPM8 channel or decreased IL-6 level in the lateral arm nucleus) will further inhibit thermogenesis of brown fat, resulting in a continuous tilt of energy balance towards weight gain (Mishra et al., 2019).

6.2 Increased liver glycogen output and abnormal lipid metabolism

Metabolic syndrome is often accompanied by disorders of glucose and lipid metabolism, with the core mechanisms being insulin resistance and chronic inflammation. Insulin resistance weakens the effect of insulin in inhibiting liver glucose production, resulting in increased liver glucose output and elevated fasting blood glucose