

**Title:** Postprandial muscle protein synthetic responses after high quality pork consumption in lean, overweight, and obese adults – **NPB #14-205**

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### Scientific Abstract:

**Background:** Excess body fat leads to diminished muscle protein synthesis rates in response to a hyperinsulinemic hyperaminoacidemic clamp. To our knowledge, no studies have compared the postprandial muscle protein synthetic response after the ingestion of a single meal containing a protein dense food source across a range of body mass indices and fat masses. **Objective:** We aimed to compare the myofibrillar protein synthetic (MPS) response and underlying nutrient sensing mechanisms after the ingestion of lean pork loin between obese, overweight, and healthy-weight adults. **Design:** 10 healthy-weight (HW; Age 24±1 y, BMI 22.7±0.4 kg/m<sup>2</sup>, HOMA-IR 1.4±0.2), 10 overweight (OW; Age 26±2 y, BMI 27.1±0.5 kg/m<sup>2</sup>, HOMA-IR 1.25±0.11), and 10 obese males and females (OB; Age 27±3 y, BMI 35.9±1.3 kg/m<sup>2</sup>, HOMA-IR 5.8±0.8) received primed continuous L-[ring-<sup>13</sup>C<sub>6</sub>]phenylalanine infusions. Blood and muscle biopsy samples were collected before and after ingestion of 170 g of pork (36 g protein and 5 g fat) to assess skeletal muscle anabolic signaling, amino acid transporters (LAT1, CD98, SNAT2), and MPS. **Results:** At baseline, OW and OB showed greater relative amounts of mTORC1 protein compared to the HW group. However, pork ingestion only increased phosphorylation of mTORC1 in the HW group ( $P=0.001$ ). LAT1 and SNAT2 protein content increased during the postprandial period in all groups (Time effect:  $P<0.05$ ). Basal MPS were not different between groups ( $P=0.43$ ). However, postprandial MPS (0-300 min) was greater in the HW group (1.6-fold;  $P=0.005$ ) after pork ingestion, compared with the OW and OB groups. **Conclusions:** There is diminished responsiveness of postprandial MPS to the ingestion of a protein dense food in overweight and obese adults as compared to healthy-weight controls. These data indicate that impaired postprandial MPS may be an early defect with increasing fat mass and may be dependent on altered anabolic signals leading to poor sensitivity to protein ingestion.

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