

the "Nordic Musculoskeletal Questionnaire" to assess musculoskeletal disorders.

**RESULTS:** The data show that there are significant groups of adolescents reporting musculoskeletal disorders in the last 12 months, mainly in the knees (42.4%), dorsal spine (36.2%), lumbar spine (31.7%), cervical spine (36.0%) and shoulders (37.1%). It is also observed that musculoskeletal disorders are more prevalent in adolescents of the female gender, from low socioeconomic classes, with height above 1.60 m and with less physical activity. SMEs were higher in adolescents with overweight/obesity, especially in the lumbar spine (75.0%), hips/thighs (83.3%) and knees (58.3%), with statistically significant differences ( $p < 0.005$ ).

**CONCLUSION:** Our study reinforces data on the increase in musculoskeletal disorders in adolescents, which have a multidimensional origin. It also shows that there are factors such as overweight that are particularly important in the prevalence of SMEs. It is therefore imperative to invest in the prevention of these pathologies through health education interventions that promote an optimized musculoskeletal functioning.

## EP-258 | The effects of obesity superimposed with aging in female mouse model

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**BACKGROUND AND AIM:** People worldwide are living longer and the prevalence of overweight and obesity is growing at an alarming rate. Moreover, obesity has proved to be typically more prevalent among women, who usually live longer than men. Based on these evidences and considering that obesity leads to body health consequences in a way resembling aging, the aim of this study is to evaluate whether obesity superimposed with aging worsens the age-dependent changes at peripheral, systemic and central level in female mice.

**METHODS:** The 4-week-old C57BL/6J female mice were fed with standard diet (SD, 10% of energy from fat) or high fat diet (HFD, 60% of energy from fat) for 8, 20, or 36 weeks. After the exposure to the diet, animals were weighted and fasting metabolic parameters (glucose, triglycerides, cholesterol, insulin, leptin) were measured in blood. The gastrointestinal transit was analyzed by the intestinal distribution of high molecular weight fluorescein isothiocyanate dextran (FITC-dextran 70 kDa). The number of fecal pellets was evaluated during 1-hour collection period, and then the fecal water content was calculated. The integrity of intestinal barrier was assessed functionally by plasma level measurement of low molecular weight FITC-dextran

4 kDa after oral gavage and by evaluation of tight junctions occludin (western blot) and zonulin-1 (ELISA) expression level. To investigate the systemic inflammation, the following serum parameters were measured by ELISA: IL-1 $\beta$ , IL-6, IL-23, IL-10. Levels of A $\beta$ 1-42 amyloid (ELISA), p-Tau, SIRT1, occludin and zonulin-1 (western blot) were evaluated in hippocampus.

**RESULTS:** In female mice, long-term HFD consumption resulted in an obese phenotype and accelerated age-dependent changes in cholesterol, glucose, insulin and leptin serum levels. Obese aged mice showed delayed intestinal transit, decreased gastric emptying, constipation, reduction in fecal water and increased intestinal permeability earlier and in an enhanced extent compared to SD aged mice. Moreover, obesity caused a further release of systemic inflammatory cytokines, previously observed during aging. Finally, HFD exposition had detrimental effects on brain barrier integrity, increased levels of A $\beta$ 1-42 amyloid and decreased SIRT1 expression in hippocampus.

**CONCLUSION:** Our results demonstrated that chronic HFD exposure worsened metabolic alterations, gastrointestinal dysfunctions and systemic inflammation observed in aged SD animals. Moreover, HFD intake caused alterations of brain barrier integrity at early time when compared to old SD mice, possibly accelerating comorbidities at central nervous system. In conclusion, obesity superimposed with aging would accelerate or aggravate the process of aging itself.

**CONFLICT OF INTEREST:** None disclosed.

## EP-259 | Novel automation of bite analysis for universal eating monitor-measured laboratory meals

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**INTRODUCTION:** Eating faster, taking more bites, and larger bite sizes have been associated with increased energy intake. Laboratory measurements of eating behavior on a universal eating monitor (UEM) that generate cumulative intake curves do not enable examination of meal microstructure at the level of individual bites of food. Our methodology yields number and size of bites and when these occur in a meal, as well as bite frequency.

**METHODS:** A manual approach to collect bite outcomes was developed from a pilot study of healthy young adults (8 females, 3 males; mean age 23.0  $\pm$  1.5 years; BMI range 19.3–33.5 kg/m<sup>2</sup>) who ate a mixed macronutrient meal of pasta, tomatoes, and cheese on a UEM. Bites were calculated during periods of UEM scale stability, with a bite as 1–31 g, and other specific criteria. From the manual approach, a flow chart was constructed and used to develop a computer code in MATLAB. The code is being used to analyze UEM data from an eating behavior intervention study that enrolled healthy young females ( $n = 65$ ; mean age 21.0  $\pm$  4.3 years; mean BMI