BOOK OF ABSTRACTS

XXIII European Conference on Food Chemistry

EUROFOODCHEM XXIII

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Neuroprotective effects of mushroom biomass: Impact of serum-available and gut microbiota metabolites in *Caenorhabditis elegans* models of Alzheimer's disease

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Mushrooms are considered a next-generation food with numerous health-promoting properties, including prebiotic and neuroprotective effects. While most research studies focus on the polysaccharide fractions of mushroom-fruiting bodies[1,2], this study investigates the neuroprotective potential of mushroom biomass (MB) from *Trametes versicolor*, *Hericium erinaceus*, and *Pleurotus ostreatus* considering the possible synergistic effects of different macromolecules found both in the mycelium and fruiting bodies. Gastrointestinal digestion was simulated using the standardized INFOGEST protocol, and the passage throughout the duodenum and jejunum was simulated. Results revealed that glucans were the most abundant group in colon and serum-available fractions (26–44% dry weight-DW). The colon-available fraction also contained proteins and peptides (<75 kDa) as well as fatty acids (oleic and linoleic acids). I*n vitro* fecal fermentation assays confirmed the potential of MB to modulate gut microbiota, promoting short-chain fatty acids production and increasing the relative abundance of key bacteria genera (e.g., *Bifidobacterium* and *Faecalibacterium*). In contrast, the serum-available fraction was richer in low molecular weight peptides (<1.2 kDa), amino acids (Tyr, Val, Phe, and Leu), total phenolic compounds (730–863 mg GAE/ 100 g DW), and demonstrated significant antioxidant capacity by different assays (e.g., FRAP: 177–305 mg ISHE/ 100 g DW). The neuroprotective potential of serum-available fractions and key gut microbiota-derived metabolites were evaluated in transgenic *Caenorhabditis elegans* models of Alzheimer's disease by analyzing chemotactic behavior and paralysis onset. Chemosensory responses in strains exhibiting neuronal tau and amyloid toxicity (BR5270 and CL2355) were improved after treatment with serum-available fractions and gut microbiota metabolites, namely, gamma-aminobutyric acid and butyric acid. Additionally, paralysis onset was delayed in the CL4176 strain, exhibiting amyloid toxicity in muscles. These findings suggest that MB confers *in vivo* protective effects against Tau neurotoxicity and amyloid toxicity in neurons and muscles. Future work is required to elucidate the underlying molecular mechanisms.