ISN Symposium: Sphingolipids and brain: pathophysiology and therapeutics

Chairs

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BUDGET (Total Expenses)

		Subtotal
Free Registration and accommodation Fees for 4 Invited Speakers	760 € = 762 USD each	3048 USD
Registration and accommodation Fee discount for 6 selected Young Scientists, presenting oral communications	241 € = 242 USD each	1452 USD

Total expenses **4500 USD**

Summary

Sphingolipids have been first described as one of the major structural constituents of biological membranes, however, their functional role has attracted increased attention over the years. Sphingolipids are crucial in the maintenance of cell homeostasis by regulating several molecular and biochemical pathways in both neuronal and non-neuronal cell populations. As matter of fact, they represent a critical player of human physiology and alteration in their metabolism/pathways is described to be associated with many pathologies including ischemic stroke and neurodegenerative disorders.

During the Symposium we discussed the role of sphingolipids in the regulation of neurochemical pathways and to highlight their potential in the development of novel and effective therapeutic strategies for brain disorders.

ISN Symposium Abstracts

Invited Speakers

Gangliosides: old dogs with new tricks

S. Sipione, J. Monyror, V. Kadam, D. Galleguillos, N. Steinberg, A. Zaidi
Department of Pharmacology and Neuroscience and Mental Health Institute, University of Alberta, Canada

Gangliosides are glycosphingolipids highly enriched in the brain. They play important modulatory functions in cell signaling and cell to cell communication. The importance of gangliosides in the brain is underscored by the fact that defects in the biosynthesis of gangliosides leads to early onset neurodegenerative diseases. Changes in ganglioside levels occur in Parkinson's disease (PD), Huntington's disease (HD) and Alzheimer's disease (AD), but their relevance to disease pathogenesis and progression remains unclear.

Restoration of ganglioside levels in cell and animal models of HD through administration of GM1, a major brain ganglioside, slows down neurodegeneration and corrects all disease symptoms. In search for the underlying mechanisms, our studies have uncovered two novel roles for gangliosides: 1) GM1 and most complex gangliosides, but not GM3 or GD3, increase cell secretion of extracellular vesicles (EVs), including EVs carrying disease-associated misfolded proteins, thereby reducing neuronal proteotoxic stress; 2) Gangliosides act as modulators of the response of microglia to inflammatory stimuli, with some gangliosides, including GM1, dampening, and others promoting inflammatory responses by microglia.

While distinct and independent from each other, the roles of gangliosides in EV secretion and microglia modulation depend on the presence of sialic acid in the glycan headgroup and the lipid tail of the ganglioside molecule.

The activity of GM1 and other gangliosides as promoters of EV and misfolded protein secretion and inhibitors of microglia-mediated neuroinflammation might contribute to the neuroprotective roles of these molecules in neurodegenerative and neuroinflammatory diseases, and could potentially be exploited for the treatment of misfolded protein disorders such as HD, PD and AD.

Role of the lysosomal impairment in the onset of neuronal degeneration

E.V. Carsana[1], M. Samarani[2], G. Lunghi[1], Stefano Duga[3,4], Letizia Straniero[3,4], Rosanna Asselta[3,4], Giulia Soldà[3,4], E. Frattini[5], N. Loberto[1], A. Di Fonzo[5], **M. Aureli[1]**
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Lysosomal homeostasis is fundamental for cell viability. Alterations in the endolysosomal function together with the accumulation of uncatabolized molecules are common features of so called lysosomal storage disorders (LSD). These pathologies are severe inherited metabolic disorders caused by loss of function mutations in specific lysosomal hydrolases or lysosomal- related proteins with the consequent accumulation of their uncatabolized substrates.

Elucidating the molecular mechanism connecting lysosomal impairment with the onset of cell damage in LSD remains one of the most important challenges in the field.

To address this issue, we generated an *in vitro* model of lysosomal impairment represented by human fibroblasts subjected to sucrose loading. Interestingly, we described the existence of a lysosome- plasma membrane axis responsible for the onset of cell damage upon the aberrant lysosomal accumulation of uncatabolized material. In addition, recent data elucidated the role of β-glucocerebrosidase deficiency, associated with the LSD Gaucher disease and GBA-related Parkinsons' disease, in the onset of cell damage. We developed two *in-vitro* models of the neuronal form of Gaucher disease represented by primary murine granule cells and human iPSCs-derived dopaminergic neurons treated with conduritol B epoxide (CBE) to suppress β-glucocerebrosidase activity. CBE-treated neurons showed the accumulation of glucosylceramide and the onset of neurodegeneration. Interestingly, using these experimental models, we observed that glucosylceramide accumulation is not confined to the lysosomes but occurs also at plasma membrane level and in the vesicles released in the extracellular milieu.

In conclusion, our data together with some other evidence reported in literature highlight the ability of impaired lysosomes to modify the plasma membrane composition, contributing, at least in part, to the onset of cell damage.

Regulation of blood-brain barrier properties by Sphingosine-1 phosphate signaling

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The blood-brain barrier (BBB) is a highly selective endothelial barrier that prevents blood born solutes, cells and pathogens from crossing into central nervous system space.
A dysfunctional BBB is a common complication of a plethora of diseases of the central nervous system, like Alzheimer's disease or epilepsy, as well as ischemic or traumatic brain injuries.
However, a functional BBB also prevents the crossing of therapeutic agents. Regulation of barrier development and tightness remains poorly understood.

We have found a signaling pathway crosstalk, where Wnt signaling interferes with S1P signaling during brain angiogenesis and S1P signaling consecutively regulates BBB tightness (Hübner et al Nat Commun. 2018).

We are using zebrafish as model system to analyze brain angiogenesis and BBB tightness in vivo and human cerebral microvascular endothelial cell (hCMEC/d3) cultures for whole proteome and phospho-proteomic analyses on vitro. Interestingly we found very time sensitive differences in the immediate and prolonged response to S1P stimulation.

We are addressing the molecular nature of the Wnt-S1P crosstalk as well as which downstream effectors of S1P-signaling regulate BBB tightness at different time points.

Tight control of S1P signaling at the Blood-Brain-Barrier

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Cerebrovascular function is critical for brain health, and endogenous vascular-protective pathways may provide therapeutic targets for neurological disorders. Sphingosine 1-phospate (S1P) signalling sustains vascular functions in other organs, and S1P receptor-1 (S1P1) modulators show promise for the treatment of ischemic and hemorrhagic stroke. We have addressed expression, roles and mechanisms of engagement of

endothelial cell S1P1 in the naïve and ischemic cerebral cortex and its potential as a therapeutic target for ischemic stroke.

S1P1 was widely expressed in the vascular endothelium throughout the cerebral cortex. Yet S1P1 engagement - revealed by analysis of S1P1-ß-arrestin coupling in S1P1 reporter mice - was limited to a subset of arterial endothelial cells. During cerebral ischemia, S1P1 signalling expanded to capillary and venous endothelial cells by recruitment of endothelial cell-autonomous S1P production. Selective deficiency of S1P1 expression or S1P production in endothelial cells strongly increased brain injury and vascular leak after middle cerebral artery occlusion in mice. By contrast, lack of plasma S1P, essential for S1P1 signaling in other vascular beds, did not impact outcome. Accordingly, analysis of receptor expression and side-selective stimulation of S1P1 in signaling reporter mice revealed that S1P1 is sorted to the abluminal surface of the endothelium after maturation of the blood-brain barrier (BBB), and thus shielded from circulating ligands. A BBB-penetrating S1P1 agonist was neuroprotective after middle cerebral artery occlusion, while no protection was observed with an agonist that induced equivalent systemic activation of S1P1 signaling but did not cross the BBB.

Our observations suggest a key protective role of S1P1 signaling in the cerebral vasculature and the need for BBB penetration for pharmacological engagement of S1P1 in the brain endothelium.

Lipids in neurodegeneration: from biomarkers to targets for intervention

Pilar Martinez-Martinez

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Sphingolipids, a very enigmatic class of lipids, play a significant role in health and disease. In spite of the improvement in the methodology to measure lipids, to date, the biological relevance of individual sphingolipid species and their dysregulation and relationship with pathophysiology are largely unknown. I will summarise the current state of knowledge of this type of lipids and their effect in neuro-inflammation and neurodegeneration in particular during Alzheimer's disease. It is well-known that changes in the homeostasis of the sphingolipid metabolism contributes to neurodegeneration and that the modulation of the concentration of specific sphingolipid species favours immune responses and neuro-inflammatory events through brain resident macrophages and astrocytes, affecting the neurodegenerative process. Moreover, since in the last years, several diagnostic and therapeutic approaches targeting this pathway are gaining ground in particular for the resolution of AD, I will highlight sphingolipid-targeting molecules as potential biomarkers and drugs able to modulate sphingolipid levels and show beneficial effects for AD.

Selected Young Scientists

Sphingomyelin 16:0 is a therapeutic target for brain pathology in acid sphingomyelinase deficiency

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Acid sphingomyelinase deficiency (ASMD) is a lysosomal storage disorder caused by mutations in the *SMPD1* gene encoding for the acid sphingomyelinase (ASM). While intravenous infusion of recombinant ASM is an effective treatment for the peripheral disease, the neurological complications of ASMD remain unaddressed. It has been shown that aberrantly high level of total brain sphingomyelin (SM) is a key pathological event leading to neurodegeneration. Using mice lacking ASM (ASMko), which mimic the disease, we here demonstrate that among the SM species, SM16:0 shows the highest accumulation and toxicity in ASMko neurons. By targeting lysosomes, SM16:0 causes permeabilization and exocytosis of these organelles and induces oxidative stress and cell death. We also show that genetic silencing of Ceramide Synthase 5, which is involved in SM16:0 synthesis and overexpressed in the ASMko brain, prevents disease phenotypes in ASMko cultured neurons and mice. The levels of SM16:0 in plasma also show a strong correlation with those in brain that is higher than in liver, even at early stages of the disease. These results identify SM16:0 both as a novel therapeutic target and potential biomarker of brain pathology in ASMD.

Ceramide system contributes to learning and memory

Liubov S. Kalinichenko[1], Laila Abdel-Hafiz[2], An-Li Wang[2], Christiane Mühle[1], Nadine Rösel[1], Fabian Schumacher[3,4], Burkhard Kleuser[3], Irena Smaga[5], Malgorzata Frankowska[5], Malgorzata Filip[5], Gerd Schaller[1], Tanja Richter-Schmidinger[1], Bernd Lenz[1,6], Erich Gulbins[4,7], Johannes Kornhuber[1], André W. C. Oliveira[8], Marilia Barros[8,9], Joseph P. Huston[2], Christian P. Müller[1,10]
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Learning and memory are crucial brain mechanisms mediating the successful adaptation of individuals to constantly changing environmental conditions (Hartley et al., 2021). Recent studies propose that memory is not purely a protein-mediated process, and a principally new, lipid-based mechanism might regulate cognition under normal and pathological conditions (Kalinichenko et al., 2022).

In our study, the specific contribution of one of the enzymes of ceramide metabolism, neutral sphingomyelinase (NSM), in memory processes was observed. In naïve male Wistar rats, high NSM activity

in the ventral striatum (VS) and dorsal hippocampus (DH) was predictive of superior performance in appetitively motivated short-term and long-term memory measured in the spontaneous alternation (SAT) and novel object recognition (NOR) tests, respectively. The observed high NSM activity was associated with the high levels of SM18:0 and Cer18:0, but not other species in the VS and DH. Similar, in male primates Callinix penicillata higher serum NSM activity was associated with better memory performance in an object discrimination test. The interaction between NSM activity and appetitive memory in mammals was confirmed by a study in transgenic mice with NSM hypoexpression (Fro mice). Female, but not male Fro mice were characterized by worse appetitively motivated memory measured in the SAT and NOR tests. This impairment might be determined by the sex-specific decrease in the expression of glutamatergic receptors GluN2B in the dorsal striatum.

Clinical data are in line with the results obtained on mammals. Superior appetitively-motivated long-term logical memory in the Rivermead Behavioral Memory Test was associated with enhanced serum NSM activity in healthy human male volunteers. Altogether, these data suggest a new sphingolipid mechanism of learning and memory performance mediated by NSM, which is affective valence-dependent and working in a brain region-selective way in rodents. NSM activity in the blood may also bear some predictive power for the assessment of learning capacity in humans. The study was supported by the German National Science Foundation (grant MU 2789/8-2, GU 335/29-2, KO 947/15- 2, HU 306/27-3).

FTY720 decreases ceramide levels in the brain and prevents memory impairments in a mouse model of familial Alzheimer's disease expressing APOE4

Daan van Kruining [1], Simone M Crivelli [2], Qian Luo [1], Caterina Giovagnoni [1], Marina Mané-Damas [1], Sandra den Hoedt [3], Dusan Berkes [4], Helga E De Vries [5], Monique T Mulder [3], Jochen Walter [6], Etienne Waelkens [7], Rita Derua [7], Johannes V Swinnen [8], Jonas Dehairs [8], Erwin P M Wijnands [9], Erhard Bieberich [10], Mario Losen [1], Pilar Martinez-Martinez [1]

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The protection mediated by the bioactive sphingolipid sphingosine-1-phosphate (S1P) declines during Alzheimer's disease (AD) progression, especially in patients carrying the apolipoprotein E ε4 (APOE4) isoform. The drug FTY720 mimics S1P bioactivity, but its efficacy in treating AD is unclear.

Two doses of FTY720 (0.1 mg / kg and 0.5 mg / kg daily) were given by oral gavage for 15 weeks to transgenic mouse models of familial AD carrying human apolipoprotein E (APOE) APOE3 (E3FAD) or APOE4 (E4FAD). After 12 weeks of treatment, animals were subjected to behavioral tests for memory,

locomotion, and anxiety. Blood was withdrawn at different time points and brains were collected for sphingolipids analysis by mass spectrometry, gene expression by RT-PCR and Aβ quantification by ELISA.

We discovered that low levels of S1P in the plasma is associated with a higher probability of failing the memory test and that FTY720 prevents memory impairments in E4FAD. The beneficial effect of FTY720 was induced by a shift of the sphingolipid metabolism in the brain towards a lower production of toxic metabolites, like ceramide d18:1/16:0 and d18:1/22:0, and reduction of amyloid-β burden and inflammation. We provide further evidence of the druggability of the sphingolipid system in AD.

Sphingolipid-dependent membrane organization and signaling orchestrating myelin repair

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Department of Medical Biotechnology and Translational Medicine, Università degli Studi di Milano, Milano, Italy

Recombinant human IgM22 (rHIgM22) binds to myelin and oligodendrocytes (OLs) and promotes remyelination in mouse models of multiple sclerosis. However, target antigen and signaling mechanisms through which rHIgM22 exerts its function are still unclear.

In vitro analysis revealed that rIHgM22 binds to sulfatide, phosphatidylinositol and phosphatidylserine. Moreover, the composition of the lipid microenvironment of its antigen can modulate the affinity of the antibody, suggesting reorganization of lipid membrane might be relevant in its biological activity.

In rat mixed glial cells (MGC), rHIgM22 induces an increase in PDGFαR levels and a dose-dependent proliferative response in all cells in the culture, with the most significant response associated with astrocytes. Moreover, rHIgM22 increases production and release of sphingosine 1-phosphate (S1P) in MGC while total levels of ceramide remain unchanged. Furthermore, release of S1P is strongly reduced by a selective inhibitor of PDGFαR. Remarkably, rHIgM22 treatment does not induce changes in the production and/or release of S1P in astrocytes, but it increases its release in BV2 cells, suggesting that rHIgM22 indirectly influences the proliferation of astrocytes in MGCs, by affecting ceramide/S1P balance.

Analysis of the effect of rHIgM22 on glycosphingolipid metabolism in MGC and astrocytes revealed no significant effects on the lipid pattern, while in OPCs and OLs the levels of gangliosides GM3 and GD3, known for their ability to interact with and modulate the activity of growth factor receptors, are increased.

Considering all this, we propose rHIgM22 protective effects might be mediated by alterations of lipid-dependent membrane organization and/or signalling in different glial cells and that a complex cross talk between these cells is underlying the repair effect elicited by this antibody.

Treatment with THI, an inhibitor of Sphingosine-1-Phosphate Lyase (SGPL1), modulates glycosphingolipid metabolism and results therapeutically effective in a mouse model of Huntington's disease

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Over the past few years our research has shown that alterations in sphingolipid metabolism represent a critical determinant in Huntington's disease (HD) pathogenesis. In particular, aberrant metabolism of gangliosides and sphingosine-1-phosphate (S1P) has been reported in multiple disease settings including human post-mortem brains from HD patients.

In this study, we investigated the potential therapeutic effect of the inhibition of S1P degradative enzyme SGPL1, by the chronic administration of THI inhibitor.

In vivo experiments were carried out in both R6/2 mice and WT littermates, starting from 6 weeks of age. THI was dissolved in DMSO, further diluted in saline (vehicle) and daily administered by intraperitoneal (i.p.) injection at dose of 0.1mg/kg of body weight. Control mice (WT and R6/2) were daily injected with the same volume of vehicle containing DMSO. The potential beneficial effect of treatment on motor performance was assessed by Horizontal Ladder Task and Rotarod tests. Sphingolipidomics was performed by LC/MS-MS.

Our findings showed that THI mitigated motor dysfunctions in HD mice. The compound evoked the activation of pro-survival pathways, normalized levels of BDNF and preserved white matter integrity in HD mice. Under metabolic point of view, THI restored normal levels of hexosylceramides (glucosylceramide) and stimulated the autophagic and lysosomal machinery, facilitating the reduction of nuclear inclusions of both wild type and mutant huntingtin proteins.

This study further highlights the potential role of (glyco)sphingolipid pathways in HD pathogenesis, and their pharmacological targeting for the development of new therapies.

S1P-lyase deficiency in the brain increases glucose breakdown evoking a P2Y1 receptor-dependent astrogliosis

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Sphingosine-1-phosphate (S1P) is an evolutionarily conserved catabolic intermediate of sphingolipid metabolism that was shown to be essential for brain development. However, its role in neurodegenerative processes is subject of debate. In order to clarify the function of S1P in the brain, we created a mouse model in which S1P-lyase (SGPL1), the enzyme which irreversibly cleaves S1P in the final step of sphingolipid catabolism, was inactivated specifically in neural cells. As a result, S1P accumulated in the brain which led to several changes such as disruption of presynaptic architecture, cognitive deficits, and tau hyperphosphorylation. In addition, ablation of SGPL1 impaired neuronal autophagy and resulted in the accumulation of aggregate-prone proteins such as amyloid precursor protein and alpha-synuclein. In the current study, we show that S1P-lyase deficiency triggers astrogliosis in both SGPL1-deficient brain tissue and in primary cultured astrocytes.

Additionally, we found that $S1PR_{2,4}$ was activated in SGPL1-deficient astrocytes, leading to an increase in glucose catabolism and ATP production, which is associated with P2Y1 receptor activation. Further consequences of the observed changes due to P2Y1 receptor activation will be shown and discussed. In conclusion, SGPL1 deficiency in the brain alters glucose metabolism via $S1PR_{2,4}$ that ultimately results in P2Y1R-dependent astrogliosis.


we are sphingolipids
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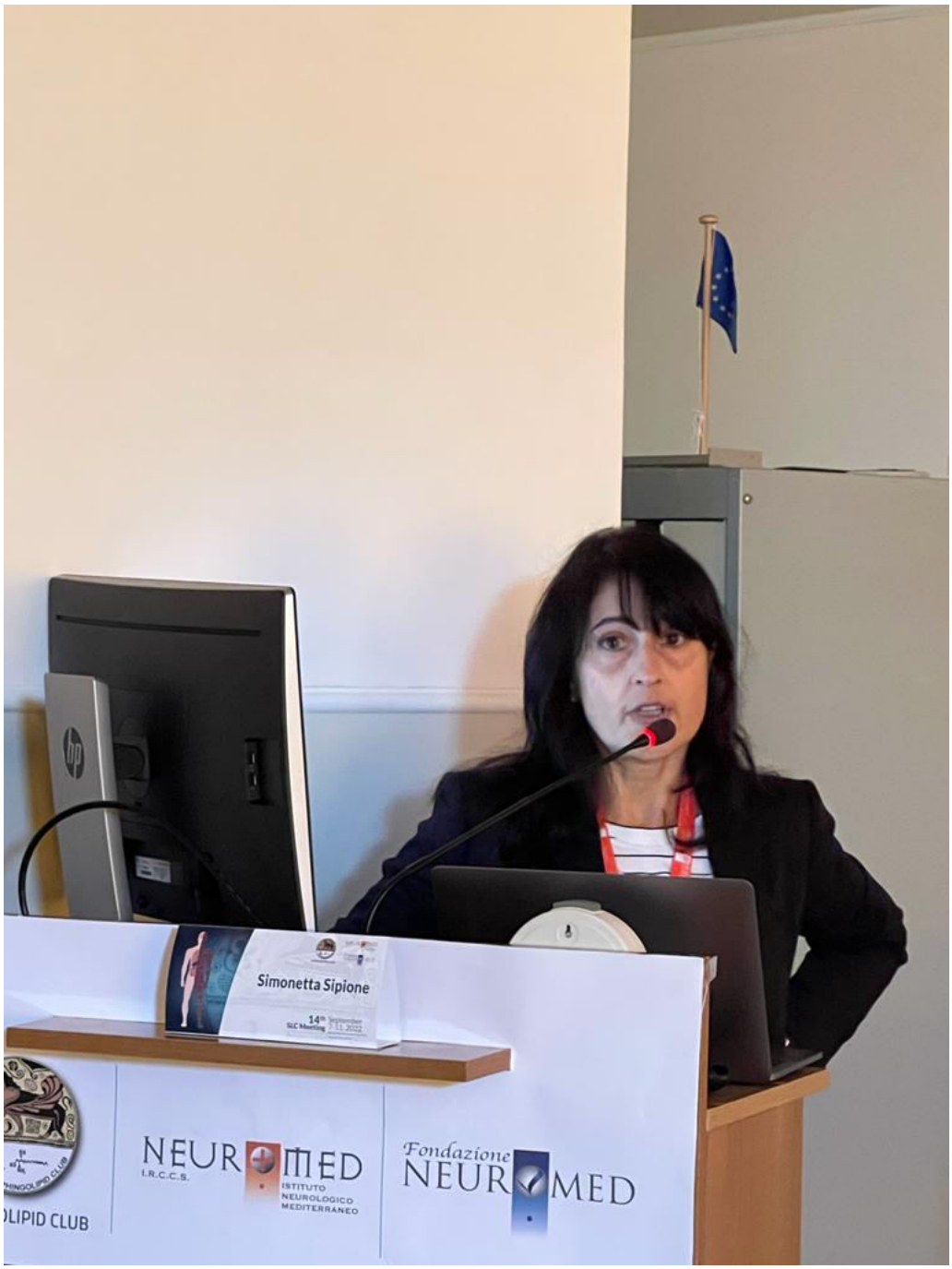
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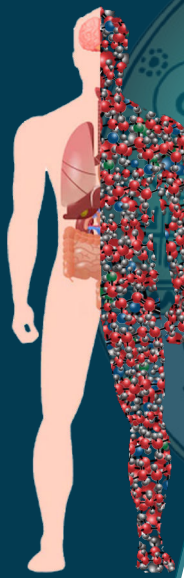

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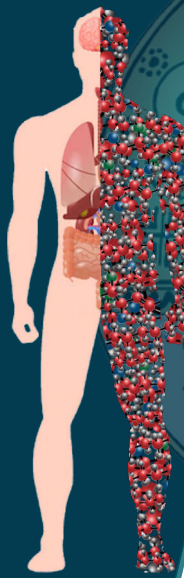

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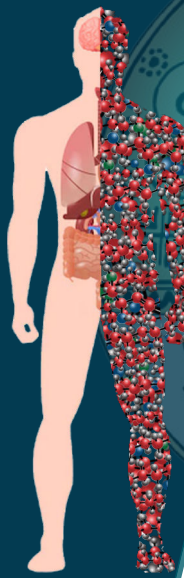

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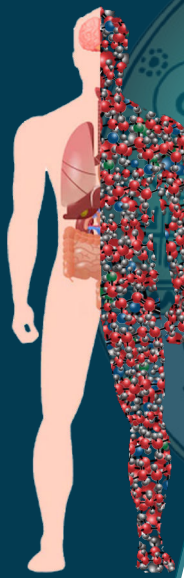

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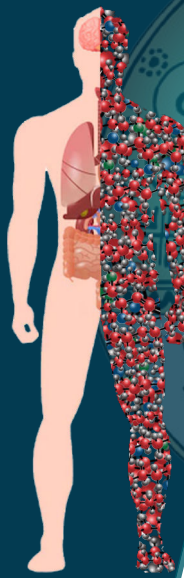

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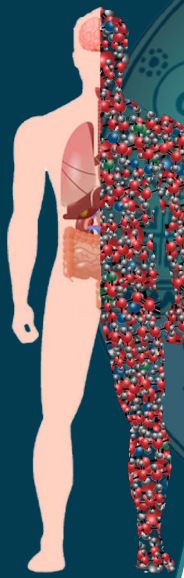

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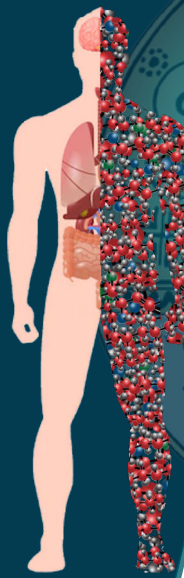

Pilar Martinez-Martinez

University of Maastricht, NL

14th SLC Meeting

September 7-11, 2022

Neuromed Research Centre, Pozzilli (Is), ITALY

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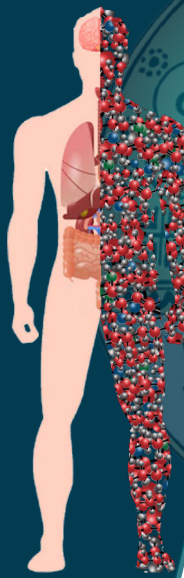

Simonetta Sipione

University of Alberta, CA

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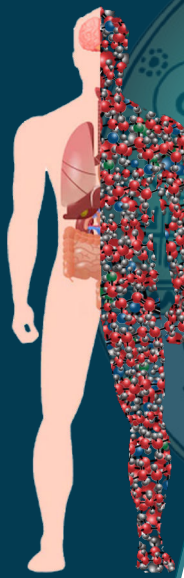

Giuseppe Pepe

IRCCS Neuromed, IT

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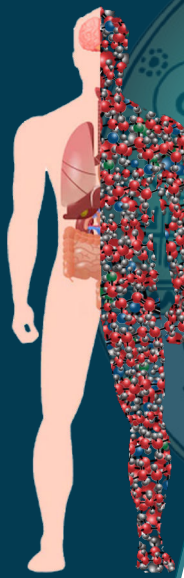

Wiebke Herzog

University of Erlangen, DE

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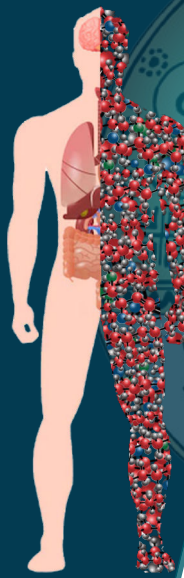

Daan van Kruining

Maastricht University, NL

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