A5). A4 was placed in a familiar location and A5 was placed in a new location between A2 and A3 previous location.
2. Spontaneous object recognition task (SOR): For this task, we follow the same experimental scheme of SLR performed in a triangular open field (60 cm long). After habituation, on the sample phase, three different objects were assembled in three combinations (AB, BC, and DE). On the choice phase, the novel object was assembled in AC (two non-shared features in the sample), and the familiar object was a copy of DE.

The results were expressed as an exploration time discrimination ratio [(novel–familiar)/total]. In the SLR, memory showed deterioration in overlapping memory discrimination in both aged groups as compared with Young animals. In the SOR, only Old rats presented a deterioration in memory, however, Middle-aged rats showed a preserved memory as compared with Young rats.

We conclude that the overlapping memories involving object location are more sensitive to age (start to decline at Middle-aged) than overlapping memories involving characteristics.

https://doi.org/10.1016/j.ibror.2019.07.1016

P20.63

Role of environmental enrichment in locomotion, anxiety, memory and social interaction of Wistar rats under chronic treatment of methylphenidate

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Chronic use of methylphenidate may have disruptive effects on the behavior of animals. These effects can be reverted by environmental modification. It has been reported that environmental enrichment can revert behavioral changes caused by the chronic consumption of this psychostimulant. The present study aims to investigate how environmental enrichment can revert the effects of chronic consumption of methylphenidate on social, emotional and exploratory behavior of Wistar rats. Animals were exposed to a 21 days physical environmental enrichment protocol which included tubes of different shapes and 14 days of drug consumption by oral administration. After 7 days of drug withdrawal they underwent behavioral tests: elevated plus maze, open field, object in place recognition and social interaction in which anxiety, locomotion, spatial memory and social behavior were measured. Our results showed that methylphenidate can reduce anxiety ($F[1,43] = 4.27$; $P = 0.009$) and some social behaviors such as following ($F[1,43] = 15.41$; $P = 0.002$) and single exploration ($F[1,43] = 5.34$ $P = 0.026$) and increase locomotion ($F[1,43] = 4.77$; $P = 0.034$). However these effects are mitigated by environmental enrichment.

https://doi.org/10.1016/j.ibror.2019.07.1017

P20.64

Dopamine-dependent modulation of memory formation in the hippocampus

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Special event is known to determine the efficacy of memory encoding, and this effect depends on enhanced dopaminergic input to the hippocampus. Loss of dopamine in Parkinson's disease has been associated with cognitive symptoms, in addition to the motor symptoms. It is unknown, however, how this dopaminergic input modulates the neuronal ensembles that encode the new memory in the hippocampus. Interestingly, dopamine receptor subtypes are differentially expressed in the specific neuronal subtypes consisting of the hippocampal circuit. Noteworthy, Drd1 and Drd2 are expressed exclusively at a subpopulation of granule cells (GCs) and most of mossy cells (MCs) in the dentate gyrus, respectively. Here our research aims to address the following questions: (1) Whether dopaminergic input modulates the neuronal ensembles of Drd1-positive GCs and Drd2-positive MCs upon exposure to novelty context. (2) Whether Drd1-positive GCs and Drd2-positive MCs cooperate to enhance the efficacy of memory encoding and then how. By taking advantage of in vivo calcium imaging approach in freely moving animals, we will visualize the neuronal activity ensembles of these dopamine receptor-positive neurons in the hippocampus while we monitor the animal behavior in response to the novelty context. By combining molecular biological, neural activity imaging, pharmacological, and behavioral approaches, we will be able to test my hypothetical model that novelty context exposure may regulate two distinct dopaminoceptive neurons, Drd1 + GCs and Drd2 + MCs and then those two neurons may crosstalk during memory encoding in the hippocampus. Any novel findings from our research may contribute to better understanding of the neuromodulatory mechanism by which novelty could influence memory formation.

https://doi.org/10.1016/j.ibror.2019.07.1018

P20.65

Investigating the effects of air pollutant nanoparticles on the onset or progression of Alzheimer's disease

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Sporadic Alzheimer's disease (AD) occurs in 99% of cases with air pollutants recognised as a major risk factor. Despite this, there has been no established link between air pollutant nanoparticles and the development of AD. The aim of the present study was to determine whether inhalation of externally derived air pollutant nanoparticles will exacerbate AD pathogenesis in a mouse model of the disease.

We derived air pollutant nanoparticles from (i) diesel emission particles (DE), (ii) micro abraded hot rolled train track iron and iron oxide particles (IRON) and (iii) magnetite nanoparticles (MNPs). Four study groups, each including 3 month-old C57BL/6 ($n = 36$) and APP/PS1 ($n = 35$) mice, received either of the aforemen-

tioned nanoparticles or saline (vehicle control), every third day, via intranasal administration [66 μg] over a 17-week experimental timeframe. The elevated plus maze test, performed at week 14 to assess anxiety, showed a significant increase in stress anxiety levels in (i) C57BL/6 exposed to IRON and DE compared to C57BL/6 wild type (WT) controls, and (ii) APP/PS1 exposed to IRON compared to APP/PS1 controls. *In-vivo* fluorescent imaging of the brain using amyloid beta (Aβ)-specific probes (CRANAD 58 and (2) showed an increase in Aβ load in (i) C57BL6 exposed to DE and MNPs, and (ii) APP/PS1 exposed to IRON, DE and MNPs compared to controls. At the cellular level, cultured human neuroblastoma cells (SH-SY5Y) and mouse microglia cells (BV2) exposed to the air pollutant nanoparticles showed (i) an increase in reactive oxygen species production; (ii) decrease in cell viability and (iii) increased expression of neuroinflammatory markers (TNF, IL-6, IL-1β and IL-1α) in both cellular models, compared to the vehicle control.

The results of this study establishes that nanoparticles derived from air pollution results in an increase in cerebral Aβ levels. Our findings suggest that air pollutants induce neurological changes via pathways associated with inflammation and oxidative stress in key cells that play a significant role in AD on-set.

https://doi.org/10.1016/j.ibror.2019.07.1019

P20.66

Effects of repeated trauma on functional brain network in mediating posttraumatic stress symptoms of firefighters

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Firefighters often experience a wide range of stress-related symptoms in response to repeated traumatic exposure including post-traumatic syndrome. Although accumulating evidence suggests a close link between repeated trauma exposure and risks for stress-related disorders in firefighters, little is known about the neural correlated reflecting this association in firefighters.

The aim of the present study is to investigate the brain correlates underlying repeated trauma exposure as well as its mediating roles in PTS symptoms. The study participants included 52 firefighters with repeated trauma exposures and 46 healthy individuals without a history of trauma. The PTS symptom severity were evaluated and alterations in brain functional network connectivity were investigated using resting-state functional magnetic resonance imaging.

A weaker connectivity strength between the salience network (SAN) and default mode network (DMN) as well as between the SAN and primary sensory network (PSN) were found in the firefighters relative to the control group. In addition, more severe PTS symptoms were associated with weaker connectivity strengths of the SAN-DMN and the SAN-DMN-central executive network (CEN) in the firefighters.

These results suggest that a dysfunctional triple network system, such as weaker connectivity strengths of the SAN-DMN and SAN-CEN-DMN, may play a mediating role in the association between repeated trauma exposure and PTS symptom severity in firefighters. Repeated trauma exposure may increase vulnerability to PTS symptoms through alterations in the large-scale brain functional networks.

https://doi.org/10.1016/j.ibror.2019.07.1020

P20.67

Mao-b-dependent gaba in the hippocampal reactive astrocytic induces cognitive impairment in animal model of rheumatoid arthritis

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Rheumatoid arthritis (RA) is an autoimmune disease, characterized by joint swelling and chronic systemic inflammation. It is known that systemic inflammation of RA affects liver, bone, vessel system and others. Interestingly, RA is commonly accompanied with mental disorders such as cognitive impairment and depression. Thus we expect that inflammation can also cause brain dysfunction, leading to cognitive impairment in RA. However, the exact mechanism of cognitive impairment in RA is unknown. Here, we show that cognitive impairment in animal model of RA is depended on the astrocyte GABA producing enzyme, monoamine oxidase-B (MAO-B). We used the most commonly used animal model of RA, collagen-induced arthritis (CIA) mouse. We found that CIA mouse revealed cognitive impairment in the novel object recognition and object place recognition behavior test which are highly related with hippocampus function. We discovered the hippocampal astrocytic GABA and tonic GABA current were increased in CIA mouse model brain. Also, we found an increase of hippocampal MAO-B mRNA level in CIA mouse hippocampus. Thus, we used newly developed reversible MAO-B inhibitor, KDS2010, to inhibit the astrocytic GABA production. KDS2010 decreased hippocampal astrocytic GABA of CIA and cognitive impairment were fully rescued by acute or chronic application of KDS2010. Incubation of brain tissue with several cytokines including IL-1β, mimicking brain inflammation, showed a similar tonic GABA current. Lastly, we found that KDS2010 also alleviated the rheumatoid arthritis in CIA mouse model. Together, we propose that astrocytic MAO-B is critical for cognitive impairment and selective inhibition of astrocytic MAO-B may serve as a therapeutic target for cognitive impairment in RA.

https://doi.org/10.1016/j.ibror.2019.07.1021

P20.68

Sociocognitive motives mediating human social knowledge sharing behavior, gossip

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Sharing gossip is an effective strategy to gather the most updated knowledge about others. Because of the bad reputation of gossip as malicious behavior, deciding whether or not to disseminate private information of absent third parties is not a simple task; it is rather a sophisticated process that requires careful consideration of various social aspects and possible consequences. However, the factors that influence gossip behavior are not yet clearly known. In this study, we examined the conditions in which various types of