BOOK OF ABSTRACTS

NATIONAL SCIENTIFIC CONFERENCE

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ORAL PRESENTATIONS

PRO-ACTIVE REGULATION OF PAIN PROCESSING IN CONTEMPLATIVE BRAIN STATES

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Background: A variety of studies have demonstrated that meditation reduces subjective pain experience. To understand these effects, neurophysiological models have been proposed. One model posits that the major effects of meditation on pain rely on *reactive cognitive control*, whereby noxious stimuli are sensed as really painful, but the unpleasantness/affect (i.e., the emotional/cognitive appraisal) does not occur. According to another model, *pro-active cognitive mechanisms* are crucial for pain processing in meditation. It is suggested that effortful cognitive processes mediated by executive attention downregulate pain signals and transform ascending nociceptive information from painful to innocuous (sensory).
Objectives: The objective of the present research was to explore pro-active preparatory attention to pain stimulus in different meditation states and different expert groups. Pro-active top-down influences have been correlated with ongoing oscillatory activity from alpha and beta frequency ranges. Specifically, attention orientation to upcoming painful stimuli is associated with a pronounced alpha desynchronization at the contra-lateral primary and secondary somatosensory/insular cortices S1 and S2-IC. Therefore, alpha and beta activities preceding pain stimulus delivery were analyzed in these regions.
Methods: EEG was recorded at 64 electrodes in response to electric left median nerve stimulation in short-term and long-term meditators (STM, LTM) during rest and three types of meditation engaging attentional and affective regulation in different ways: focused attention meditation (FAM), open monitoring meditation (OMM) and loving kindness meditation (LKM). In each condition, multi-spectral pain-related oscillations (PROs) were analyzed after the stimulus. The total power of alpha and beta oscillatory activity was analyzed for a period of 450 ms before pain stimulus delivery to reflect pro-active modulation of cortical excitability. A control registration of spontaneous EEG in no-pain condition was also conducted.
Results: In STM, no lateralized modulations of pre-stimulus alpha or beta power were detected in the resting state. In contrast, only in LTM was the pre-stimulus alpha activity significantly increased at S1 and S2-IC contra-lateral to pain stimulation, such that this increase predicted a suppressed synchronization of multispectral PROs during rest. Moreover, LTM manifested a further pre-stimulus alpha increase at S1 and S2-IC in all meditation states, mostly in FAM.
Conclusion: The contra-lateral augmentation of pre-stimulus alpha in LTM before pain stimulation may reflect an active involvement of inhibitory mechanisms to suppress painful input. LTM appears to be able to deliberately induce a state of inhibition by actively amplifying the alpha rhythms at task-relevant (but not irrelevant, as traditionally found) cortical regions where the painful afferent input is arriving. These results reveal a unique capacity of long-term meditation practitioners to pro-actively control their alpha rhythms. Thus, new evidence is provided for the presence of pro-active top-down inhibition mediated by controlled guidance of alpha oscillations in meditation.
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TRAIT AND STATE EFFECTS OF CONTEMPLATION ON NEUROPHYSIOLOGIC MECHANISMS OF PAIN: EVIDENCE FROM PAIN-RELATED POTENTIALS AND OSCILLATIONS

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Background: Contemplative brain states represented by meditation have been consistently associated with a decrease in perceived subjective pain. Pain experience is conceptualized as an integrative process combining bottom-up (sensory) and top-down (emotion/attention) influences. The positive impact of meditation on noxious event perception has been associated mainly with the modulation of the emotional and attention-related components of pain. However, how exactly mediation affects pain mechanisms is not precisely understood.
Objectives: To analyze pain-related potentials (PRPs) in time- and time-frequency domains in order to explore the trait- and state-effects of meditation on the neural mechanisms of pain processing.
Methods: EEG responses to noxious electric stimulation of the median nerve were recorded at 64 electrodes from long-term meditators (LTM) and novice short-term meditators (STM) during a non-meditative resting state (REST) and three types of meditation: focused attention meditation (FAM), open monitoring meditation (OMM) and loving kindness meditation (LKM). To characterize bottom-up somatosensory processes, the local temporal and spatial synchronization of multi-spectral pain-related oscillations (PROs) was assessed at primary somatosensory (S1) and secondary somatosensory/insular (S2-IC) regions. Because the parietal P3b PRP component is a reliable index of involuntary attentional shifts and emotional processing of nociceptive events, the P3b was analyzed to reflect the cognitive/affective appraisal of pain information. Subjective pain scores (intensity, aversion, identification) also were registered.
Results: In all conditions, PROs in both STM and LTM were characterized by phase-locked delta (1-3 Hz), theta-alpha (5-13 Hz), beta (15-25 Hz), gamma-1 (25-35 Hz) and gamma-2 (35-48 Hz) time-frequency components within 200 ms after nociceptive stimulation. During REST, STM and LTM had similar subjective scores of pain perception. Yet, the temporal and spatial synchronizations of multispectral pain oscillations were substantially suppressed at S1 and S2-IC in LTM relative to STM, while the reduction of P3b in LTM relative to STM was not significant. Phase-locked PROs were not affected by meditation type in any of the groups. However, only STM manifested a pronounced decrease of P3b in LKM relative to REST.
Conclusions: These novel neurophysiological observations challenge current understating by revealing that experienced meditators exhibit (1) suppressed bottom-up pain processes in both non-meditative and meditative brain states, and (2) preserved emotional/cognitive appraisal of noxious input. The results demonstrate major trait effects reflecting neuroplasticity of pain processing networks as a result of long-standing practice and show that tonic fluctuations induced by different mediation states are less efficient to alter pain processes at the neurophysiologic level.
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HEISENBERG UNCERTAINTY PRINCIPLE FOR MENTAL STATES

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It is generally assumed that the variability of the response times scales linearly with their magnitude (analogously to Weber's law for perception), namely, slower response times have larger variability within the same condition. There are, however, systematic violations of this law in certain circumstances. One such case is the priming of the stimulus, which leads to shorter mean response times but, paradoxically, to larger standard deviations of the response times as well. This effect is obtained in a variety of paradigms, e.g., lexical decision and numerosity estimation, with both explicit and subliminal priming manipulations and working within and across trials (inter-trial priming). The effect is most pronounced for the strongest attended stimuli, where a dissociation is observed between the mean of the response times within a condition, which is reduced, and the standard deviation, which is inflated, compared to the non-primed stimuli. It is weaker for the less attended stimuli and absent in control conditions, such as non-words. Computational drift-diffusion models can reproduce this effect by a change in the variability in the initial condition, whereby reduced variability results in shorter mean response time and also larger variability. An explanation can also be constructed by assuming Bayesian updating of the information by the cognitive agent, which produces anti-Weber type of behavior in terms of the variability of the response times. However, a structurally simple explanation also exists under the assumption of quantum or quantum-like information processing in the brain, whereby the observed dissociation is analogous to the Heisenberg uncertainty principle applied to the position and momentum parameters of the quantum system. Within that analogy, the effect of priming is to reduce the uncertainty in the position parameter of the quantum behaving system (i.e., less variable position of the mental state in abstract parameter space), which in turn results in more variable momentum parameters, according to the uncertainty principle. This increased variability is reflected in the variability of the response times, under the assumption that a longer response time reflects a longer time for a perturbed quantum system to settle back to the ground state, which in turn reflects a larger momentum associated with the state. Thus, the observed dissociation effect is directly analogous to the Heisenberg uncertainty principle for quantum systems, assuming that the priming manipulation affects the position of the mental state and the response time is a proxy, indirect measure of the momentum of the mental state as a physical state in the brain. This constitutes weak, indirect evidence for quantum or quantum-like physical processes occurring in the brain.

(UN)EXPECTED RESEARCH CHALLENGES – PREDATORY JOURNALS

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An essential part of the research is publishing the results and interpretating them. Publications are one of the most important indicators of the progress of research work and how successful a research project has been. However, the publishing process usually has to occur under certain constraints. The peer-review and publication acceptance process often takes considerable time and involves significant funding for open-access options. These circumstances create a niche for the emergence and expansion of journals and entire publishing groups that offer a rapid publication process for a fee, most often driven by financial interests to the detriment of research quality. There is growing concern in the scientific community about these practices and their consequences. Numerous articles have warned against the "predatory publishers" since the term was introduced in 2010, and later precisely defined in 2019. This presentation discusses definitions of "predatory publishers," some of their characteristics, the challenges they provoke, and the harm they cause, and also provides advice on how to avoid them.
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THE CONCEPT OF INCLUSIVE RESEARCH. SHARING OF EXPERIENCES

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The concept of inclusive research is at the heart of the shift from research on people to research with people. The application of this approach for more than a decade in various fields of science has proven to reduce researcher bias and stereotyping, ensure diversity and representativeness of the research population, and improve the quality and validity of studies. At the same time, this approach can provoke active discussion between researchers and participants and encourage them to consider the issues that concern them together. The opportunity to exchange perspectives, experiences, expertise and knowledge between researchers and participants has educational value, making individuals more prepared to participate in research and researchers more open to working with diverse populations. In this presentation, we look at some key features of the concept of inclusion, equality, and diversity in research and how these can be considered at each research project stage. We reflect on how this approach can be applied, drawing on our experience, the times, and the context in which we live.

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RECEPTOR MECHANISMS OF ANTINOCICEPTION INDUCED BY ANG 1-7 AND ITS SYNTHETIC ANALOGS

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Introduction - Many endogenous substances and systems are involved in the transduction of the pain signal and its modulation by antinociception. The renin-angiotensin system (RAS), which is essential for maintaining homeostasis and regulating various body functions, is involved in the mechanism of pain regulation. It consists of precursors, enzymes, and signal peptides forming two balancing arms: ACE/AngII/ AT1R, and ACE2/Ang1-7/Mas1R. Angiotensin II is the main active peptide of the classical arm which mediates vasoconstriction, proliferative, and pro inflammatory effects via binding to AT1-type receptors throughout the body. On the other hand, Angiotensin 1-7 (Ang 1-7) is a peptide belonging to the alternative (protective) arm of the RAS. Ang1-7 effects oppose the effects of AngII. Our study aimed to investigate receptor mechanisms of antinociception induced by Ang1-7 and its four structural synthetic analogs in the formalin test in mice.
The method involved an in vivo model - male ICR mice, which were distributed in three groups. The first group was administered intraperitoneally (i.p.) with Ang1-7 peptide and its analogs alone, second group was pretreated with A779 (a selective Mas1-receptor antagonist) before i.p. Ang1-7 peptide and its analogs, and the third group was pretreated with the selective opioid receptor's antagonist Naloxone before i.p. Ang1-7 peptide and its analogs. The Formalin test was used to evaluate the acute phase and inflammatory phase of nociception. The stereotype responses of licking or shaking the injected paw were observed and measured in seconds, separately for each phase of the experiment.
Results from the first group showed that Ang1-7 peptide and its analogs administered alone have antinociceptive effects, both in acute and inflammatory phases of formalin-induced nociception with the only exception of Pc5- peptide. The second group showed inhibition of antinociception compared to the first group for some of the peptides (Ang1-7, P2) and almost no change for P1 and Pc6 in the acute phase. Similar data was observed for the inflammatory phase - inhibition of antinociception effects compared to first group, where a tendency of inhibition of antinociception for Pc6 was detected. In the third group (peptides plus Nal) has been observed inhibition of antinociception effects in acute phase with high statistical significance for Ang1-7, P1, and P2 compared to first group (peptides administered alone) and similar results for the inflammatory phase – inhibition of antinociception for Ang1-7, P1, and P2. No significant changes in effect were observed for Pc 6 and especially for Pc5.
Conclusions - Present data showed well-defined antinociceptive effect of Ang1-7 and its synthetic analogs when used alone (with exception of the structural analog Pc5) in both acute and inflammatory pain. The Mas1 receptor is involved in the antinociception produced by Ang1-7 and P2 in the acute and inflammatory phases, but does not influence the antinociceptive effect of P1, suggesting a lack of involvement of Mas1 in the P1 effect. The antinociception of the investigated peptides, with the exception of Pc6 and especially Pc5, was reversible by naloxone, suggesting an opioidergic mechanism of their effects.
Acknowledgments: This work was financially supported by the Bulgarian National Scientific Fund project КП-06-Н71/9 of the Ministry of Education and Science, Bulgaria.

INVESTIGATING THE ROLE OF MELATONIN DEFICIENCY AND AGING ON MECHANISMS RELATED TO MEMORY FUNCTIONS

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Memory decline is a typical aspect of aging, though the link between melatonin deficiency and cognitive function remains complex and incompletely understood. This study examined the impact of age-related melatonin deficiency on working, short-term recognition, and spatial memory in rats, using the Y-maze, object recognition, and radial arm maze tests. Results showed age-dependent memory decline, with reduced brain-derived neurotrophic factor (BDNF) expression in the hippocampus. Pinealectomy, inducing melatonin deficiency, intensified BDNF reduction in the CA3 region of 3- and 14-month-old rats. Additionally, young adult rats with pinealectomy exhibited region-specific decreases in extracellular signal-regulated kinase (ERK)1/2 and phosphorylated ERK1/2, while middle-aged rats showed varied ERK1/2 expression across hippocampal regions. A lower pCREB/CREB ratio in the frontal cortex and hippocampus correlated with memory deficits in young and middle-aged melatonin-deficient rats, with no effect in older rats. These findings illuminate the role of the BDNF/ERK1/2/CREB pathway in age-related memory changes linked to melatonin deficiency and suggest a critical intervention window.
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E-POSTER PRESENTATIONS

EFFECTS OF EVENT PROBABILITY ON MOVEMENT REGULATION: A STUDY OF EVENT-RELATED POTENTIALS

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Background: It has been demonstrated that motor cortical potentials are reduced in simple as compared to choice-reaction tasks. Since in choice-reaction tasks, as compared to simple tasks, motor targets are delivered with lower probability, it has remained less well understood if modulations of motor potentials stem from different target probabilities or different task complexities. Also, it has been acknowledged that performance monitoring reflected by a medial-frontal negative potential is affected by the degree of mismatch between the planned and actual movement, but the effects of probability as a potential source of mismatch-guided monitoring have not been precisely explored for correct motor responses.
Objective: The present study aimed to clarify the effects of different target probabilities on motor and performance monitoring processes. To this end, a Go/NoGo task was used to elicit motor potentials at cortical regions, generating the response and correct response negativity (Nc) at medial frontal regions as markers of motor-related processes and performance monitoring, respectively.
Methods: Electroencephalographic signals were recorded at 32 electrodes in a group of young adults (n=27) while they produced responses with the two hands to Go stimuli in three auditory Go/NoGo tasks, each with different target probabilities (p=0.15, p=0.50, and p=0.85). The three Go/NoGo conditions were counterbalanced across participants. Performance markers (error rate and reaction times) were registered. Motor response potentials (MRPs) were computed and analyzed at electrodes over contra-lateral primary motor areas (C3 and C4). Correct response negativity Nc was analyzed at medial frontal/fronto-central electrodes. Repeated measures ANOVA designs were employed to explore the effects of probability (low vs. equal vs. high) and response side (left vs. right).
Results: Major results demonstrated that MRPs at movement generation regions contralateral to the response were larger for low- than equal- and high-probability targets. This effect was asymmetric and was more pronounced for right-hand responses over the left hemisphere. Nc did not depend on response probability. Response times were longer for equal-probability targets as compared to low- and high-probability targets.
Conclusions: These MRP observations demonstrate that motor generation processes are modulated by response probability. Yet, the effects of probability on MRP and response speed were different. Both the RT and MRP results can be explained with pre-activations preceding target delivery, with the dominating effect of local probability on RT and global probability on MRP. Also, probability effects on MRPs found in the right and left hemispheres are in line with stronger inhibitory pre-activations in the right hemisphere. Nc results suggest that in response inhibition contexts, performance monitoring is a continuous active process that is not modulated by implicit motor response probabilities.
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NO HEMISPHERIC ASYMMETRY IN THE GAIN OF IMPLICIT KNOWEDGE

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Background: Implicit abstract learning occurs when structured information is acquired without conscious access to the learned material. To explore implicit memory formation in humans, the Serial Reaction Time Task (SRTT) has been introduced. In the visual SRTT, the stimulation material has an overt (visuomotor choice reaction) and covert structure (predetermined sequence of items). In some, but not all people, implicit knowledge (ImK) is acquired during SRTT and is marked by faster and more accurate responses to regular sequences, without any awareness of sequence presence or structure. Brain mechanisms supporting the gain of ImK about abstract regularities remain largely unknown. Neurocognitive research has demonstrated that sensorimotor representations are coded and memorized in the hemisphere contralateral to the side of stimulation/response. It is acknowledged, however, that there is a right hemisphere dominance for visual attention and visual short-term memory, leading to benefits for items in the left visual field. Moreover, behavioural hemifield asymmetries may be reversed depending on memory load, with a left hemisphere dominance for complex as compared to simple memory sets. In addition, hemispheric differences have been established specifically for SRTT variants, suggesting that the left premotor cortex is activated during sequence acquisition, whereas the right premotor cortex is strongly engaged in the storage of sequences during advanced learning. In the context of these findings, the role of hemispheric lateralization as a source of ImK gain merits further examination.
Objective: The present research aimed to further highlight the hemispheric lateralization in implicit memory formation. To induce, by virtue of neuroanatomical projections, predominant sensorimotor activations and memory engrams in the contra-lateral hemisphere (left or right), a lateralized visual SRTT variant is employed. The relevant stimuli (different colors) appear in one hemi-field only, left or right, requiring choice responses with the ipsilateral hand. To manipulate memory load, complex and simple SRTT variants are designed.
Methods: The lateralized visual SRTT was practiced in implicit conditions. In one experiment, 109 participants trained a complex SRTT version (4 stimulus/response types followed a fixed sequence of 12 in regular blocks with 55 sequence repetitions). In a second experiment, 51 participants trained in a simple version of SRTT (3 stimulus/response types followed a fixed sequence of 6 with 60 sequence repetitions). Approximately half of the subjects in each experiment practiced the task on the left side, and the other half – on the right side. The gain of ImK was evaluated based on reaction time slowing upon regularity violation.
Results: In the complex SRTT, 43.6% and 40.7% of participants gained ImK upon training on the left and on the right side, respectively. In the simple SRTT, 37.5% and 47.4% of participants gained ImK upon training on the left and on the right. Chi-square statistics showed that in none of the conditions was there a significant difference in ImK gain between the left and right side training. Also, no difference was found for ImK gain between the complex and simple SRTT.
Conclusions: The accumulation of ImK about abstract sensorimotor regularities is not biased by hemispheric dominance at the behavioural level. Even if specific neural processes of ImK formation linked to engram encoding, representation storage, short-term memory capacity and attention are lateralized, asymmetric biases appear to be compensated at the level of behavioural output.
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HYPERURICEMINA AND ACUTE ISCHAEMIC STROKE

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Hyperuricemia is a condition characterized by an excessive increase in uric acid levels in the blood, affecting patients of all ages. It is often asymptomatic and is usually detected in association with symptoms such as joint pain, arthritis, gout, or complications involving the kidneys, cardiovascular system, or diabetes. Evidence worldwide indicates an increasing incidence of hyperuricemia, with prevalence varying based on ethnicity and region.
To date, only one epidemiological study on the prevalence of hyperuricemia (HU) has been published in Bulgaria, reporting a prevalence of 33.9% among 1242 patients in 2020. Globally, prevalence rates vary depending on ethnicity and region. Furthermore, there is a limited number of studies examining HU as a risk factor for acute ischemic stroke (AIS), and the existing data are often contradictory.
Objective: To evaluate the incidence of HU over a 6-year period in patients hospitalized with ischemic cerebral stroke at the Neurology Clinic of University First MHAT – Sofia, "St. Joan Krastitel."
Methods: A retrospective case-controlled study is conducted based on data from medical records of patients with AIS hospitalized between January 1, 2016, and November 17, 2022. The analysis included data from 2,807 patients with ischemic stroke and a control group of 3,241 individuals without stroke.
Results: The analysis revealed that the prevalence of hyperuricemia was significantly higher in the ischemic stroke group (42.4%) compared to the control group (36.7%). Female patients were predominant in the hyperuricemia group, particularly in combination with the non-modifiable risk factor of age over 60 years. Among ischemic stroke patients, the prevalence of hyperuricemia combined with triglyceridemia and arterial hypertension was higher, particularly in females over 60 years old.
Conclusion: This retrospective study indicates a significant relationship between hyperuricemia and ischemic stroke, particularly when combined with risk factors such as arterial hypertension, triglyceridemia, and hypercholesterolemia. Early identification of individuals with elevated uric acid levels and ischemic stroke risk may be an important step toward preventing stroke progression and reducing the occurrence of subsequent cardiovascular events.

NATURAL ISOQUINOLINE ALKALOIDS AFFECTING NEURODEGENERATIVE DISEASES

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Multifactorial diseases are a versatile group of medical conditions with complex etiology, caused by multiple genetic and environmental factors. Pathologies such as neurodegenerative disorders (NDDs), chronic inflammation, cardiovascular diseases, infectious diseases, diabetes, and cancer pose a hidden threat to global public health due to their increasing prevalence and the lack of effective therapies. The World Health Organization (WHO) warns about the growing burden of NDDs, primarily due to their prevalence, morbidity, and disability rates. Moreover, considering that neurological conditions increase with age, a significant rise in cases is expected as the global population progressively ages, posing a critical challenge for healthcare systems worldwide.

Parkinson's disease (PD) and Alzheimer's disease (AD) are the most common NDDs, pathophysiologically characterized by progressive neuronal loss associated with the accumulation of misfolded proteins. Their chronic, degenerative nature leads to a gradual decline in patients' quality of life. Advanced stages are entirely disabling, requiring specialized care and causing immense emotional and economic strain.

The discovery of effective and safe drugs remains essential for managing complex diseases. Despite advancements, treatments for multifactorial diseases often focus on single-target medications, which are insufficient in the long term. To address this, medicinal chemistry is exploring strategies to overcome traditional limitations, with multitarget drugs demonstrating significant promise. Natural products offer a unique source of biologically active heterocyclic compounds with diverse structures and multitarget potential. Alkaloids, in particular, possess anti-inflammatory, anticancer, cardioprotective, and neuroprotective properties, making them valuable candidates for treating chronic multifactorial diseases. Isoquinoline alkaloids, specifically, have attracted significant attention for their multimodal potential, especially in NDDs and cancer.

Given their structural diversity and widespread occurrence in nature, research on natural isoquinolines focuses on their distribution, pharmacological activities, molecular targets, and structural motifs. This review aims to summarize the current knowledge on isoquinoline alkaloids, highlighting their potential as multitarget therapeutic agents for complex diseases and providing a foundation for future studies in this promising area.

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METABOLIC EFFECTS OF THE AT1 RECEPTOR ANTAGONIST LOSARTAN IN YOUNG AND MIDDLE AGED SPONTANEOUSLY HYPERTENSIVE RATS

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Hypertension is a common disease associated with significant metabolic and cardiovascular alterations, often studied using rodent models to elucidate mechanisms and therapeutic interventions.

This study investigates the effects of aging and hypertension on metabolic parameters in 3-month-old (young adult) and 14-month-old (middle-aged) rats, focusing on the role of angiotensin II type 1 (AT1) receptor signaling. Losartan, a selective AT1 receptor antagonist, was administered to spontaneously hypertensive rats (SHRs) of both age groups for two weeks at a dose of 10 mg/kg, i.p. to evaluate its effects on metabolic homeostasis.

Age-related metabolic changes, including elevated plasma glucose levels and triglycerides were found in SHRs vehicle-treated. Losartan treatment significantly attenuated these alterations by reducing glucose levels and regulating the lipid profile in the plasma of both age groups. There were no significant changes in the 24h locomotor activity whereas the middle-aged group treated with Losartan showed an improvement in the distance traveled.

These findings highlight the therapeutic potential of AT1 receptor antagonism in alleviating hypertension-associated metabolic dysfunction in both young adult and middle-aged rats.

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IN SILICO MODELING OF BDNF SUPPRESSION EFFECTS ON SYNAPTIC PLASTICITY: A PATH TOWARD THERAPEUTIC INSIGHTS

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Brain-derived neurotrophic factor (BDNF) is crucial for regulating synaptic plasticity, influencing processes like long-term potentiation (LTP) and long-term depression (LTD) that underlie learning and memory. Understanding how BDNF gene suppression impacts these mechanisms can provide valuable insights into potential therapeutic approaches for neurodegenerative diseases, where synaptic function is often compromised. By utilizing digital modeling techniques, we can simulate genetic modifications, such as BDNF suppression, in a controlled virtual environment, enabling precise predictions of neural behavior changes. This approach allows researchers to explore complex biological interactions and test therapeutic hypotheses in silico, reducing the need for costly and time-consuming laboratory experiments.

This study investigates the effect of BDNF gene suppression on synaptic plasticity, particularly focusing on mechanisms such as LTP and LTD. Namely, suppression of the BDNF gene will significantly reduce BDNF production levels, leading to decreased synaptic plasticity, as indicated by reduced levels of LTP and increased LTD.

By modeling genetic modifications in silico, including CRISPR-based control of BDNF expression, this research aims to predict therapeutic outcomes for potential clinical interventions. The study utilized NetPyNE, an open-source Python package that facilitates the development, parallel simulation, analysis, and optimization of biological neuronal networks using the NEURON simulator to construct and simulate the neural network. This approach enabled the precise representation of pyramidal neurons with distinct excitatory and inhibitory properties, allowing for accurate simulation of synaptic plasticity changes under BDNF gene suppression.

Results: The study establishes a neural network with pyramidal neurons and simulates the impact of BDNF suppression, revealing reduced LTP and increased LTD. Future directions include extending the model to disease contexts, such as neurodegenerative conditions, and evaluating BDNF-based therapeutic strategies to restore synaptic function and reverse deficits observed in these disease models.

Future steps:

The next phase of this research will involve expanding the current model to encompass larger neural circuits or specific brain regions impacted by neurodegenerative diseases, such as Alzheimer's and Huntington's, to better understand the role of BDNF suppression in disease pathophysiology. Additionally, simulations will be conducted to assess the effects of BDNF gene therapy or pharmacological interventions, with the aim of evaluating their potential to restore synaptic function and reverse synaptic deficits in these disease models.

The digital modeling of BDNF gene suppression provides valuable insights into its impact on synaptic plasticity, particularly through reduced LTP and increased LTD. These findings highlight the potential of in silico approaches to predict therapeutic outcomes, guiding future research toward targeted interventions for neurodegenerative diseases.

MUTUAL INFLUENCE OF NOVEL ISOQUINOLINE DERIVATIVES AND MAIN NEUROTRANSMITTERS

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Isoquinoline derivatives have garnered significant interest in pharmacological research due to their diverse biological activities and potential as therapeutic agents. This study focuses on the interaction between novel isoquinoline derivatives and two primary neurotransmitters: acetylcholine (ACh) and serotonin (5-HT). Understanding these interactions can illuminate the role of isoquinoline derivatives in modulating cognitive, mood, and neurodegenerative processes.

Isoquinolines, their precursors, and biologically active derivatives are under investigation due to their chemical diversity and potential to form new categories of pharmaceutical agents. Evaluating their specific actions as bioactive compounds is essential for advancing drug development. In this study, two novel derivatives, IQP and DIQ, were synthesized and characterized. IQP is an isoquinoline precursor with an amide structure, while DIQ is a dihydroisoquinoline derivative with a cyclic structure. Their effects on smooth muscle (SM) contractility and neurotransmitter modulation were examined.

Experiments showed distinct differences between IQP and DIQ in influencing the spontaneous contractile activity of circular smooth muscle from rat stomachs. IQP had a stabilizing effect on ACh-induced contractile responses (1×10^{-6} M), with no significant changes observed after premedication with 5×10^{-5} M IQP for 10 minutes. However, IQP significantly reduced the contractile response induced by 5-HT. Additionally, IQP-induced relaxation was enhanced nearly threefold in the presence of 5-HT, suggesting the activation of parallel pathways. DIQ (5×10^{-5} M) demonstrated contrasting effects compared to IQP, highlighting the role of structural differences in determining bioactivity.

These findings illustrate the bidirectional influence between exogenously applied neurotransmitters and newly synthesized isoquinoline derivatives. The ability of IQP and DIQ to selectively modulate neurotransmitter systems suggests their potential application in the treatment of gastrointestinal and neuropsychiatric disorders.

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PROTECTIVE EFFECT OF RESVERATROL LOADED POLYMERIC MICELLES AGAINST H_2O_2 – INDUSED OXIDATIVE STRESS IN U87MG GLIOMA CELLS

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Oxidative stress plays a critical role in the progression of Alzheimer's disease. Numerous studies have reported that resveratrol (RVT) exhibits strong antioxidant and cytoprotective effects in brain pathologies associated with oxidative stress. However, its low solubility and bioavailability limit its therapeutic potential. Encapsulation of RVT in nanoparticles offers a promising strategy to enhance its effectiveness.

The aim of this study was to evaluate the effectiveness of resveratrol loaded micelles (mRVT) as a neuronal delivery system. For this purpose, we assessed cell viability and cellular uptake efficiency after mRVT treatment. In addition, DNA damage protective effect of mRVT and its antioxidant capacity against hydrogen peroxide (H_2O_2)-induced cytotoxicity in U87MG glioma cells were also tracked. Pluronic ((P123/F127) copolymer micelles, with an average diameter of less than 50 nm, were formed via a film hydration method. The micelles' cellular uptake efficiency was evaluated using red fluorescent rhodamine B as a visible marker. The cytoprotective effect of mRVT in concentrations 1, 3, 10 and 30 μM were evaluated in H_2O_2 (200 μM) induced oxidative stress model in human glioblastoma cells (U87). The cells were pretreated with mRVT for 24h. The cell viability was quantified by MTT assay, and DNA damage by the alkaline comet assay. The specific biomarkers of oxidative stress – glutathione peroxidase (GPx) and acetylcholine esterase (AChE) inhibitory activity of mRVT were determined by spectrophotometric methods.

Our findings revealed that rhodamine B-loaded nanoparticles were localized in the cytoplasm of U87MG cells within 1 hour of incubation. mRVT (1 μM) pretreatment significantly increased, reduced by the H_2O_2 cells viability, increased DNA damage index, and decreased AChE and GPx activity.

The present data indicate the protective capacity of the water solution of resveratrol loaded in micelles against the oxidative stress induced by hydrogen peroxide. However, further studies are needed to dipper understand the neuroprotective mechanism of nanoparticles.

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OXIDATIVE STRESS IN THE LIVER AND KIDNEYS OF MICE EXPOSED TO MICROPLASTICS – PILOT STUDIES

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The increasing use of plastic and their low recycling have contributed to widespread environmental pollution, posing a significant threat to ecosystems. Recent research has focused on the impact of micro- and nanoplastics on organisms, highlighting their ability to penetrate tissues and organs. In this regard, this pilot study aimed to evaluate the effect of polystyrene microplastics (PS-MPs) on the oxidative status of kidneys and liver, measured by the levels of lipid peroxidation (LPO) and glutathione (GSH) in them. Six sexually mature male ICR mice were used. They were divided into a control group and an experimental group, in which the animals received 1 μm PS-MPs at a dose of 0.1 mg/24 hours through drinking water for 14 days. Results showed that after the PS-MPs intake by the mice, the LPO levels in both studied organs decreased significantly (liver $P \leq 0.001$ and kidneys $P \leq 0.01$), while the GSH concentration increased significantly in the liver ($P \leq 0.01$) and decreased insignificantly in the kidneys. In conclusion, the results of the conducted pilot study revealed that the intake of MPs probably activates the liver antioxidant defense, which prevents the development of lipid peroxidation. Further studies are needed to elucidate the mechanisms of action of PS-MPs on organs.

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EFFECT OF POLYSTYRENE MICROPLASTS ON THE OXIDATIVE STATUS OF THE REPRODUCTIVE ORGANS OF LABORATORY MALE AND FEMALE RATS – PILOT STUDIES

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One of the serious problems of modern society is the progressively increasing infertility, which coincides with the increasing pollution of the environment with microplastics. In recent years, more and more data have linked the two problems as a cause-and-effect relationship. However, the studies conducted are insufficient. In this regard, our study aimed to assess the influence of polystyrene microplastics (PS-MPs) on the oxidative status of the reproductive organs - ovaries and testis. Six sexually mature male and female WISTAR rats were used. They were divided into control and experimental groups. The latter received 1 μm PS-MPs through drinking water at a dose of 0.1 mg/24 hours for 2 weeks. In the tissue homogenates, the main markers of oxidative stress - lipid peroxidation (LPO) and glutathione (GSH) levels were measured spectrophotometrically. The results showed that the intake of PS-MPs led to a decrease in the levels of LPO in the ovaries, while in the testis an increase was observed. In comparison to control animals, the intake of PS-MPs led to a significant decrease in the GSH concentration in both sexes (♀ 1130.69±157.81 vs 852.41±72.67 ng GSH/mg protein; ♂ 422.12±3.11 vs 203.50±4.39 ng GSH/mg protein; $P \leq 0.001$). In conclusion, the first pilot study in Bulgaria of the effect of PS-MP on the reproductive organs of male and female rats showed that PS-MPs induced different degrees of oxidative stress. Undoubtedly, future research is needed to uncover the mechanisms of damage to the reproductive organs by PS-MPs and to seek measures to address the problem.

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EXPERIMENTAL MODEL OF ALZHEIMER'S TYPE DEMENTIA: BEHAVIOURAL EFFECTS OF A NEWLY SYNTHESIZED ARYLHYDRAZONE DERIVATIVE OF 5-METHOXYINDOLE-2-CARBOXYLIC ACID

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Dementia, in particular Alzheimer's disease, is the most prevailing global neurodegenerative disorder, with increasing levels of both incidence and mortality. Due to the still unknown exact aetiology of the disease and its complex pathophysiological mechanisms, current treatment is symptomatic, giving rise to the need for multi-targeted therapy agents. N'-(3,4-dihydroxybenzylidene)-5-methoxy-1H-indole-2-carbohydrazide (5MeO), an arylhydrazone derivative of 5-methoxyindole-2-carboxylic acid, presents potential as such an agent owing to its promising safety profile, significant neuroprotective effects, and its blood-brain barrier permeability. The neurobiological activity of 5MeO was investigated in an Alzheimer-type dementia model induced by Scopolamine (2mg/kg) in adult male Wistar rats, with effects on learning and memory evaluated by behavioural assessments such as the Step-through inhibitory (passive) avoidance test and the Barnes maze test. Animals were divided into the following groups: Control, Scopolamine (Sco), Rivastigmine and Scopolamine (Riva+Sco), Polyethylene glycol (PEG+NaCl), 5MeO and Scopolamine (5MeO+Sco). Results were consistent with Alzheimer-type dementia. Scopolamine induced memory and learning acquisition impairments were seen on the 24h after the first scopolamine injection, namely, decreased step-through latency in the step-through test and on the 12th day after the start of scopolamine application, namely, increased head-dips in the Barnes maze test. 5MeO demonstrated neuroprotective effects in both behavioural assessments: in the Step-through test, levels of step-through latency in rats treated with scopolamine were restored to those of the rats in the control group, while the number of head-dips of scopolamine-treated animals in the Barnes maze test decreased to that of the control group.
In conclusion, our investigations demonstrated the derivative's neuroprotective effects on memory and learning and present N'-(3,4-dihydroxybenzylidene)-5-methoxy-1H-indole-2-carbohydrazide (5MeO) as a persuasive candidate for further pharmacological studies.
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IN VITRO EFFECTS ON *A.BASILARIS* OF GRAPHENE OXIDE, REDUCED GRAPHENE OXIDE AND RGO AS CARRIER OF ACHILLEA MILLEFOLIUM EXTRACT

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The aim of our study was to investigate the *in vitro* effects of graphene oxide (GO), reduced graphene oxide (RGO), and the RGO-A. millefolium composite on small blood vessels.
Yarrow alcohol extract (*A. millefolium*) was prepared by maceration and applied by impregnation onto reduced graphene oxide (RGO). The samples were characterized by X-ray diffraction (XRD), ATR-FTIR, and SEM. The samples were examined in vitro for physiological effects on small blood vessels by wire myography.
The samples showed a transient effect on the tone of the vascular wall (in GO) and insignificant changes in RGO. The samples of impregnated RGO showed a distinct reduction in vascular tone, to a greater extent than the extract applied alone. The effects are probably due to the increased reactivity of GO due to the presence of oxygen-containing functional groups in its structure and presumably induction of intracellular oxidative stress.

POLLUTION WITH FINE DUST PARTICLES AS FACTOR IN THE DEVELOPMENT OF SOME NEURODEGENERATIVE DISEASES

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Environmental pollution with various persistent chemical pollutants is an important etiological factor in the development of some neurodegenerative diseases. The harmful effects of fine dust particles (FDP) are well documented in respiratory, reproductive and cardiovascular diseases, but in neurological and cognitive disorders, they are still the subject of analysis and evidence gathering. The exact mechanism of their impact on the processes of neurodegeneration is still not well understood.

New research shows that air pollution with FDP can accelerate or worsen the progression of dementia, not only in the elderly but also in the younger generation. It has been found that long-term exposure to FDP increases the development of Parkinson's disease, Huntington's disease, multiple sclerosis, etc.

The summarized literature data reveal the complex relationship between the neurotoxicity of FDP and some neurodegenerative diseases.

The search for alternative therapeutic pathways at the cellular, subcellular, and molecular levels includes the development of new methods for timely diagnosis and prevention.

This also provokes the need for preventive measures for stricter public health policies.