

Speakers

► Speaker 1

What Does Molecular Neuropsychiatry Promise?

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ABSTRACT

This study aims to evaluate the promising paradigms offered by molecular neuropsychiatry in understanding and treating complex central nervous system disorders [1,2]. Traditional approaches in psychiatry and neurology rely heavily on clinical observation and symptom-based diagnostic frameworks; however, contemporary advances in biotechnology necessitate a shift toward etiology-driven biological interventions [3].

Molecular neuropsychiatry integrates genomics, pharmacogenetics, neuroinflammatory cascades, and synaptic plasticity pathways into clinical practice to elucidate the core pathophysiology of neuropsychiatric conditions [1,3]. By combining neurotechnological modalities—such as quantitative EEG and functional brain mapping—with biomarker discovery and genetic profiling, this field offers personalized treatment options. [2]. Recent clinical data indicate that biomarker-guided precision medicine significantly improves treatment outcomes in treatment-resistant psychiatric cases and reduces the risk of adverse effects through optimized pharmacotherapy. Consequently, molecular neuropsychiatry transforms psychiatric conditions from purely symptom-oriented diagnostic clusters into personalized, targeted, and evidence-based biological medicine [2,3].

Keywords: Biomarkers, Pharmacogenetics, Precision medicine, Personalized medicine molecular neuropsychiatry, Neuroplasticity.

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► Speaker 2

Molecular Development of a Neuron** F. Figen Kaymaz**

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ABSTRACT

Neuronal development is a highly coordinated process through which neural stem cells acquire neuronal identity, migrate to appropriate locations, establish polarity, extend axons, and form functional synaptic connections. Neural development begins during gastrulation with neural induction, in which inhibition of BMP signaling promotes neural fate. Subsequent interactions among Wnt, FGF, and Sonic Hedgehog (Shh) signaling pathways regulate the regional patterning of the developing neural tube. Neural stem and progenitor cells expressing factors such as SOX2, Nestin, and PAX6 undergo tightly controlled proliferation and differentiation. Notch signaling maintains progenitor characteristics, whereas proneural transcription factors including Neurogenin, ASCL1, and NeuroD promote neuronal differentiation and cell-cycle exit.

Following neurogenesis, newly generated neurons migrate through radial and tangential mechanisms to reach their appropriate destinations. Reelin, LIS1, and Doublecortin contribute to neuronal migration and cytoskeletal organization. Neuronal polarization subsequently establishes a single axon and multiple dendrites through coordinated activity of molecular pathways including PAR3/PAR6, LKB1, and PI3K-Akt. Growing axons navigate toward their targets by responding to extracellular guidance molecules such as Netrin, Slit, Semaphorin, and Ephrin. Once target regions are reached, synaptogenesis is mediated by coordinated pre- and postsynaptic molecular interactions. Neurotrophins, including NGF, BDNF, and NT-3, further regulate neuronal survival, maturation, and synaptic plasticity. Finally, developmental synaptic pruning refines neural circuits and contributes to functional maturation. Disruption of these processes may result in neurodevelopmental and neurological disorders. Understanding the molecular mechanisms underlying

neuronal development provides important insights into normal brain development, disease pathogenesis, and potential regenerative strategies.

Keywords: Neuronal development, Neural stem cells, Neurogenesis, Axon guidance

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► Speaker 3

TRP Channels in Bipolar Disorder

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ABSTRACT

Bipolar disorder is a chronic neuropsychiatric disorder characterized by alternating episodes of mania and depression and involving complex neurobiological mechanisms. The etiological mechanisms underlying bipolar disorder at the cellular and molecular levels have not yet been fully elucidated. Recent studies have demonstrated that abnormal neuronal excitability, impaired synaptic plasticity, dysregulation of intracellular calcium (Ca²⁺) homeostasis, and chronic low-grade neuroinflammation may play important roles in the etiopathogenesis of the disorder.

Dysregulation of calcium signaling is considered one of several mechanisms contributing to the pathophysiology of bipolar disorder. Abnormal calcium homeostasis may affect neuronal excitability, neurogenesis, and neuroinflammation, all of which are closely associated with the regulation of mood.

Transient receptor potential (TRP) ion channels are non-selective cation channels that detect a variety of cellular and environmental stimuli, including temperature, mechanical stress, lipid-derived signals, changes in pH, and reactive oxygen species (ROS).

TRP channels are widely expressed throughout the central nervous system (CNS), including in neurons, astrocytes, and microglial cells. By sensing changes in the cellular microenvironment, these channels regulate the influx of Ca²⁺ ions into cells and thereby modulate a wide range of intracellular signaling pathways. Increasing evidence suggests that dysregulation of TRP channel activity may contribute to the pathophysiology of mood disorders.

More specifically, several studies suggest that certain TRP channel subtypes, including TRPC4/5 and TRPV1, may contribute to the regulation of mood through their roles in calcium signaling, synaptic plasticity, and neuroinflammation.

Keywords: TRP Channels, Bipolar disorder, Intracellular calcium, Neuroinflammation, Neuronal excitability.

► Speaker 4

Keywords: Coefficient of error, Coefficient of variation, Disector, Stereology, Systematic random sampling

Quantitation of Tissues and Organs by The Stereological Techniques

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ABSTRACT

Stereological techniques, which provide unbiased and efficient quantitative analysis of tissues and organs, are important for data estimation. These methods allow sampling from every part of the organ, tissue, and tissue components under examination, enabling the acquisition of information about the whole from representative parts of the samples. The foundation of stereology, a science encompassing many methods, rests on two main pillars: systematic random sampling and efficiency. The first rule ensures equal sampling opportunities for every part in the biological structure being examined. Another critical step in the stereological analysis, efficiency, is determining the appropriate sampling strategy through a pilot study. Data obtained by stereological methods are independent of tissue complexity and content because they assess samples in 3D. In tissue or organ evaluation, the Cavalieri principle is used with a point probe to estimate the volume of tissue components and the ratio of each component to the total volume, while the disector tool, a volume probe, is used for cell counting. The disector method is widely used to estimate the total number, regardless of particle size or orientation. Particle counting, i.e., synapses, cells, and glomeruli, can be performed using two methods based on the same logic: physical and optical disectors. In stereological analysis of tissues and organs, the use of appropriate parameters and the appropriate number of subjects (i.e., efficiency) depends on the coefficients of error (<0.05) of a subject and the coefficients of variation (<0.2) for a group, both of which can be estimated in a pilot study. These techniques are widely used in neuroscience research to detect neurodegeneration. In conclusion, stereological methods enable the numerically reliable and reproducible determination of the morphological characteristics of biological structures.

► Speaker 5**Keywords:** Neurocardiology, Cardiovascular disorders**A Neuroscientific Perspective on Heart Function**** Ahmet Avcı**

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ABSTRACT

The nervous and cardiovascular systems play a crucial role in regulating human physiological, cognitive, and emotional states. The brain and heart are in constant interaction through anatomical and functional connections. When a problem arises in this relationship, neurological conditions can lead to cardiovascular disorders. Conversely, cardiovascular disorders can significantly affect brain health. The connection between the brain and the heart is established via neural, mechanical and biochemical pathways. The physiology of the brain and heart, and their interactions, have been of interest to researchers in neuroscience, psychiatry, neurology, and cardiology for many years.

The term 'neurocardiology' began to appear in clinical articles published in the 1980s and 1990s. These articles argued that attention must be paid to the complex interactions between the brain and the heart, as failure to do so could lead to delays in diagnosis and treatment. Neurocardiology emerged as a field that bridges the clinical gap between the brain and the heart. It provided insights into how pathologies in one system might affect the other. However, it took time for the interest in neurocardiology to be reflected in clinical practice, and apart from a few articles, it was largely overlooked until the 2010s. The interrelationships between brain and heart pathologies and their clinical outcomes are attracting increasing attention. Neurological conditions such as stroke, epilepsy, panic disorders and mood disorders can lead to arrhythmias and other cardiovascular disorders. Hundreds of important studies have been published to date regarding the relationships between the brain and the heart. This number has increased significantly, particularly in the last decade. However, these relationships, vital to understanding interactions between the two organs, remain largely undefined.

► Speaker 6

5-HT7 Receptors as New Therapeutic Targets**ID Elif Çadircı**

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ABSTRACT

Serotonin (5-hydroxytryptamine, 5-HT) is a biological amine and the presence of 14 different receptor subtypes makes it easy to explain the broad physiological effects of this neurotransmitter. Serotonin plays a role in the pathophysiology of many diseases such as depression, anxiety, circadian rhythm, schizophrenia, bulimia nervosa, anorexia nervosa, asthma, inflammation, etc. It is known that chronic inflammation has a vital role in tumor carcinogenesis and tumor development. 5-HT7 is also one of the most recently discovered but least characterized receptors for serotonin. The presence of this receptor was first demonstrated in 1993 by many independent laboratories. When tissue distribution is investigated, it has been shown in thalamus, hypothalamus, cerebral cortex, hippocampus and amygdala. In recent years, many studies have been conducted on the functions of these receptors. The effects of 5-HT7 receptors on the central nervous system have been studied in detail, and it has been shown that they play a role in circadian rhythm and thermoregulation. It has been claimed that it plays a role in learning and memory functions related to the hippocampus and also contributes to neuroendocrine regulation. However, studies on the peripheral expression of these receptors are very limited. In peripheral tissues, 5-HT7 receptor mRNA has been demonstrated in the ileum, spleen, endocrine glands, and arteries. Because of their presence on smooth muscles in the periphery, their possible effects have been investigated and it has been shown to play a role in irritable bowel syndrome. It has been shown that these receptors are located in arteries such as pulmonary, coronary arteries and aorta. It has been claimed that this effect of 5-HT7 receptors in the relaxation of smooth muscles in the gastrointestinal tract and vascular bed is mediated by adenylate cyclase.

The roles of these receptors in inflammatory processes,

whose peripheral effects are less studied than their central effects, were studied by our research group. It has been shown in our laboratories that 5-HT7 receptors, which are low in healthy tissues in the periphery, increase in conditions such as inflammation, sepsis, myocardial infarction. In these pathologies modeled in experimental animals, the 5-HT7 receptor agonist caused both a decrease in inflammation and an improvement in other parameters. An increase in 5-HT7 receptor mRNA expression has been shown in liver damage induced by different chemicals such as paracetamol, ethanol and carbon tetrachloride, and it has been shown that both the damage and this expression increase regress when a 5-HT7 receptor agonist is applied. In the continuation of these studies, the roles of 5-HT7 receptors, which we think are related to the immune system and contribute to pathological events as a defense mechanism of our body, in stomach, breast and prostate cancers were investigated. An increase in the expression of 5-HT7 receptors was observed in these cancers, and a significant anticancer effect was demonstrated in the treatment with a receptor antagonist. Thus, it has been shown that 5-HT7 receptors are involved in the development and treatment of cancer as well as playing a role in many physiological mechanisms in the central nervous system. The expression of these receptors was higher in breast, prostate and gastric cancerous tissues compared to healthy tissues. The increased expression of 5-HT7 receptors in cancer tissues suggested that these receptors may be increased to contribute the bad prognosis in cancer and/or rebound mechanism of the defense system. In our studies, the increase of 5-HT7 receptors in different pathologies and the regression of the pathologies after agonist and/or antagonist administration are very important for the development of alternative treatment methods. In this context, 5-HT7 receptors should be examined in more detail in in vitro, in vivo and clinical studies as new drug targets.

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Keywords: Serotonin, 5-HT7 receptors, Inflammation, Sepsis, Cancer

► Speaker 7

Free-Living Amoeba: From Environmental Amoeba to Brain-Eating Pathogen – Cellular and Molecular Mechanisms of Neuroinvasion**id Mehmet Aykur**

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ABSTRACT

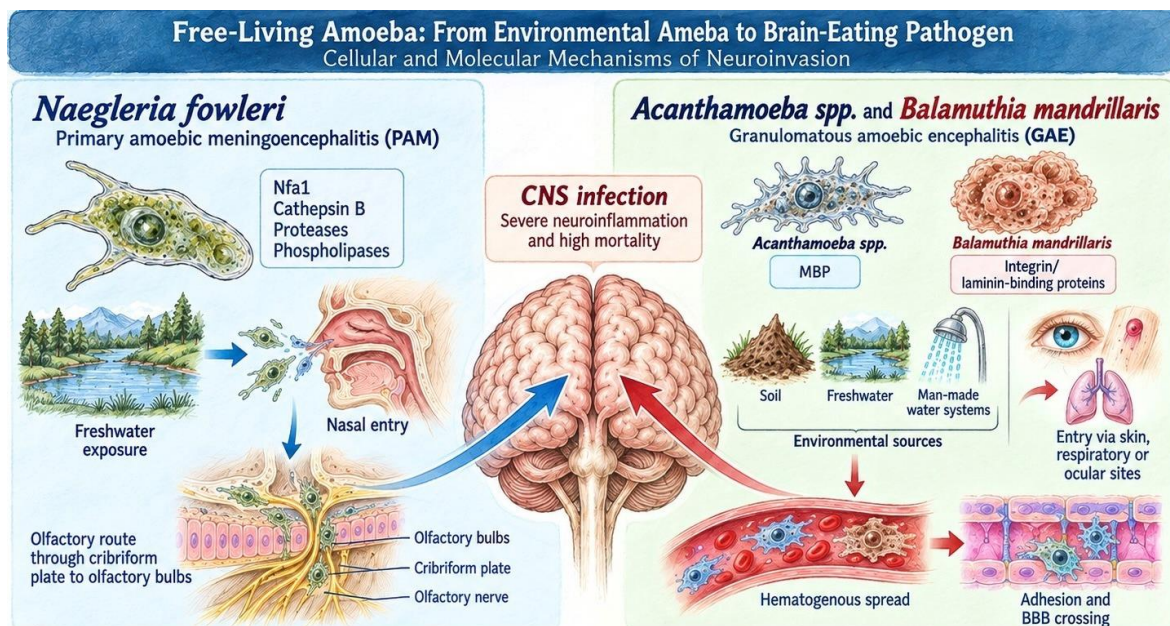
Free-living amoebas are ubiquitous protozoa that inhabit a variety of environmental settings, including freshwater, soil, thermal waters, and man-made water systems. Pathogenic free-living amoebas such as *Naegleria fowleri*, *Acanthamoeba* spp., and *Balamuthia mandrillaris* cause central nervous system (CNS) infections with mortality rates ranging from 80% to 97%. *N. fowleri* causes primary amoebic meningoencephalitis (PAM), a rapidly progressing infection leading to death within hours to days, while *Acanthamoeba* spp. and *B. mandrillaris* cause granulomatous amoebic encephalitis (GAE), a subacute to chronic disease that occurs mostly in immunosuppressed but rarely in immunocompetent hosts. This study aims to provide a comparative overview of the cellular and molecular mechanisms underlying the transformation of these organisms from free-living environmental amoebas to brain-penetrating pathogens.

N. fowleri enters the host via nasal exposure to contaminated freshwater, adheres to the olfactory epithelium via the Nfa1 adhesion protein and cathepsin B, and disrupts epithelial integrity via naegleriapores, proteases, and phospholipases. Trophozoites migrate directly to the olfactory bulbs via the cribriform plate

along the olfactory nerve, completely bypassing the blood-brain barrier (BBB). In contrast, *Acanthamoeba* species adhere via mannose-binding protein (MBP) and laminin/fibronectin receptors, while *B. mandrillaris* utilizes integrin and laminin-binding adhesion proteins; both genera secrete metalloproteases that degrade serine, cysteine, and tight junction proteins (e.g., ZO-1, occludin), enabling paracellular and transcellular crossing of the blood-brain barrier following hematogenous diffusion from primary skin, respiratory, or ocular sites. Despite these divergent entry routes, all three genera converge on shared virulence strategies, including contact-dependent adhesion, cytoskeletal remodeling, proteolytic barrier disruption, oxidative stress resistance, and manipulation of host signalling pathways such as MAPK, PI3K/Akt, and NF- κ B. Once within the CNS, trophozoites activate microglia and astrocytes, triggering the release of pro-inflammatory cytokines (IL-1 β , IL-6, TNF- α) and reactive oxygen species, driving neuroinflammation, blood-brain barrier breakdown, and ultimately neuronal injury. Clinical management remains severely limited by nonspecific early symptoms that delay diagnosis, poor CNS penetration of available antimicrobial agents, and the absence of standardized, evidence-based treatment protocols.

Although *Naegleria*, *Acanthamoeba*, and *Balamuthia* differ in their portals of entry and speed of CNS invasion, they converge on common virulence mechanisms, including adhesion, proteolytic barrier disruption, and manipulation of host signalling pathways. Advancing cerebral organoid models, multi-omics biomarker discovery, and improved BBB-penetrating drug delivery systems will be critical for enabling earlier diagnosis and more effective therapeutic strategies against these rare but highly lethal infections.

Keywords: Free-living amoeba, *Naegleria fowleri*, *Acanthamoeba* spp., *Balamuthia mandrillaris*, Neuroinvasion, Blood-brain barrier, Neuroinflammation



► Speaker 8

From Biomolecular Modeling to *In Silico* Drug Discovery: Current Approaches and Perspectives

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ABSTRACT

The integration of computational approaches into modern drug discovery has significantly accelerated the identification and characterization of promising therapeutic candidates. Biomolecular modeling provides a powerful framework for investigating molecular structure, ligand-target interactions, and the physicochemical properties that influence drug activity and developability. In this context, *in silico* methods have emerged as valuable tools for reducing the time, cost, and experimental workload associated with conventional drug discovery pipelines. This presentation provides an overview of current approaches in biomolecular modeling and their applications in *in silico* drug discovery, with particular emphasis on molecular docking, computational ADMET profiling, and toxicity prediction. The former enables the

characterization of potential ligand-protein interactions of potential ligand-protein interactions and the prediction of plausible binding modes, thereby supporting the identification and prioritization of candidate molecules. Complementary *in silico* ADMET analyses are employed to evaluate key pharmacokinetic and drug-likeness properties, including absorption, distribution, metabolism, and excretion, while computational toxicity assessment provides an early indication of potential safety liabilities.

By integrating structural modeling, molecular docking, ADMET profiling, and toxicity prediction, computational workflows can provide a comprehensive assessment of candidate molecules beyond binding affinity alone. Such integrated approaches facilitate the prioritization of compounds with favorable efficacy-related and pharmacokinetic profiles while identifying potential toxicity concerns at an early stage of the drug discovery process. Overall, these computational strategies highlight the growing role of biomolecular modeling in guiding rational drug discovery and demonstrate the potential of integrated *in silico* workflows to complement experimental approaches and improve the efficiency of modern pharmaceutical research.

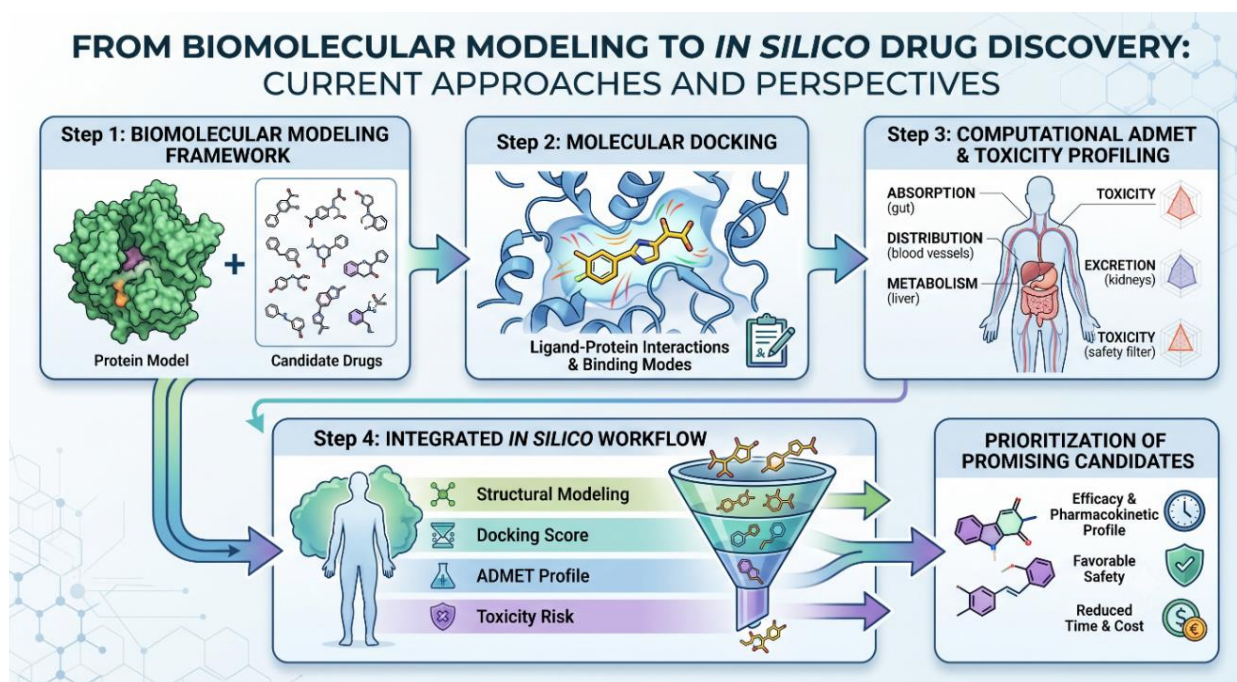


Figure 1. Schematic representation of the computational drug discovery pipeline

► Speaker 9**Artificial Intelligence, Robotics, and Digital Transformation in Neurosurgical Practice**** Aydın Sinan Apaydin**

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ABSTRACT

Rapid advances in artificial intelligence (AI), robotics, and digital technologies are transforming modern neurosurgical practice. These technologies have increasingly become integrated into different stages of patient care, including diagnosis, preoperative planning, intraoperative guidance, surgical treatment, and postoperative follow-up. AI-based approaches, particularly machine learning and deep learning, have demonstrated important applications in medical image analysis, tumor segmentation, prediction of clinical outcomes, and complication risk assessment. Deep learning architectures such as U-Net have also enabled automated analysis of complex neuroimaging data and may contribute to more accurate and efficient clinical decision-making.

Robotic systems represent another important component of this transformation. Platforms such as ROSA, ExcelsiusGPS, and Mazor X provide enhanced precision, reproducibility, and navigation during cranial and spinal procedures. In spinal surgery, robotic assistance may improve screw placement accuracy while potentially reducing radiation exposure, blood loss, and operative time. Furthermore, advanced three-dimensional navigation, augmented reality, and virtual reality technologies offer new opportunities for surgical planning, intraoperative visualization, and neurosurgical training.

The integration of 5G technology and telemedicine may further expand neurosurgical care by enabling remote monitoring and potentially supporting telesurgical applications. However, these developments also introduce important challenges regarding patient data security, algorithmic transparency, ethical responsibility, and legal regulation. Explainable AI and appropriate regulatory frameworks will therefore be essential for safe clinical implementation.

In conclusion, AI, robotics, and digital technologies have significant potential to improve precision, safety, efficiency, and personalized decision-making in neurosurgery. Their responsible integration is expected to play an increasingly important role in the future of neurosurgical practice.