different parvalbumin-expressing basket cells fired preferentially at different episodes during the task (e.g. stimulus, delay, execution). A difference in the firing of parvalbumin-expressing basket cells according to the decisions was observed often only after its execution and during or shortly after reward consumption. Thus, parvalbumin-expressing basket cells support the encoding of temporal sequences and distinct task episodes during working memory and decision making.

We have developed a novel technique that allows the recording of unequivocally identified neurons and show how distinct types of neuron contribute to prefrontal network operations and executive behavior. Our results indicate that GABAergic interneurons release GABA at distinct times to different domains of pyramidal cells contributing to the formation of cell assemblies and representations in the prefrontal cortex.

Speaker 3: John Krystal, USA
Title: Cortical disinhibition, noisy circuits, and schizophrenia: clinical and therapeutic implications.

John H. Krystal, M.D. (1), Alan Anticevic, Ph.D. (1), Genevieve Yang (1), Naomi Driesen, Ph.D. (1), Jose Cortes-Briones, Ph.D. (1), James M. Stone, MBBS. (2), Xiao-Jing Wang, Ph.D. (3), John D. Murray, Ph.D. (1)
(1) Departments of Psychiatry, Neuroscience, and Psychology, Yale University, New Haven, CT USA; (2) Institute of Psychiatry, Psychology, and Neuroscience, Kings College, London, UK; (3) Center for Neural Science, New York University, New York, NY USA

Abstract
It is not yet clear how the molecular and cellular alterations identified in post-mortem tissue from individuals diagnosed with schizophrenia translate into the disabling symptoms and cognitive impairments associated with this disorder.

Approach: The purpose of this presentation is to present results from a series of fMRI and EEG studies evaluating healthy individuals, schizophrenia patients, and healthy individuals administered the NMDA-R antagonist, ketamine that have been analyzed and interpreted within the context of parallel studies employing biophysically-informed computational neuroscience models of the behavior of cortical microcircuits and macrocircuits.

Results: Three types of network disinhibition are described: 1) gross disinhibition, “hyperactivity” that is associated with symptoms of psychosis, 2) disinhibition of the spatial dispersion of activation resulting in impairment in sparse coding of information, reduced memory precision, increased cortical signal variance “noise”, and the emergence of “false” memories, and 3) disinhibition of suppressed inputs, resulting in pathological hyperconnectivity. We will show evidence that the progression of schizophrenia is associated with altered signal properties of cortical functional connectivity, potentially creating the opportunity for illness phase-specific pharmacotherapy for this disorder.

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Speaker 4: Etienne Siblee, Canada
Title: SST-positive GABA neurons in depression and antidepressant treatment

Abstract
Background: Somatostatin (SST) deficits are common features in neurological disorders with mood disturbances, but little is known about the contribution or cause of these deficits to mood symptoms.

Methods: Human postmortem molecular and animal genetic/pharmacological studies.

Results: Results from human postmortem brains demonstrate molecular changes affecting SST-positive GABA neurons in depression. The mouse genetic studies suggest that low SST and reduced SST-positive GABA neurons have causal roles in generating illness symptoms and are targets for novel antidepressant modalities. Specifically, we show that mice lacking Sst exhibit elevated behavioral emotionality, high basal plasma corticosterone and reduced gene expression that recapitulate behavioral, neuroendocrine and molecular features of human depression. Using laser-capture microdissection, we show that cortical SST-positive interneurons display greater transcriptome deregulations after chronic stress compared to pyramidal neurons. Protein translation through eukaryotic initiation factor 2 (eIF2) signaling, a pathway implicated in neurodegenerative diseases, was most affected and suppressed in stress-exposed SST neurons. We show that activating eIF2 signaling through eIF2 kinase inhibition mitigated stress-induced behavioral emotionality in mice. Finally, as the function of SST-positive GABA neurons is mediated by post-synaptic GABA-A receptors containing the alpha5 subunit, we show that boosting Alpha5-mediated GABA function (through positive allosteric modulation) has antidepressant activity in chronically stress mice.

Conclusions: The data presented suggest that (1) low SST plays a causal role in mood-related phenotypes, (2) deregulated...
EIF2-mediated protein translation may represent a mechanism for vulnerability of SST neurons, (3) global EIF2 signaling has antidepressant/anxiolytic potential, and (4) boosting postsynaptic SST-positive GABA neuron signaling has antidepressant/anxiolytic potential.

Results: A summary of the results of the study, including sufficient details to support those conclusions. Include number of subjects and relevant statistics, and a clear statement about the novel and unpublished findings that will be presented. Abstracts with “results promised at a later date” will receive lower scores. These may be presented in a brief table. Data must be provided. Abstracts stating “data will be provided at a later date” will not be considered.

Conclusions: A statement concerning the significance of the work and its possible implications for future research.

S3: Neuroscience-based Nomenclature for Psychotropics
Chair: Pierre Blier, Canada
Co-Chair: Hiroyuki Uchida, Japan

Speaker 1: David Nutt, UK
Title: The new classification of drugs used for anxiety and insomnia
Abstract
My talk will focus on the use of the new ECNP nomenclature system to explain the mechanisms of actions of drugs for anxiety and insomnia so that prescribers have a better understanding of their modes of actions and adverse effects.

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Zohar J, Stahl S, Moller HJ, Blier P, Kupfer D, Yamawaki S, Uchida H, Spedding M, Goodwin GM, Nutt DJ (2015) A review of the current nomenclature for psychotropic agents and an introduction to the Neuroscience-based Classifications and Neuroanatomical Considerations of Psychotropic Drugs. Eur Neuropsychopharmacol. 25(8):711-722.
Nutt DJ. (2009) Beyond psychoanaleptics - can we improve antidepressant drug nomenclature? J Psychopharmacol. 23(4):343–5. Erratum in: J Psychopharmacol. 23(7):861.

Speaker 2: Pierre Blier, Canada
Title: The new classification of drugs used of major depressive disorder
Abstract
Background: The term antidepressant can be misleading in the field of health care. For instance, if the use of antidepressants is compared to the prevalence of major depressive disorder, it may lead to the conclusion that these medications are over prescribed. This is because antidepressants are used in mood and anxiety disorders, but also for the treatment of insomnia, chronic pain, and migraine prophylaxis.

Methods: 38 medications previously denoted as antidepressants were classified on the basis of their primary neurotransmitter target and others, if any, and how they interact with various neuronal elements. These elements could be for example reuptake transporters, receptors, and enzymes. When interacting with receptors, these medications could be acting as agonists, partial agonists, or antagonists.

Results: Classification of these medications, beyond the initial indication for which they were introduced on the market, led to a comprehensive scheme describing their exact pharmacology and helping physicians prescribe the next step when a first treatment or subsequent attempts have been made. Additional features of these medications summarized in an additional level of description provides other beneficial actions (sometimes with indications of specific targets being engaged at different doses), their most frequent side effects, and drug interactions, including warnings of potential deleterious actions.

Conclusions: This new nomenclature of medications used for depression will facilitate their rational site directed prescription and increase compliance by not exposing patients to a medication initially indicated for another psychiatric disorder, thus helping reduce stigma.

Speaker 3: Hiroyuki Uchida, Japan
Title: The new classification of drugs used for mania and psychosis
Abstract
The current nomenclature of drugs used for mania and psychosis is based on clinical indications; they are classified as “mood stabilizers” and “antipsychotic drugs”, respectively. While this conventional nomenclature has been widely used in clinical as well as research settings, there are a number of limitations to this system. First, boundaries among various categories of psychotropic drugs, using the current nomenclature have become unclear. “Antipsychotic drugs” and “mood stabilizers” are good examples; antipsychotic drugs are used for not only schizophrenia, but also mood disorders, including bipolar disorder and treatment resistant depression. On the other hand, mood stabilizers are often prescribed for a mood component in any psychiatric disorder. This discrepancy between their names and indications often confuses patients and their caregivers and sometimes leads to a misunderstanding of the effects of prescribed medications. This misunderstanding could have negative consequences on medication adherence. Second, up-to-date scientific knowledge on these drugs has not been reflected in the current nomenclature. This is a serious issue since the current system was created nearly half a century ago. For example, dopamine receptor antagonists and a partial dopamine receptor agonist are currently included in the same category of “antipsychotic drugs” despite the difference in their drug profiles. Moreover, the involvement of the serotonergic system also has to be considered for some drugs. However, such differences are not reflected in the current system. Third, unique and sometimes catchy labeling of particular drugs such as multi-acting receptor targeted antipsychotics (MARTA), serotonin dopamine antagonist (SDA), and dopamine system stabilizer (DSS), are proposed and initiated by pharmaceutical companies and often well accepted; however, they do not always accurately describe the mechanisms of action of those drugs. To overcome these limitations of the current nomenclature, the neuroscience-based nomenclature (NbN) was developed, which reflects our current neuroscience advances in a scientifically sound classification system. Antipsychotic drugs and mood stabilizers are now classified under the categories of “drugs used for psychosis” and “drugs for relapse prevention”, respectively. Moreover, within each category, drugs are classified based on their pharmacological profiles. So far, 26 drugs used for psychosis and 5 drugs for relapse prevention have been included, and more drugs will be added in the future. In this presentation, examples of multidimensional classification with respect to medications used for the treatment of psychosis and mania will be presented.