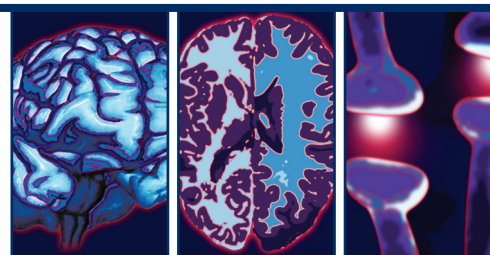


CONFERENCE SCENE



The American Academy of Child and Adolescent Psychiatry 57th Annual Meeting

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American Academy of Child and Adolescent Psychiatry, New York City, NY, USA, 26–31 October 2010

The American Academy of Child and Adolescent Psychiatry (AACAP) held its 57th Annual Meeting in New York City, NY, USA, on 26–31 October 2010. With over 5000 attendees, it was the largest AACAP meeting ever held. An incredible amount of information was disseminated, with a broad range of topics designed to inform attendees from diverse backgrounds and interests. The following three lectures help give a perspective of the breadth of the information shared.

In the Joseph Noshpitz Memorial Lecture, Judith Rapoport (National Institute of Mental Health, MD, USA) discussed the topic of ‘Psychopharmacology: too much or too little?’ In the USA, more so than any other country, there have been significant increases in the use of medications to treat psychiatric disorders in general, as well as childhood psychiatric disorders specifically. The use of medications in children has often been fraught with controversy. For example, historical skepticism on the diagnosis of attention deficit/hyperactivity disorder (ADHD) in general, and the use of stimulant medications, has more recently focused on whether or not patients with subsyndromal ADHD are being treated with medications when they may not need pharmacotherapy. Thankfully, significant advances in our knowledge base have helped to inform treatment planning for a variety of pediatric psychiatric disorders. Recent seminal studies, such as the Treatment

for Adolescents with Depression Study (TADS), the Child/Adolescent Anxiety Multimodal Study (CAMS) and the Pediatric Obsessive–Compulsive Disorder Treatment Study (POTS) have demonstrated the benefits of combined treatment (medication plus individual therapy) in achieving remission of symptoms for these diagnoses.

Interestingly, while we are gaining a better understanding of the role of currently available pharmacotherapies, the field has a significant void in the development of new targets around which medications are designed. Psychiatry is far behind other fields, such as cardiology, which have developed multiple new targets for the medications they use. In fact, when one looks at psychotropics in use today, the mechanisms of action have largely been known since the 1950s. The focus of today appears to be more so on modes of delivery and isolation of isomers.

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The idea that neuroscience is much more difficult to understand is likely central to the lack of new research in this area. Some challenging aspects may be attributable to the fact that many psychiatric diagnoses do not get made until patients have had the disease for a long time. Furthermore, there is an enormous underutilization of services when one considers the frequency of mental health disorders. And patients overall are more ill now than they ever were before. The National Institute of Mental Health is attempting to address these issues in a variety of ways, because it is clear that a better understanding of the neurobiological underpinnings of mental disorders will help to provide direction within our field and will shape what our field is able to accomplish in treating psychiatric disease. Therefore, significant opportunity exists in neuroscience research targeted at understanding, diagnosing and treating psychiatric disorders.

Even as the field of child psychiatry increases its understanding of neuroscience, often our understanding is applicable only to research arenas and not generalizable to clinical practice. Jeffrey Newcorn (Mount Sinai Medical Center, NY, USA) discussed the 'Clinical implications of neuroimaging in attention deficit/hyperactivity disorder', in an effort to begin to cross this chasm. The neurobiological basis for ADHD has generally been established for quite some time. Initially, however, the focus was isolated to the prefrontal cortex, but our understanding of the neurocircuitry involved in ADHD has broadened significantly to include the locus coeruleus, substantia nigra and the ventral tegmental area. Dopamine and norepinephrine regulate many of the manifestations of symptoms seen in ADHD. Dopamine suppresses irrelevant signals and norepinephrine enhances the relevant signals and helps to regulate dopamine. Many sites in the frontostriatal network are likely involved in these processes, including the prefrontal cortex (dopamine transporters D_1 , D_4 and D_5 , norepinephrine transporter, $\alpha 2a$), striatum (dopamine transporter, D_1), posterior parietal cortex

(norepinephrine transporter, $\alpha 2a$) and the cerebellum (dopamine transporters D_3 and D_2). Interestingly, as discussed later, we now understand that certain aspects of ADHD are not only attention related, but also motivation related.

Functional neuroimaging has been helpful in examining the brain at work. In functional MRI studies, for example, adults with ADHD were shown to process information less efficiently. Furthermore, one PET study in parents of children with ADHD showed decreased glucose metabolism in the premotor cortex and superior prefrontal cortex. Another PET study demonstrated decreased perfusion of bilateral orbitofrontal cortices as well as the cerebellum in children with ADHD, with increased perfusion of the right parietal and left parieto-occipital cortices. While it is difficult to directly translate these findings into clinically useful material, these data do demonstrate the biological underpinnings of ADHD and eventually may help to develop new targets for treatment. For example, another PET study looked at the dopamine transporter and its relationship to inattention. Individuals with greater inattention generally had lower levels of dopamine transporter. This, Newcorn suggested, could be a new target for treatment.

Whereas structural imaging is restricted in its ability to assess brain function, studies using these technologies have been useful in assessing structure and size. Consistently, lower cerebellum volumes and smaller prefrontal cortex volume have been reported in ADHD. However, it is not known how much of the difference in prefrontal cortex volume is white or gray matter and if it is localized to one area or not. Some studies have shown a smaller total caudate and corpus callosum area as well. However, not all findings are consistent. For example, another study demonstrated that caudate size seems to normalize over time, so it may not be as integral to understanding ADHD as initially conceptualized. Overall, total cerebral volume appears to be 3% less in ADHD youth, but effects from age and gender still appear to be much more influential. While there may be maturation over time, ADHD

children as a group have delayed cortical maturation that does not ever appear to catch up.

Lastly, in an interesting neuroimaging study assessing treatment effects, methylphenidate given to children with ADHD increased activation of the striatum and many other areas. However, when methylphenidate was given to control groups there was decreased activation in multiple areas of the brain. These results are difficult to interpret and the specific meaning of this is unknown.

While the neuroimaging studies are helpful in our understanding of the structural and functional biology of certain diseases, Daniel Pine (National Institute of Mental Health) gave an intriguing lecture on 'Anxiety and neuroscience', as an example of how neuroscience can help guide new treatments in psychiatry. Anxiety disorders are very common in children, however, many are transient and not maintained as adults. Nevertheless, even in studies where children show adequate response to medication, such as CAMS, approximately 50% of children had ongoing symptoms following treatment. Pine hypothesizes that new medications could be developed that target those areas of the brain that influence the remission or extinction of anxiety symptoms.

As an example, facilitating extinction through pharmacotherapy was discussed. Extinction is an important concept in anxiety disorders. A stimulus can be classified into three groups: always safe, used to be dangerous and still dangerous.

People with anxiety disorders cannot reclassify a stimulus as "no longer afraid" of something they used to be afraid of. Another aspect of anxiety is generalization of the fear. For example, in panic disorder, patients do not form the subtle boundaries around actual threats relative to ambiguous things. Pine encourages us to think of extinction and reclassification as forms of new learning. The NMDA receptor is important for new learning and may play a role in the ability to extinguish or reclassify. A glycine receptor site exists on the NMDA receptor and when this receptor is stimulated it seems to help extinguish anxious responses. In fact, ten studies of

D-cycloserine have been performed where subjects were randomized to either placebo or D-cycloserine, taken prior to exposure to a noxious stimulus. The compound seemed to facilitate extinction and a more robust decrease in anxiety occurred, which may help to usher in new strategies for the treatment of acute anxiety within the context of exposure therapy.

The potential for the field of neuroscience and its applications to child psychiatry are limitless. Not only do we need

to further understand the diseases with the help of neuroimaging, but new medication targets must be developed to alleviate suffering for the significant numbers who have psychiatric disorders. Our understanding of mental health disease and the treatments that will benefit patients the most is just beginning.

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