Treatments for People with Autism and Other Pervasive Developmental Disorders: Research Perspectives
Sponsored by: The NIH Autism Coordinating Committee and the Department of Education, Office of Special Education
Neuroscience Center, Rockville, MD 20852
November 8-9, 1999

A research workshop, jointly sponsored by four NIH institutes--NIMH, NICHD, NIDCD and NINDS--and the Department of Education, Office of Special Education, was held in November, 1999 at the Neuroscience Center in Rockville, Maryland. The purpose of the workshop was to develop a research agenda agenda with which to (1) further evaluate and refine existing treatments, (2) develop promising new treatments, and (3) extrapolate back from the clues that treatment research may offer to elucidate the underlying neurobiology of autism spectrum disorders. The workshop was organized into three sessions--psychoeducational/behavioral approaches, psychopharmacology, and emerging biomedical/clinical neuroscience approaches. Within each session, broad-based reviews of treatment-relevant research were presented and discussed to evaluate the state-of-the-science and identify critical research issues and needs. Following the presentations and discussions, four working groups met to formulate their recommendations to the NIH and Department of Education for needed research. Prior to adjourning, each group reported their recommendations back to the full session for final discussion.

The written reports and recommendations from the four working groups follow. The workshop agenda is appended at the end.

Please note that a special section or issue of the *Journal of Autism and Developmental Disorders* to be published later this year will contain papers based on the workshop presentations and responses.

Disclaimer: The views expressed herein reflect the opinions expressed in the working groups at the meeting and do not necessarily reflect the official views of the Autism Coordinating Committee of the National Institutes of Health, the Department of Health and Human Services, or the Department of Education.

Report of the Working Groups

Psychoeducational/Behavioral Interventions Working Group

Leader: Laura Schreibman, Ph.D.

Rapporteur: Helen Tager-Flusberg, Ph.D.

Research priorities

The group agreed that behavioral and psychoeducational treatments have been and will remain a high priority. It seems likely that all children with autism will receive these treatments in addition to other (psychopharmacological or other biologically-based) treatments. General agreement was reached that the following represent research priorities (not ranked for order of importance):

(1) Research to better understand variables (child-related, neurobiological, family-related, and cultural) that predict the range of responses (from excellent outcome to nonresponders) to early interventions.

(2) Research on early interventions for toddlers and young children that address core symptoms and underlying features of the disorder.

(3) Research to define the component features of interventions, replicate findings on efficacy, and compare existing treatment approaches available for individuals at different ages and developmental stages, including continuing care models and long term follow-up of intervention effects.

(4) Research which measures both the proximal and the distal effects of intervention, including developmental ecological and multisystemic outcomes.

(5) Research to identify developmentally sensitive outcome measures, including behavioral and neurobiological outcome measures, that are hypothesized to be associated with behavioral change.

(6) Research on the application of existing effective treatments to a range of settings, including schools and community settings.

(7) Research to develop innovative approaches, including the use of newly emerging technologies for treating children who are not responding to current treatment methods.

(8) Research to examine the effects of behavioral and psychopharmacological treatments, both in isolation and in combination, especially for problem behaviors.

Psychopharmacology Working Group

Leader: Donald Cohen, M.D.
Rapporteur: Eugene Arnold, M.D., M.Ed.

A. Questions and issues

The psychopharmacology workgroup considered the following questions and issues:

(1) What objectives are realistic, given the current limited knowledge on the pathogenesis of autism? Should the goal be to treat autism (i.e., disordered development per se) or to treat associated and secondary symptoms?

For now, we are restricted mainly to treating the latter. However, there is strong replicable evidence for a serotonin abnormality in autism and evidence in adults of the effectiveness of selective serotonin reuptake inhibitors (SSRIs). Nonetheless, it is unclear whether these findings extend to children with autism; the developmental aspect is not understood. We need to develop drugs that target and promote development.

(2) Should medication be seen mainly as a way to facilitate behavioral treatment or as a primary modality in its own right? Some of each is appropriate, but very little drug development research has been focused on the role that medications might play in facilitating the effects of behavioral treatments.

(3) How should psychopharmacology be informed / guided by family history and genetics?

(4) How can we achieve an understanding of the possibility of critical time windows in development for pharmacological intervention, analogous to the critical times for behavioral and educational intervention? In studying this, how do we sort out the confounds imposed by the use of other treatments and by the environment during the critical times?

(5) What is the appropriate length of a medication trial? The expense of long trials needs to be balanced against the need to continue the trials long enough to permit effects on the developmental process to be observed and evaluated. The appropriate length will vary depending on the target and goals of the treatment, but generally should be longer than the few weeks or months usually reported in the literature.

(6) How can we best study the effects of medication on learning? Medication may have ripple effects that impact upon school learning and performance. Skill acquisition curves would be a valuable addition to assessments in drug trials.

Obstacles to research that were identified were the small number of trained pharmacological investigators, lack of interest in developing drugs for autism on the part of the drug industry, subject flow for many studies, the sparsity of regional assessment centers able to carry out such research, and problems getting clinicians to refer subjects for medication trials.

B. Research needs and strategies

The following research needs and strategies to meet them were recommended:

Collaborative centers of excellence such as pediatric psychopharmacology research units (PPRUs) and research units on pediatric psychopharmacology (RUPPs) should be encouraged.

Rather than simple comparisons of new medications to placebo, drugs should also be compared to each other.

There should be a dual thrust which addresses both the development of new drugs that target developmental deviations and the testing of drugs used in clinical practice for which efficacy and safety data in children are lacking or inadequate. The development of new drugs could be led by specialized collaborative centers, while large comparison trials of already-marketed drugs could be done in clinical practice settings in collaboration with academic centers.

There should be research on polypharmacy, including interactions of drugs with each other and with nutrients. In other areas of medicine, drugs have long been recognized to require certain nutrient supplementation, and investigation of such interaction seems especially appropriate in autism, where nutritional and dietary treatments have been touted.

Clinicians should be educated about the advantages to their patients and themselves of referring to specialized centers. Greater effort should be devoted to developing collaborations between researchers and clinicians.

The full heuristic potential of subject samples should be mined by follow-up study of refractory subgroups and placebo responders. Such studies might initially be exploratory, leading to hypotheses to be tested prospectively. Although placebo response is considerably less common in autism than in most other child disorders, study of the occasional placebo responder may be very useful.

The natural waxing and waning of symptoms should be studied further, both for the confounding of the assessment of drug effects and for possible predictive clues as to which medication strategy may be most effective for which patients.

A common set of core assessment measures to allow comparison across studies would be useful, with additional measures specific to each study's goals.

Current gaps in assessment protocols could be addressed by innovative strategies such as wearable data collectors to measure effects in vivo. This will require some thought and decision as to what to measure and how.

The effect of drugs on facilitation of behavioral treatments should be studied more. This may at times be piggy-backed on more basic studies, but may well also require some specially designed studies with random assignment to different behavioral treatments crossed with placebo vs. active drug.

The value of family history as a predictor of treatment response could be tested easily and inexpensively by incorporating family history data into most trials by collecting family history at baseline (regarding both psychiatric diagnoses and drug responsiveness).

Studies should use narrower age ranges or stratify on age to prevent the clouding of results by developmental differences.

Target symptoms should be precisely defined and evaluated with respect to medication effects, while at the same time checking for overflow benefits in unexpected areas.

Drug effects on learning should be studied more, possibly by simply adding such assessments to more basic studies.

At least some proportion of trials should follow the subjects long enough to examine effects on developmental trajectories.

Biomedical and Clinical Neuroscience Working Group

Leader: Edwin Cook, M.D.
Rapporteur: Nancy Minshew, M.D.

Generally speaking, there is limited empirical data related to the prevalence or type of metabolic, immunologic, and seizure-related abnormalities in autism spectrum disorders. Such information is an important precursor to rational,

hypothesis-driven treatment research that is based on potential pathophysiologic mechanisms. Thus, improved understanding of the genetic and neurobiologic bases of autism will likely lead to significant improvements in treatment.

A. Neurochemistry and metabolism

Early neurochemistry research in autism produced little in the way of replicable findings with the exception of serotonergic abnormalities. Research related to serotonin reuptake inhibitors, the serotonin transporter gene, serotonin receptors, and serotonin metabolism are areas of ongoing fruitful and promising study that should be supported. There is also a certain degree of interest in the role of the hypothalamic pituitary axis in terms of frequent sleep disorders, the possible role of oxytocin in affiliative behavior, and the effects of stress on brain function and perhaps structure. These issues may be secondary in terms of their likely pathophysiologic contribution to symptomatology in autism spectrum disorders. Preliminary reports of abnormalities in GABA receptor binding in the hippocampus in autism may lead to new research in this area as neurotransmitters are found to be more closely linked to particular cell populations and histologic abnormalities in autism.

The limited available data indicate that known inborn errors of metabolism are rare in autism. Currently available metabolic studies do not support routine screening for known inborn errors of metabolism in the absence of atypical features, dysmorphology or other evidence of specific metabolic defects. Nonetheless, two examples of treatable metabolic disorders associated with autistic symptomatology are untreated phenylketonuria (PKU) and increased 5’nucleotidase activity. For the latter, a small and incompletely described controlled trial with uridine and uridine analogues has reported improvement. It is notable that children with increased 5'nucleotidase activity have distinctive clinical features in addition to autism and have not been confirmed to meet criteria for a pervasive developmental disorder (PDD) based on research diagnostic criteria and methods. Histidinemia has also been reported in cases of autism, but the relationship to autism is unclear. Any metabolic treatment strategies proposed for autism need to focus on subgroups with the specific metabolic error targeted for remediation.

Consideration of a mechanism for identifying and investigating selected issues that may impact clinical practice guidelines for assessment and treatment is needed, while awaiting more definitive research. This would aid physicians and families in providing for current needs of children.

B. Immunology

Although there is a longstanding interest in a relationship between autism and autoimmune disorders, there is no evidence at present, positive or negative, that immunologic mechanisms cause or contribute to the central nervous system (CNS) abnormalities in autism. Before pursuing the development of immunomodulatory therapies, it would be important to first establish that there is evidence in the brain or cerebrospinal fluid (CSF) (but preferably in the brain) that immune factors are contributing to CNS abnormalities in autism.

There is no empirical evidence at present demonstrating the treatment efficacy of currently proposed immunologic interventions. For example, no double blind placebo controlled trials have been published demonstrating efficacy of intravenous immunoglobulin (IVIG). D8/17 antibodies are being investigated for an association with the repetitive ritualistic behavior of autism, but if confirmed, this association is likely to be diagnostically nonspecific, since it is also seen in some patients with obsessive-compulsive disorder and/or tic disorders and in some patients is a marker for rheumatic fever. This has no treatment implications at present, but does represent a line of research designed to investigate a potential immunologic mechanism.

C. Seizures, abnormal electroencephalograms (EEGs), regression, and anticonvulsant response

A number of major treatment issues exist in relationship to seizures. Seizures have been reported to occur in 20-30% of individuals with autism. Isolated observations have suggested that complex partial seizures may also be a major unrecognized treatment problem, but these have not been investigated. Abnormal EEGs are thought to be substantially more common than seizure disorders in autism. However, most EEG studies are from a much earlier period when children with other causes of brain damage were commonly included in the autistic populations studied. The prevalence of generalized motor and complex partial seizures in today's populations is unknown and represents a major treatment issue in autism. The prevalence and treatment implications of an abnormal EEG are likewise unknown. Anticonvulsants are being widely used clinically for their psychotropic effects to treat mood instability without the benefit of pharmacologic studies on efficacy. Another major contemporary treatment issue is the role of seizures in cases of autism presenting with regression and in cases of childhood disintegrative disorder, the two of which are not synonymous in terms of likely pathophysiology and underlying etiologies. Thus, the relationship of seizures and abnormal EEGs to regression, language deficits, and behavior problems is unknown.

Studies with an adequate number of subjects are needed to determine the prevalence of seizures and EEG abnormalities in the pervasive developmental

disorders across the age and severity span and to define the relationship of seizures and EEG abnormalities to regression and behavioral and cognitive abnormalities. Controlled trials are needed to assess the efficacy of antiepileptic drugs (AEDs) and steroids in the treatment of children with regression, epileptiform activity, behavior problems (such as aggression and irritability), and deficits (such as verbal auditory agnosia) that have a good likelihood of having a seizure basis. Improved understanding of EEG abnormalities and their mechanisms can lead to the rational and effective use of AEDs in autism.

There is no role for surgery in the treatment of autistic disorder, in the absence of rare and unrelated tumors or some cases of the classic form of the rare Landau-Kleffner Syndrome (acquired epileptic aphasia) in which seizures are refractory to all other known treatments.

D. Neural network modeling

This is another avenue for investigating the computational problems in the brain in autism. It emphasizes the importance of a neural systems perspective in the pathophysiology of autism. Although addressing only a few cognitive aspects or phenomena at present, these are important methods for characterizing the neural basis of cognition and behavior in autism. Additional methods are needed for assessing neural systems in autism and their relationship to cognition and behavior. Further knowledge is needed about the normal mechanisms of development of neural systems, their genetic control, and their abnormalities in autism. Such knowledge could provide the foundation for more definitive neurobiologic interventions in the future. At present, methods for studying neural networks are needed if changes in these systems are to be considered as the target of current treatments.

The importance of increasing knowledge of the pathophysiology of autism to guide further development of new treatments must be emphasized. Further neuropathological investigation is a critical and timely need. Other opportunities include study of the developmental neurobiologic mechanisms and the development of symptoms of autism in subgroups of children with tuberous sclerosis complex (TSC)-1, TSC2, fragile X syndrome (FRAX), Angelman syndrome and Rett syndrome.

E. Neurobiologic basis of treatment response

Early behavioral interventions may improve outcome in autism. The biologic basis of this improvement, where documented in randomized and replicated controlled trials, is not known, nor is it known what biologic factors might predict treatment response.

Integrative Issues Working Group

Leader: Pauline Filipek, M.D.
Rapporteur: Bryna Siegel, Ph.D.

A. Mission of the integrative issues group

The composition of this group was quite diverse, with a range of participants from clinically-oriented researchers representing methodologies oriented to asking questions about behavior changes to researchers interested in neuropathology representing medical models of basic research. Also represented were members of parent advocacy groups who contributed the perspective that any research design must be prepared to address concerns relevant to adults with autism and not focus exclusively on childhood or early childhood.

Given the very limited time for discussion in this group and some internal dispute about mission, discussion needed to be focused and so narrowed quickly to discussions within the framework of one methodological approach, the multi-center collaborative model. There clearly was concern in the group that such an approach precluded smaller scale innovative studies that could be quickly piloted or investigations that would warrant smaller scale exploratory studies before investing in larger studies more appropriate for confirmatory analyses.

B. Multisite versus individual investigations

Group discussion resulted in a compromised view that would allow for smaller scale investigations in the context of multisite collaborations. A major unresolved concern was that substantial funding remains seated in centers belonging to a multisite collaborative network and thereby may preclude individual investigators with high quality research who work on their own—either because of methodology, disciplinary perspective, or geographical or personal preference. There seemed to be a sense that the multisite approach, while potentially sound methodologically, would allow a small number of already advantaged major centers to hoard research funds, further disadvantaging investigators outside the network. Another concern raised about a potential multicenter 'juggernaut' was that the 'weight', complexity, and needed statistical power to address various questions might not always require the 'firepower' of a multisite investigation, although at other times, this would be clearly advantageous.

C. Ecological validity and the need for evaluation research

Another area neglected by the focus on the multicenter collaborative approach is the idea of more naturalistic, in situ evaluation research designs, such as used in educational and other social science research. Of particular concern here was the lack of attention to the scientific basis (or lack thereof) for autism treatment as it currently exists in the 'real world'—how autistic children are being treated by schools, behavior analysis methods, speech and language therapies, etc. This is to say that standards for the ecological validity of autism research were not encompassed in the discussions of this group. While the group realized that it was incumbent upon them to consider a wise spending of public dollars on research, the issue of how public funds for autism treatment are spent was not linked to the research agenda—and particularly to the question of what methodologies might be appropriate for analyzing and identifying the outcomes of extant treatment practices.

D. Consensus on valid methodologies

(1) Global research design requirements

General guidelines for treatment research were specified. It was agreed that intervention studies would need to include (a) pre-treatment measures that would allow a priori or post-hoc analyses of responder characteristics, (b) process measures to characterize the intervention being used, (c) outcome measures specific to hypothesized treatment effects, as well as (d) main effects of group (placebo / control) on outcome.

There was a general consensus that a set of standards would need to be developed for information that would be considered relevant for a core database or for protocol-specific data. This included (a) information regarding potential etiology, (b) measures for characterizing symptoms / symptom severity, (c) demographics, (d) procedures to assure standardized use of a treatment such as medical intervention, educational curriculum, or behavioral method, and (e) relevance to needed outcomes such as a change in social skills, adaptive behavior, or vocational functioning.

(2) A model for multicenter research

A primary area of focus and consensus was that a multicenter research approach that was similar to the National Cancer Institute's (NCI) Pediatric Oncology Group (POG), also known as the Children's Oncology Group (COG), could be implemented for studies in autism. The model would involve a core database on all subjects at all sites which would be funded through a core operations block

grant—overseen by a 'database site' which would be one of two 'principal institutes' which together would function as lead sites. Funding to other sites from the core operations principal site would be to collect and maintain a core database on subjects with agreed upon characteristics. The second principal institute, the lead 'data' site, would administer a protocol-specific block grant for individual research initiatives: As investigators within the network proposed new research protocols—which could be approved by a mechanism less formal than a NIH review, other sites could choose to participate or offer appropriate subjects for study. In the NCI POG/COG model, there are apparently three year funding cycles to the principle institutes, with these sites then distributing funding to other sites within each cycle.

The data would be centrally managed at the principal institute responsible for protocols, so that all sites could quickly modify (or terminate) a protocol as the incoming data stream indicated. Data in the POG/COG model are collected in three phases: Phase I: Feasibility (collection of small, even single/serial case samples), Phase II: Evidence of efficacy (multisite study of a treatment response that has been shown feasible), and Phase III: Implementation (de novo studies of the efficacy of a treatment shown in Phase II studies to be beneficial, e.g., with dissemination to sites that may not have been involved in establishing initial validity—to control for site-specific variability).

Some strengths of the POG/COG methodology that were noted included (a) population-based sampling, (b) the ability to add sites to a protocol if questions about site specific results arose, and (c) the ability to have non-treatment groups when placebo effects seemed to account for variance in outcome measures (e.g., as being reported in secretin trials)

(3) Contents of the core database

Much discussion focused on what should constitute a core data base. There was a general 'less is more' bias. Nevertheless, it was felt that a number of measures should be candidates for more careful evaluation for inclusion in a core database, including: (a) expert clinician diagnosis with Autism Diagnostic Interview-Revised (ADI-R) corroboration, (b) possibly an Autism Diagnostic Observation Schedule-Generic (ADOS-G) and/or possibly other measures of diagnostic traits, (c) possibly birth history, genetic and/or metabolic work-ups, (d) a measure of adaptive/ maladaptive behaviors (such as the Vineland or the Adaptive Behavior Checklist), (e) an IQ test (as appropriate to developmental level), (f) possibly a language measure(s), (g) possibly other measures with sufficient sensitivity to gauge treatment changes that could be valid in repeated measures designs (including post-test designs) such as (i) behavior ratings, (ii) language measures, (iii) executive function measures, and (iv) data on medication use, and

education. After some discussion, there seemed to be consensus that the last set of measures (i.e., g above) should probably be reserved for specific protocols.

(4) Developmental trajectories and the natural history of autism

There was a brief, interesting discussion on whether the core database should include some way of characterizing the natural history of the disorder through the use of selected measures and growth curve models to construct developmental trajectories and whether such developmental trajectories could be used as predictor and/or outcome measures. Those interested in adult issues noted that such models might be piloted with retrospective data on adults and might also have implications in the future for understanding geriatric issues in autism.

Agenda
Treatments for People with Autism and Other Pervasive Developmental Disorders: Research Perspectives

Monday, November 8, 1999

7:00 - 8:00 a.m.	Registration and Continental Breakfast
8:00 - 8:15 a.m.	Welcome and Announcements

Session I. Psychoeducational/Behavioral Treatments
Chair: Wendy Stone, Ph.D. (Vanderbilt)

8:15 - 8:35	Intensive Behavioral/Psychoeducational Treatments - Laura Schreibman, Ph.D. (UCSD)
8:35 - 8:45	Response - Mark Wolery, Ph.D. (UNC Chapel Hill)
8:45 - 8:55	Questions/Comments
8:55 - 9:15	Interventions to Facilitate Communication - Lynn Koegel, Ph.D. (UC Santa Barbara)
9:15 - 9:25	Response - Cathy Lord, Ph.D. (U. of Chicago)
9:25 - 9:35	Questions/Comments
9:35 - 9:50	Break
9:50 - 10:10	Interventions to Facilitate Socialization - Sally Rogers, Ph.D. (U. of Colorado)
10:10 - 10:20	Response - Patricia Krantz, Ph.D. (Princeton Child Development Institute)
10:20 - 10:30	Questions/Comments

10:30 - 10:50	Other Interventions to Facilitate Auditory, Visual, and Motor Integration - Geraldine Dawson, Ph.D. (U. of Washington)
10:50 - 11:00	Response - Howard Goldstein, Ph.D. (Florida State University)
11:00 - 11:10	Questions / Comments
11:10 - 11:30	Systems and Cost Benefit Analysis of Treatments - James Mulick, Ph.D. (Ohio State University)
11:30 - 11:40	Response - John Jacobson, Ph.D. (NY State Office of Mental Retardation)
11:40 - 11:50	Questions / Comments
11:50 - 1:00	Lunch
1:00 - 2:00	General Discussion

Session II. Pharmacological Treatments
Chair: Donald Cohen, M.D. (Yale)

2:00 - 2:20	Treatment of Compulsions and Stereotypies - Christopher McDougle, M.D. (Indiana University)
2:20 - 2:30	Response - C. T. Gordon (University of Maryland)
2:30 - 2:40	Questions / Comments
2:40 - 3:00	Treatment of Mood Disturbances, Agression, Self-Injury - Bryan King, M.D. (Dartmouth)
3:00 - 3:10	Response - Monique Ernst, M.D., Ph.D. (NIDA)
3:10 - 3:20	Questions / Comments
3:20 - 3:35	Break
3:40 - 4:00	Treatment of Attention Deficit Hyperactivity - Michael Aman, Ph.D. (Ohio State University)
4:00 - 4:10	Response - Xavier Castellanos, M.D. (NIMH)
4:10 - 4:20	Questions / Comments
4:20 - 5:05	General Discussion

Tuesday, November 9, 1999

7:00 - 8:00	Continental Breakfast

Session III. Emerging Biomedical/Clinical Neuroscience Approaches
Chair: Edwin H. Cook, M.D. (U. of Chicago)

8:00 - 8:20	Metabolic Approaches - Theodore Page, M.D. (UCSD)
8:20 - 8:30	Response - Michael Johnston, M.D. (Kennedy-Krieger)

8:30 - 8:40	Questions / Comments
8:40 - 9:00	Immunological Approaches - Sudhir Gupta, M.D., Ph.D. (UC-Irvine)
9:00 - 9:10	Response - Andrew Zimmerman, M.D. (Kennedy-Krieger)
9:10 - 9:20	Questions / Comments
9:20 - 9:40	Treatment of Seizure Disorders and EEG Abnormalities - Roberto Tuchman, M.D. (University of Miami)
9:40 - 9:50	Response - Andres Kanner, M.D. (St. Lukes-Rush Presbyterian, Chicago)
9:50 - 10:00	Questions / Comments
10:00 - 10:15	Break
10:20 - 10:40	Origins and Remediation of Autistic Deficits: Some Preliminary Suggestions Based on Neural Network Models - Jay McClelland, Ph.D. (Carnegie-Mellon)
10:40 - 10:50	Response - Barry Gordon, M.D., Ph.D. (Hopkins)
10:50 - 11:00	Questions / Comments
11:00 - 12:00	General Discussion
12:00 - 1:00	Lunch
1:00 - 3:00	**Working Groups Meet to Formulate Recommendations for Needed Research**

Psychoeducational / Behavioral - 1st floor Conference Room A
Chair: Wendy Stone, Ph.D. (Vanderbilt)
Rapporteur: Helen Tager-Flusberg (Eunice Kennedy Schriver Center)

Psychopharmacology - 1st floor Conference Room B
Chair: Donald Cohen, M.D. (Yale)
Rapporteur: Mark A. Riddle, M.D. (Hopkins)

Emerging Biomedical / Clinical Neuroscience Approaches - 6th floor, Conference Room 6109
Chair: Edwin H. Cook, M.D. (University of Chicago)
Rapporteur: Nancy J. Minshew, Ph.D. (University of Pittsburgh)

Integrative Issues - 8th floor, Conference Room 8120
Chair: Pauline A. Filipek, M.D. (UC-Irvine)
Rapporteur: Bryna Siegel, Ph.D. (UCSF)

3:00 - 3:15	Break

Reports Back to Full Session

3:20 - 3:40	Integrative Issues
3:40 - 4:00	Emerging Biomedical/Clinical Neuroscience Approaches
4:00 - 4:20	Psychopharmacology
4:20 - 4:40	Psychoeducational/Behavioral
4:40 - 5:15	Final Discussion and Closing Comments