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Proceedings of the 2008 annual meeting of the Fetal Alcohol Spectrum Disorders Study Group

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Abstract

The annual meeting of the Fetal Alcohol Spectrum Disorders Study Group (FASDSG) was held on June 28, 2008 in Washington DC, as a satellite to the Research Society on Alcoholism meeting. The FASDSG membership includes clinical, basic and social scientists, who meet to discuss recent advances and issues in FASD research. The main theme of the meeting was “Factors that Influence Brain and Behavioral Development: Implications for Prevention and Intervention.” Two keynote speakers, Dr. Stephen Suomi and Dr. Carl Keen addressed how early environment and nutrition may influence outcome following prenatal alcohol exposure. The final keynote speaker, Kathy Mitchell, addressed issues regarding the relationship between scientists and the families with children with FASD. Members of the FASDSG provided updates on new findings through brief (FASt) data reports, and national agency representative provided updates of activities and funding priorities. Presentations were also made by recipients of the Student Research Merit award and Rosett award.

Keywords

fetal alcohol syndrome; fetal alcohol spectrum disorders; teratology; ethanol; prenatal

On June 28, 2008, the annual meeting of the Fetal Alcohol Spectrum Disorders Study Group was held at the Grand Hyatt in Washington DC. The main theme of the meeting was “Factors that Influence Brain and Behavioral Development: Implications for Prevention and Intervention.”

The first keynote speaker was Dr. Carl Keen from University of California Davis, with a talk entitled, “Nutrition and the Brain: How Nutritional Factors May Increase Risk or Protect Against Fetal Alcohol Spectrum Disorders.” Dr. Keen discussed how nutritional status, both of the mother and the conceptus, may influence the expression of FASD, with the subthemes that selective micronutrient deficiencies may increase risk of FASD whereas micronutrient supplements may reduce the risk of FASD or modulate expression later in life. The idea that diet influences birth outcome is certainly not novel, but identifying which nutrients to focus on is complicated by the overlap of effects of various nutrient deficiencies as well as

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interactions among nutrients. Given this overlap, Dr. Keen emphasized that one needs to take a global perspective, rather than focusing on single nutrients.

Dr. Keen discussed the teratogenic effects of maternal zinc deficiency in greater detail. Zinc deficiency is related to increased pregnancy risks and animal model studies have shown that as few as 2–3 days of zinc deficiency can lead to massive cell death and teratogenic effects. These findings emphasize that good homeostatic controls to regulate fetal nutrition when maternal nutrient status is compromised do not exist and that the adverse effects of nutrient deficiencies can be very fast-acting. Nutritional deficiencies may not only be affected by dietary intake (primary deficiencies), but can also be created by secondary factors, such as genetic polymorphisms, interactions of nutrient uptake with drugs (like alcohol) and/or disease states, as well as nutrient interactions. It is well documented that alcoholics have difficulties with zinc metabolism and alcohol consumption during pregnancy is associated with reduced zinc levels both in maternal plasma and fetal core blood, which may contribute to alcohol's adverse effects on development. Indeed, animal studies indicate that a marginal zinc deficiency exacerbates ethanol's teratogenic effects.

But not only can marginal prenatal nutrient deficiencies cause short-term effects, they can also lead to long-term problems in the offspring via epigenetic factors, problems that are evident throughout the lifespan and across generations, even when the offspring are placed on a control diet postnatally. For example, marginal prenatal zinc deficiency compromises immunological responses and marginal prenatal iron deficiency leads to long-lasting changes in systolic blood pressure. Even the pre-conception nutritional environment can have long-lasting effects. There is limited data that suggest that individuals with FASD may have continuing problems with zinc and iron metabolism postnatally.

While micronutrient supplementation may seem like a solution, simply providing single nutrient supplements may not be sufficient for preventing prenatal nutritional deficiencies, since there are synergistic effects of multiple nutrients. Zinc, copper and iron interact with one another and supplements of one may lead to deficiencies in others, so levels have to be carefully considered for rehabilitative effects. Nevertheless, recent data from animal studies suggest that carefully timed postnatal iron supplements can block persistent epigenetic effects of iron deficiency on lifespan blood pressure, which provides hope that supplementation interventions may be effective, even during the early postnatal period. What about supplements for FASD? Some animal studies have shown that zinc supplementation can prevent prenatal ethanol's adverse effects on behavioral development. There is also evidence that supplementation of folic acid or choline may eliminate or reduce ethanol's teratogenic effects, so nutrient supplements should be considered as therapy programs both for pregnant women and children with FASD.

Several key questions need to be addressed. First, to what extent does suboptimal maternal nutritional status influence ethanol's teratogenic effects? Secondly, what are the mechanisms by which nutritional deficiencies exacerbate FASD? Finally, how persistent, specific and important are the effects? One challenge to answering these questions is assessment of nutritional state; clear biomarkers of nutrient status do not always exist, and dietary recall studies may underestimate the incidence of deficiency. Dr. Keen emphasized the need for large prospective studies that examine nutritional states at regular intervals to determine both acute and chronic effects. This then needs to be followed up with effective implementation plans of nutritional interventions.

The second keynote speaker was Dr. Stephen Suomi, Chief of the Laboratory of Comparative Ethology, National Institute of Child Health and Human Development at the National Institutes of Health. Dr. Suomi's talk was entitled, "Risk, Resilience, and Gene $\times$ Environment

Interactions in Primates”. Dr. Suomi has been working with a colony of rhesus monkeys, examining the genetic and environmental factors that influence individual differences in temperament, such as anxiety and aggression. Typically, infant monkeys use their mothers as a safety base from which to explore the environment and begin interacting with their peers from 1–3 years of age, engaging in play behavior that provides practice for behavioral patterns of adulthood involved in socialization, aggression, and reproductive behaviors

Research by Harry Harlow has shown that early rearing by peers leads to development of hyper-attachment bonds among the peers, bonds that are strong but not functional. Exploration and sophistication of play is reduced. Such differential rearing leads to long-lasting increases in fearfulness, failures to explore novel situations, and increased cortisol levels. Peer-reared individuals also start showing explosive aggression with reduced serotonin metabolism, as measured by reduced 5-HIAA levels. Early rearing also influences brain activity. PET scans show reduced serotonin binding potential and reduced cerebral blood flow in 2-year-old peer-reared monkeys compared to mother-reared individuals. Thus, early social environment has profound effects on brain structure and function.

Genetic factors are also important and interact with environmental factors. Polymorphisms of the serotonin transporter gene, which regulates reuptake of serotonin, influence serotonergic functioning. The short allele is characterized by less efficient transcription than the long allele, which may underlie differences in 5-HT metabolism. These alleles are found in both humans and rhesus monkeys. Although data relating the 5-HT transporter gene polymorphisms and behaviors among humans is mixed, there is some evidence individuals with the short allele are disproportionately likely to be incarcerated for aggression, hospitalized for severe depression, or victims of suicide. An intriguing study by Avshalom Caspi reported that humans with the short allele had increased levels of depression, but only if they also experienced high levels of concurrent stress or had a history of childhood neglect or abuse. Others have reported that young children with the short allele are behaviorally inhibited, a risk factor for affective disorders, but only if they also had an insecure attachment relationship with their mother. It is clear that the gene $\times$ environment interactions influence the developmental trajectory.

Dr. Suomi provided evidence of such gene $\times$ environment interactions within the monkey population. Peer-reared monkeys with the short 5-HT transporter allele exhibit reduced 5-HIAA levels, with concomitant increases in aggression and alcohol consumption, but that the risk associated with the short allele was buffered by good mothering. Peer-reared individuals, but not mother-reared individuals, with the short allele of the 5-HT transporter gene also show deficits in visual and auditory orienting, tasks that predict temperament. Finally, overactivity of the HPA axis and increased ACTH levels are associated with the combination of short allele and peer-rearing. Other candidate genes are also being investigated and it is clear that such risk genes are often only expressed if also accompanied with childhood neglect or abuse, a finding observed in both humans and rhesus monkeys. Interestingly, Mary Schneider has shown that prenatal alcohol exposure in combination with the short allele leads to irritability (as measured by the Brazelton), increased ACTH and cortisol levels.

In sum, good mothering as an early social experience is very important and could buffer against risk genes. Importantly, mothering patterns can be transferred across generations, independent of genes. Cross-fostering studies show that the offspring exhibit the mothering pattern of the foster rather than the biological mother. Thus, the genes that contribute to adverse outcome can remain in a population, as long as the early experience is good. But if the early environment and mothering is compromised by unstable conditions, disaster, alcohol drinking, reduced food supply, then the genetic risks are expressed.

The types of social interactions between mother and infant are critical. Dr. Suomi showed a video of face-to-face interactions of a rhesus monkey and her infant, gestures that were once thought to be uniquely human. Both human and monkey newborns exhibit face-to-face imitative behaviors, including lip smacking and tongue protrusions. It's believed that these behaviors are mediated by mirror neurons, which may play a role in theory of mind, empathy, and the development of language. Disruptions in mirror neurons may underlie autism spectrum disorders.

Individual differences in imitative behavior may be predictive of development of temperament. Not all infants imitate in the 1st week of life. When tested on the Brazelton, monkeys who do not imitate do not differ in body weight or gross motor ability/maturity, but their abilities to reach and grasp, which requires motor/sensory coordination and likely depends on the mirror neurons, is compromised. At 4–6 months, non-imitators exhibit reduced levels of social play and at 2 years, exhibit more self-directed behaviors and stereotypical patterns. Early experience influences imitative behaviors. Over 95% of mother-reared monkeys imitate, whereas only 60% of peer-reared monkeys imitate. Differences in EEG recordings in the newborns correspond to imitative behavior. Possible interventions are now being explored.

In the future, they will look at early mother/infant interactions and consider how these may be compromised during fetal alcohol exposure and during possible continued use of ethanol in the postnatal environment.

Dr. Michael Charness from Harvard University, Boston University School of Medicine and VA Boston Healthcare System, served as a discussant. One important focus of our study group is to identify how we can reduce the incidence of FASD and reduce the consequences among individuals with FASD. In other words, how can we prevent FASD and how can we intervene even after prenatal alcohol exposure?

In terms of prevention, nutrition has probably been understressed. Across the globe, FASDs are more prevalent where nutrition is poorest. Administration of micronutrients could 1) replete an inadequate state or 2) act as pharmacological agents. For example, thiamin (vitamin B1) is essential for energy metabolism of glucose and fatty acids. Alcohol adversely affects thiamin absorption, utilization and homeostasis. Thus alcoholics can have a higher thiamin requirement and may benefit from pharmacological-level intakes. However, when considering nutritional interventions, one must also be aware of potential undesirable consequences.

Secondly, opportunities to intervene are often postnatal so we are not only dealing with gene $\times$ environment interactions, but interactions of gene $\times$ environment $\times$ permanent developmental effects of ethanol exposure $\times$ continued exposure of mother and child to ethanol. For example, given that prenatal alcohol exposure permanently affects development of serotonergic systems, another layer is added to the interactions of genetic polymorphisms and the challenges of an early drinking environment that a child with FASD may experience. There may be opportunities to intervene among all of these dimensions. Social intervention is under-explored, in part because it is difficult and expensive, but given the potential to reduce secondary disabilities, such interventions should be investigated. There is a need to tap into the plasticity of the nervous system, by exploring interventions at pharmacological, nutritional, and social levels. In follow up questions to Drs. Keen and Suomi, it is clear that interventions, whether nutritional or social, require a change in society priorities.

As a regular part of the annual meeting, the FASDSG provides the opportunity for members to briefly present their newest data, in what is referred to as “*FASt Data Sessions*”. These presentations highlight members' most important recent findings, featured with a 5-min presentation of a single slide followed by 1 min of questions and answers. The *FASt* data presented in the meeting are described briefly below (presenters in bold-typed).

FASt Data Session on “Prevention & Risk”

Alexandra Regentova and T. Balachove (St. Petersburg State Univ Russia and Oklahoma Univ USA) reported data from a survey conducted in Russia examining how women's perceptions on prophylaxis of FAS were influenced by the presentation of materials and information. The brochures with information in FAQ format and easy-to-understand language were better received; photographs and visual presentation of both positive and negative reinforcement were effective. **Vivian Kulaga** and G. Koren (Toronto's Hospital for Sick Children) analyzed fatty acid ethyl esters (FAEE) in hair samples from an at-risk cohort of parents to detect excessive drinking. Results suggested that FAEE hair analysis may be a useful tool in detecting heavy alcohol use in the perinatal period. **Echo Rufer**, T. Tran, and S. Smith, (Univ. Wisconsin-Madison and East Carolina Univ.) indicated that maternal iron inadequacy causes neurobehavioral deficits that parallel those of FASD, and demonstrated that maternal iron inadequacy exacerbates the neurobehavioral changes induced by prenatal alcohol exposure. **Chris Downing**, A Kimball, H. Broncucia, J. Biers, P. Carosone-Link; T.E. Johnson and D. Gilliam (Univ. Colorado) performed QTL mapping to study ethanol teratogenesis in BXD recombinant inbred mice, and reported that chromosome 7 contains a cluster of imprinted genes which may partially mediate the differential sensitivity to ethanol teratogenesis observed in B6 and D2 mice.

FASt Data on “Treatment and Intervention”

Kristen Wellman, B. Lewis, A.H. Kehrberg, M. Carter, L. Carter, J.M. Littleton, and S. Barron (Univ Kentucky) demonstrated that a new compound, JR-220, that blocks the polyamine site of the glutamate NMDA receptor can reduce spatial memory deficits in juvenile rat pups following 3rd trimester-equivalent ethanol exposure. N.N.H. McGough, **Nirelia M. Idrus**, J.D. Thomas and E.P. Riley (San Diego State Univ) reported that administration of memantine, an NMDA receptor antagonist currently being used clinically for Alzheimer's disease, during ethanol withdrawal attenuates ethanol-induced Purkinje cell loss in the developing cerebellum. **Jennifer Wagner**, D. Garzon, A.Y. Klintsova, W.T. Greenough, and C. R. Goodlett (Indiana Univ/Purdue Univ at Indianapolis, Univ Delaware, and Univ Illinois) examined whether learning deficits induced by binge alcohol exposure in rats during the 3rd trimester equivalent can be ameliorated with behavioral interventions. Their study indicated that training on a complex motor task can improve acquisition of classical conditioning of the eyeblink response, illustrating that enhancement in cerebellar function with one type of training generalizes to other cerebellar-dependent tasks. **Mary J. O'Connor**, B. Paley, V. Keil, R. Bernier, and F. Frankel (UCLA) reported that social cognitive processing is an area to address in interventions with children who have a history of prenatal exposure to alcohol. Interventions targeting the manner in which children with prenatal exposure to alcohol process social information may help them to overcome their social skills deficits and find appropriate solutions to common social dilemmas.

FASt Data on “Cellular and Molecular Mechanisms & Consequences”

Sathyan Pratheesh and R. C. Miranda (Texas A&M Health Science Center) demonstrated that fibroblast growth factor, which controls neural stem cell renewal, also promotes the expression of miR335, an ethanol-sensitive microRNA. Their data suggest that miR335 is an integral part of a complex signaling web that controls some of the pleiotropic effects prenatal ethanol exposure. **Michael Puglia** and C.F. Valenzuela (Univ New Mexico) reported that alcohol exposure during the 3rd trimester equivalent increased the expression of the glutamate AMPA receptors, which may alter the course of hippocampal circuitry development. **Amber M. Eade**, J.C. Molina, N.E. Spear, P.F. Kent, L.M. Youngentob, and S.L. Youngentob (SUNY Upstate Med Univ, Syracuse) reported that, in the rat, a combined fetal and adolescent alcohol

exposure yielded a persistent behavioral olfactory response to ethanol odor during adulthood, suggesting that early alcohol leads to olfactory plasticity that contributes to the progressive ethanol acceptance later in life. **Tiffany Polanco**, D. Sarkar and W.S. Cohick (Rutgers State Univ of New Jersey) reported that prenatal alcohol exposure can lead to enhanced vulnerability to mammary tumorigenesis in rat offspring. Alcohol altered the mammary gland environment, likely by increasing circulating estradiol, and subsequently increased susceptibility to mammary tumorigenesis in adulthood.

FASt Data on “Behavioral Consequences”

Alana M. Mihic, C.R. Green, R. Hakvoort, D.P. Munoz and J.N. Reynolds (Queen’s Univ, Canada) compared eye movement behavior associated with Attention Deficit/Hyperactivity Disorder (ADHD) and FASD. Their data demonstrated that (1) deficits in eye movement control among children with FASD differ between those co-morbid for ADHD and those not co-morbid; and (2) in comparison to children with ADHD, there are both distinct and overlapping alterations in eye movement control. **Christie L. McGee**, O.A. Bjorkquist, J.M. Price, S. N. Mattson, and E.P. Riley (San Diego State Univ) indicated that children with prenatal alcohol exposure demonstrate significant impairments in social information processing, specifically in difficulty on the goal, response generation, and response evaluation steps in group entry situations, and difficulty with encoding, attribution, response evaluation, and enactment during provocation situations.

FASt Data On “Imaging Analyses of Brain Impairment”

Shonagh K. O’Leary-Moore, E.A. Myers, S. E. Parnell, Y. Jiang, D. B. Dehart, M. A. Styner, G.A Johnson and K.K Sulik (Univ North Carolina) used diffusion tensor imaging (DTI), a MRI study of fiber tract integrity, to examine white matter in the fetal mouse brain exposed to alcohol. They found olfactory bulb and frontal lobe deficiencies, including absence and/or abnormalities of the septal region and caudate. In brains exhibiting holoprosencephaly, the anterior limbs of the anterior commissure were absent and a thickened dysplastic fornix was found. Using MRI analysis, **Sara Stevens**, K. Nash, D. Kamino, E. D. Sheard, and J. Rovet (University of Toronto) reported that FASD children showing marked impairments in socioemotional functioning have significantly smaller orbitofrontal cortex (OFC) volumes, but no significant difference in organization of the white matter tissue was found in the OFC using DTI. In contrast, **Jeff Wozniak** (Univ of Minnesota) reported that DTI analysis of frontal white matter revealed less organized white matter that was associated with poor working memory and behavioral regulation in FASD children. White matter abnormalities were more significant among individuals with the full-blown fetal alcohol syndrome (FAS) compared to non-dysmorphic children with FASD. **Neal C. Dodge**, E. Garcia, J. L. Jacobson, and S.W. Jacobson, M.J. Avison (Wayne State University) reported that functional MRI (fMRI) analyses of young adults who were exposed prenatally to moderate levels of alcohol, indicated less activation of the angular gyrus during number processing than during letter matching. Together, their psychometric and fMRI analysis suggest that reading ability is relatively spared compared with arithmetic among individuals with FASD.

A brief business meeting was held. Dr. Susan Smith from the Department of Nutritional Sciences, University of Wisconsin was elected as the incoming FASDSG secretary and treasurer.

Agency updates were provided. First, Dr. Sally Anderson, NIAAA, the coordinator and executive secretary for the Interagency Coordinating Committee on FAS (ICCFAS), discussed current activities. The purpose of ICCFAS is to encourage and coordinate communication, cooperation and collaboration among federal agencies that deal with prenatal alcohol effects.

Missions include prevention of drinking during pregnancy, intervening with children and families affected by prenatal alcohol exposure, improvement of methods for diagnosis and case identification, increasing research on etiology and pathogenesis, and increasing information dissemination. Dr. Anderson discussed specific activities of the Dept. of Education, the Dept. of Justice, and some Dept. of Health and Human Services agencies that are members of the ICCFAS [Agency for Healthcare Research and Quality, Health Resources and Services Administration, Indian Health Service, and National Institute of Child Health and Human Development]. As examples of collaborations among agencies, work groups that comprise agency representatives, researchers, educators, parents, medical and legal professionals, have been created to address particular issues. Dr. Anderson discussed the activities of the Education Work Group, the Justice Work Group, and the Women, Drinking, and Pregnancy Work Group.

Dr. Louse Floyd provided updates from the Centers for Disease Control and Prevention (CDC). Dr. Floyd discussed the activities of the National Task Force on FAS, including published FAS diagnostic guidelines in 2004, a re-release of the Surgeon General's advisory on drinking during pregnancy, and reports on reducing alcohol-exposed pregnancies after reviewing the prevention literature, as well as a call to action on "Advancing Essential Services and Research on Fetal Alcohol Spectrum Disorders." The CDC has funded five intervention studies, four of which found positive results. Project CHOICES is an intervention for pre-conception women, focusing on women who are drinking, sexually active and not using contraception. Women who received the intervention were more likely to reduce risk drinking and/or increase contraception use. The CDC is currently marketing and disseminating materials for the field. Dr. Floyd mentioned the training efforts of CDC and global activities to prevent FAS. Finally, several funding opportunities and upcoming activities, including the partnering with ICCFAS to address ARND diagnostic criteria, were discussed.

Callie Gass from the SAMHSA FASD Center for Excellence spoke on the efforts of the Center to build systems of care in states and localities by transferring evidence-based practices through training and technical assistance, raising awareness and training professional staff to respond to various systems of care. The initial focus was on training and technical assistance with a lot of material development. More recent efforts involve integrating evidence-based prevention and treatment in states, localities and juvenile courts. Twenty-three new subcontractors have been added this year: 15 involving prevention and 8 on screening and diagnosis. The goal is replicate programs that are working and integrate these within existing systems of care, such as WIC and Healthy Start. A request for practices in the field that need to be evaluated was put out to the membership of the FASDSG.

Dr. Ken Warren, deputy director of NIAAA, provided an update of activities around FASD. NIAAA is very invested in FASD research, with the research program guided by the strategic plan. Challenges in FASD research include enhancement of the understanding of the etiology of FASDs, as understanding the mechanisms of alcohol-related damage may lead to identification of effective preventions and treatments for FASDs. A second challenge is to improve diagnosis with better recognition of facial dysmorphologies, including the use of 3-D imaging. A third challenge is to improve identification of nondysmorphic individuals with FASDs via maternal biomarkers of prenatal alcohol exposure as well as postnatal biomarkers in the FASD individual, including a better understanding of the neurodevelopmental phenotype. A fourth challenge is to identify the neurobehavioral phenotype associated with prenatal alcohol exposure to better inform development of interventions. And finally, there is a need to enhance prevention efforts. Dr. Warren then discussed the activities of two international collaborative projects: the Collaborative Initiative on FASD (CIFASD) and Prenatal Alcohol SIDS and Stillbirth Network (PASS). New opportunities for funding will include transformative research with the new T-RO1 mechanism, which would support the

introduction of new paradigms into FASD research. One suggested area of research is epigenetic modifications that may lead to multi-generational changes in the offspring.

This year's Student Merit Award was presented to Dr. Jayanth Ramadoss, who completed his Ph.D. with Dr. Tim Cudd at Texas A & M. Dr. Ramadoss' talk was entitled, "Acid Sensitive Channel Inhibition Prevents Fetal Alcohol Spectrum Disorders Cerebellar Purkinje Cell Loss." Noting a close association between blood alcohol level and blood pH, Dr. Ramadoss used a sheep model of alcohol exposure during the brain growth spurt on the TWIK-related K⁺ channels (TASK-1, TASK-2, TASK-3 and TREK-1), most of which respond to extracellular pH level. Both TASK-1 and TASK-3 isoforms are found in the cerebellum. Reducing pH during alcohol exposure attenuated the severity of alcohol-related cerebellar Purkinje cell loss; however, the protection was even greater using pharmacological blockers of the TASK channels. In fact, cerebellar Purkinje cell number was not significantly different among ethanol-exposed subjects treated with TASK blockers and controls. Dr. Ramadoss discussed the expression of TASK isoforms in various subpopulations within the cerebellum and hippocampus, two CNS regions known to be sensitive to prenatal alcohol exposure. This study shows promise of a mechanism-based intervention that emphasizes the role of the mother and maternal-fetal unit in the teratogenesis of ethanol. Interestingly, it is possible that other interventions, such as choline and NMDA receptor antagonists, may also block the TASK channels. These findings indicate another promising route of intervention.

The final keynote speaker was Kathy Mitchell, Vice President and National Spokesperson for the National Organization on Fetal Alcohol Syndrome (NOFAS), with a talk entitled, "From Research to Real Life: One Family's Story." Ms. Mitchell described her personal experiences within a family dealing with fetal alcohol spectrum disorders. She emphasized that FASD involve both the mother and the child. There is a need to promote the concept that alcoholism is a disease and that understanding the etiology of alcoholism is key to preventing FASD. Ms. Mitchell expressed the need to target individuals in addiction treatment centers to collect data on alcohol exposure and to identify potential children with FASD so that intervention can be initiated as early as possible.

The label of fetal alcohol syndrome and fetal alcohol spectrum disorders, which identifies the etiology, may be very useful for clinicians and researchers; however, the label does create a stigma, not only for the individuals with FASD, but also all of the members of the birth family. Such a stigma may lead to denial of the problem, reluctance to pursue diagnosis, reluctance of medical professionals to diagnose, and potential relapse of the mother. Moreover, there are many misconceptions of the behavioral phenotype of individuals with FASD and negative associations may create additional difficulties for the individual as well as their family. Thus, greater sensitivity to the label of FASD is needed.

Areas of research need include the determination of risk factors and to identification of effective preventions. Development of better neurodevelopmental screening tools, distinguishing FAS from other Axis I disorders, and determining which brain areas are more or less affected are also of importance, ultimately, as this may lead to the development of effective interventions. But in addition to addressing research questions, increased communication among researchers and the families of individuals with FASD is also needed. Better conduits of information exchange are desired by the families so they can be aware of research findings and the implications of those findings for the lives of those dealing directly with FASD. Conversely, families would like to be involved in the formation of priorities to help guide research directions. More efforts need to be extended so that research findings need to be translated to the families and health care systems. Our responsible as researchers extends beyond the laboratory to the families of individuals with FASDs.

The meeting concluded with the presentation of the Rosett Award, to this year's recipient, Dr. Laurie Foudin from NIAAA. Initially an NIH-funded alcohol investigator, Dr. Foudin left academics to join NIH where she managed the portfolio of grants that funded most of the research on alcohol and pregnancy conducted in the U.S. She played an active and influential role in shaping the direction of FASD research through identification of emerging research opportunities, organization of workshops and symposia, and the mentoring and support of investigators. Dr. Foudin reflected on the tremendous research progress on many fronts in the relatively short 40 years since fetal alcohol syndrome was first identified, but that there is a continuing need to integrate findings from research into health care systems in a cost-effective and sustainable manner. She cautioned that we do not need to wait until we fully understand every aspect of FAS to begin to improve the outcomes for children. The knowledge exists to develop useful clinical tools, including biomarkers of exposure and diagnostic markers, and treatments (pharmacological, nutritional, cognitive, behavioral, and tissue engineering) for affected individuals. We can begin to evaluate existing therapies for conditions with similar functional deficits as well as therapeutic approaches that enhance normal cognitive and motor function. She stressed the value in having a clinical research registry and a repository for human and animal tissue, which will require a partnership between researchers, individuals affected by FASD and their parents, and potential funding sponsors. As researchers, we should be proactive advocates for FASD research, educating government officials, health care professionals, educators, academic colleagues, and the public about FASD.

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