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Special Report

Highlights from the 37th Annual Meeting for ISPAD, Miami

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The 37th Annual Meeting for the International Society of Pediatric and Adolescent Diabetes was held in Miami Beach, Florida, USA. The meeting, titled 'Possibilities for Prevention and Diabetes and its Complications', attracted over 1000 delegates from 52 countries. Fifty-six oral abstracts were presented, along with 294 posters, representing the diversity of research and clinical innovations in the field of pediatric and adolescent diabetes around the world. Abstracts to the Oral and Poster Sessions can be found in a recent supplement of Pediatric Diabetes (1). Here are some highlights from the plenary sessions, symposia, and oral presentations.

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In the **Opening Session 'Reversal of type1 diabetes: vision or reality', Prof. Camillo Ricordi (USA)** described the exciting research journey of islet transplantation, which began at Washington University in 1995. He reminded us that autotransplantation of islets is a present reality and anti-rejection drugs are not required (2); some of the challenges of allogenic transplantation have been addressed through improvements in the Edmonton protocol, which involves percutaneous transhepatic infusion of islets using a glucocorticoid-free immunosuppression regimen (3). The primary objective remains to be able to achieve β-cell replacement without chronic immunosuppression. Possible avenues include advances in more selective and less toxic anti-rejection drugs, tissue engineering and protection of cells from recurrent autoimmunity by encapsulation using poly mer coating (4). Replacement or regeneration of β-cells may be achieved through xenotransplanta tion with porcine islets or use of stem cells, including embryonic (e.g., amniotic progenitor cells), or stem cells derived from cord blood, mesenchymal tissue, and bone marrow. An alternative approach is to reprogram or trans-differentiate existing cell types, such as pancreatic ductal cells or liver cells, into β-cells (4). Each of these approaches has unique physiological and ethical implications. A clinical islet transplantation consortium is now in its first phase III trial (www.citisletstudy.org).

Plenary Session I: future directions for type 1 diabetes management

Dr Richard Insel (USA) presented the Juvenile Diabetes Research Foundation (JDRF) International vision and strategy for type 1 diabetes prevention, highlighting that prevention may be an easier strategy than reversal. A two-pronged approach will focus on primary (preautoimmunity) and secondary (postautoimmunity) prevention. Primary prevention will be directed at development of universal childhood vaccines. Multiple vaccine routes are being explored including oral and nasal, while novel targets include enteroviruses and the human microbiome. Secondary prevention requires screening to identify high risk individuals. While diabetes autoantibodies

are useful, other biomarkers are needed to stage and predict disease progression. These include markers of β-cell stress to allow appropriate stratification for intervention trials.

Dr Gregory Clark (USA) gave an exciting lecture on the potential novel use of leptin as well as other adjuvant therapies for the treatment of type 1 diabetes. He presented a compelling argument for considering not just insulin deficiency in type 1 diabetes, but also high glucose variability, free fatty acids, paradoxical postprandial glucagon excess, and peripheral hyperinsulinemia. Leptin is a hormone derived from adipocytes and linked to satiety. Treatment with leptin in non-obese diabetic (NOD) mice suppresses hyperglucagonemia, normalizes hyperglycemia, reduces free fatty acids by oxidation, and reduces insulin-stimulated lipogenesis and cholesterologenesis (5, 6). The reduction in free fatty acids with exogenous insulin occurs through re-esterification of free fatty acids into stored lipid; however, in a state of insulin deficiency, the free fatty acids are released back into the circulation. It is unclear how leptin reduces hyperglycemia; further work is needed to investigate this. We await with interest the results of the Metreleptin trial that is already underway in adults with type 1 diabetes. Pramlintide (aymlin), GLP-1 analogues (liraglutide), and metformin are other promising adjuvant therapies that need further evaluation before being routinely used in type 1 diabetes. Pramlintide improves postprandial hyperglycemia but risks hypoglycemia, particularly when used in conjunction with rapid acting insulins. Liraglutide decreases glycemic variability, insulin dose, mean blood glucose levels, hemoglobin A1c (HbA1c), and weight (7). The pros and cons of leptin therapy of childhood diabetes have been recently debated in Pediatric Diabetes (8, 9).

Plenary Session II: preventing complications of diabetes – screening, risk factors, and interventions

Prof. Trevor Orchard (USA) emphasized that cardiovascular disease remains the biggest cause of mortality in adults with type 1 diabetes, based on data from large scale epidemiological studies (10, 11). Associations between glycemia and cardiovascular disease in type 1 diabetes remain inconsistent, suggesting multiple risk pathways are involved. Insulin resistance (measured by estimated glucose disposal rate) and haptoglobin genotype are predictors of cardiovascular disease in type 1 diabetes. For youth with diabetes, both childhood cardiovascular risk factors and abnormal surrogate measures of macrovascular disease track into adulthood. Whether these risk factors predict adult cardiovascular disease, or intervention reverses progression, remains unclear.

Dr Bruce Perkins (Canada) discussed established and novel biomarkers of microvascular complications, including the predictive value of the monofilament test in diabetic neuropathy (12). Measurement of intraepidermal nerve fiber diameter on skin biopsy and corneal confocal microscopy may provide novel morphological biomarkers for diabetic neuropathy (13).

Oral Session I: acute and chronic complications

Dr Paul Benitez-Aguirre (Australia) demonstrated that retinal vascular geometry measures, such as higher retinal length to diameter ratio and lower venular simple tortuosity, predict the onset of renal dysfunction in adolescents with type 1 diabetes. Dr M Nordwall (Sweden) reported long-term data on the prevalence of diabetic microangiopathy in young adults after 20–25 yr of type 1 diabetes (microalbuminuria 11%, proliferative retinopathy 13%) and confirmed the relationship with glycemic control. Dr H Margeirsdottir (Norway) provided evidence for increased inflammatory markers in young people with type 1 diabetes; demonstrating a positive association with HbA1c. Dr Yoon-Hi Cho (Australia) reported that lower sex hormone-binding globulin, an index of insulin resistance, along with higher HbA1c, may identify early cardiac autonomic dysfunction in adolescents with type 1 diabetes.

Oral Session II: education and psychosocial issues

Dr J Robert (France) found a high prevalence (55%) of psychiatric disorders in a pediatric diabetes unit: 33% had an anxiety disorder 13% an affective disorder and 12% a disruptive behavior disorder; the risk was positively associated with HbA1c. Prof. Carine de Beaufort (France), on behalf of the Hvidoere Study Group, reported significantly better quality of life (QOL) in young people receiving intensive therapy compared with conventional therapy, with the exception of psychological well-being. Dr Lange (Germany), for the Hvidoere Study Group, reported clinically significant emotional and behavior problems in 17% of young people with type 1 diabetes; the prevalence was lower in children using insulin pumps or multiple daily injection regimens and the risk was positively associated with HbA1c. Dr Serlachius (Australia) reported significant improvements in productive coping skills, diabetes-specific self-efficacy, stress levels, and QOL in young people randomized to a coping skills program compared to controls during 3 months follow-up.

Oral Session III: genetics and immunology

Dr Ingvild Sørensen (Norway) reported that a maternal serum level of 25-hydroxy vitamin D in the lowest

quartile during pregnancy was associated with twofold increased risk of type 1 diabetes in their offspring. Prof. Antonio Toniolo (Italy) reported a high prevalence (79%) of different species of enteroviruses in peripheral blood leukocytes of children at type 1 diabetes onset. Dr Lindehammer (Sweden) demonstrated an association between enterovirus IgM positivity in early pregnancy, HLA DQ 2/2 or 2/X genotype and risk of islet autoimmunity at delivery (odds ratio 3.1). Dr Ken Coppieters (USA) presented novel data from the network for Pancreatic Organ Donors on persistent MHC class I upregulation on pancreatic islets from 4/39 cases with longstanding type 1 diabetes. Interestingly, there was no evidence of chronic enteroviral infection.

Thursday, 20 October 2011

In his Lestradet Award for Education and Advocacy lecture, **Prof. Stuart Brink (USA)** described his professional journey as a pediatric endocrinologist through various landmark events and collaborations, which he entitled, 'an Educational Journey with empowerment'. Prime examples have been the Declaration of Kos, written one evening during the ISPAD congress in Greece when he was ISPAD's Secretary General, and the dramatic improvements in glycemic outcomes achieved through partnerships and focus on education and support of young people and their families in countries such as Romania (with Professors Serban and Dorchy) and the more recent multi-country collaborations throughout Africa. He inspired the audience to consider a paradigm shift in diabetes management, motivational interviewing, where patients and family members become directors for own care, and the role of the health professional around the world is to provide appropriate expertise and help refocus the family's efforts in management instead of only 'telling' people what to do.

Plenary Session III: advances in obesity

Dr Jack Yanovski (USA) focused on the leptin signaling cascade in animal and clinical models to demonstrate key concepts in obesity genetics. Individuals with leptin deficiency present with early onset obesity, hypoleptinemia and hyperphagia, which is reversed by leptin replacement. The vast majority of obese children, however have high plasma leptin levels secreted by adipose tissue, with little efficacy of leptin therapy. In non-leptin deficient children, unusually high leptin levels may therefore indicate a defect in leptin signaling or the feedback loop. These children with apparent leptin 'resistance' demonstrate greater body mass index (BMI) gain over time, independent of baseline BMI (14). Bardet Biedel syndrome is characterized by leptin resistance and obesity (15). Leptin signaling also occurs via melanocortin receptors MC4R and MC3R (16). Dr Yanovski's lecture gave a convincing presentation of how elucidating the genetic etiology and signaling pathways for energy intake may help direct specific targeted therapy for obesity.

Prof. Thomas Inge (USA) presented data demonstrating that surgery provides effective early weight loss for morbidly obese adolescents. Co-morbidities of obesity, such as type 2 diabetes, obstructive sleep apnea and metabolic syndrome features are dramatically improved postsurgery (17). Dr Inge proposed that early intervention may prevent the development of secondary complications of pediatric morbid obesity and potentially be more cost effective (18). Future studies comparing mechanisms, safety, and efficacy of the procedures will allow more tailored approach to treatment. Some of the answers to these questions will be provided by the Teen-Longitudinal Assessment of Bariatric Surgery, a multi-center NIH funded study, and related sub-studies and ancillary studies. More information about this study, as well as access to data and specimens through the ancillary studies mechanism, can be found at the study website (www.teen-labs.org).

Dr Elissa Jelalian (USA) emphasized the psychosocial aspects of obesity, highlighting that heavier children are more likely to have impaired QOL and lower self-identity (19), leading to greater risk of internalizing symptoms and disordered eating behavior. Family connectedness and family meals are believed to be protective factors, while meta-analysis demonstrates that lifestyle interventions are effective (20). Family-based behavioral interventions remain the first line of treatment for children and adolescents, while peer-based interventions also appear to have positive effects on weight reduction and psychological parameters.

Symposium I: VIDIS symposium

Prof. Heikki Hyöty (Finland), the chair of the Viruses and Diabetes International Study Group (VIDIS), which has recently become affiliated with ISPAD, presented interesting new data on the association between enterovirus infection and type 1diabetes, highlighting the role of Coxsackievirus B (CVB) group, particularly CVB1, in diabetes pathogenesis. Enterovirus infection during the 6-month window before onset of type 1 diabetes appears to a critical time and is associated with the strongest risk of diabetes (odds ratio ~8) (21). He also noted that a recent meta-analysis, which contains studies conducted by many members of the VIDIS group, demonstrated an association between enterovirus infection and both autoimmunity (odds ratio ~4) and type 1 diabetes (odds ratio ~10) (22). Development of an enterovirus

vaccine may provide hope for future prevention of type 1 diabetes.

Prof. Marian Rewers (USA) summarized the evidence from longitudinal studies of enterovirus infection in type 1 diabetes, including data from the Diabetes Autoimmunity Study in the Young study, which showed enterovirus infection was associated with risk of progression from islet autoimmunity to type 1 diabetes (23). He reminded us that the role of enteroviruses in diabetes pathogenesis has been examined over many decades, yet controversies remain.

Dr Barbara Schulte (Netherlands) provided new insights into the critical role of dendritic cells in the interplay between enteroviruses and the immune system in the development of type 1 diabetes. Her talk focused on how enterovirus infection of β-cells triggers innate immune responses in human dendritic cells (24). She emphasized the importance of production by dendritic cells of cytokines, such as interferon-α, in the antiviral response. The induction of an antiviral state may alter dendritic cell programming, thereby influencing development of regulatory and effector T cells.

Symposium IV: behavioural challenges in youth with diabetes

Dr Korey Hood (USA) emphasized the need to screen and intervene for depression in young people with type 1 diabetes. Depression is common, impacts on glycemic control and overall individual health, and places additional burden the health system. Treatment options, mostly supported by adult data, include antidepressants and cognitive behavioral therapy. Multi-component interventions which take into consideration emotional, social, and family processes have been shown to improve adherence in pediatric type 1 diabetes (25). Future studies will need to incorporate multi-component interventions for depressed teens with type 1 diabetes.

Dr Jean Lawrence (USA) presented results from the 'SEARCH' Diabetes in Youth Study relating to measures of health-related QOL and depressive symptoms. This multi-centre US study examined specific demographic and clinical factors associated with higher prevalence of mood disorders, and the risk differences in those with type 1 and 2 diabetes. Findings included a greater risk of depressive symptoms in youth with type 2 diabetes compared to those with type 1 diabetes, after adjusting for demographic differences (26). Future studies will include further work on longitudinal analysis of QOL measures and HbA1c outcomes.

Dr Jill Weissberg-Benchell (USA) emphasized the importance of screening for risk taking behaviors in adolescents with diabetes, whose brain and cognitive processes are still undergoing maturation. Disordered eating behavior can result in poor glycemic control and increased risk of long-term complications, hypoglycemia during driving behaviors impairs judgment, while drug and alcohol use can lead to diabetic ketoacidosis and death. Prevention strategies and the importance of provider–patient communication were also discussed.

Plenary Session VI: vitamin D in diabetes: much Ado about nothing . . . or something

Dr Jeremy Allgrove (UK) provided an overview of vitamin D deficiency (defined as 25-OHD less than 50 nmol/L). A number of factors influence vitamin D levels, including sunlight exposure, skin color, cultural differences, genetic factors, associated conditions such as coeliac disease, obesity, and diabetes. Around 80% of vitamin D is derived from sunlight, but this can only occur with exposure to the appropriate ultraviolet wavelength (~295 nm), the remainder comes from the diet. Any reduction in sunlight exposure must be compensated for by dietary supplementation. Individuals with darker skin pigmentation need more sunlight to produce the same amount of vitamin D than those with lighter skin pigmentation. In the non-diabetic population, there is also a link between obesity, vitamin D deficiency, and insulin resistance (27). An association between genetic variants of vitamin D genes and predisposition to type 1 diabetes has been described, providing some evidence for the link between vitamin D and type 1 diabetes (28–30). Although a number of published studies have linked low vitamin D levels in children with type 1 diabetes, it is unclear whether vitamin D deficiency causes or precipitates diabetes in susceptible individuals, or whether supplementation can improve glycemic control in those with established diabetes.

Dr Shayne Taback (Canada) presented an overview of vitamin D prevention and intervention trials in diabetes. Vitamin D has multiple metabolic effects and biological functions, including immunomodulatory and protective cardiovascular effects, which may be relevant to diabetes. Evidence linking vitamin D deficiency and type 1 diabetes comes from human immunological, epidemiological, genomic, and genetic data, as well as vitamin D deficient animal models (31, 32). A small pilot trial in Canada has been undertaken in healthy newborns with high risk HLA genotypes for type 1 diabetes, with newborns randomized to 2000 IU/d (n = 5) for 1 yr vs. 400 IU/d Vitamin D3 (n = 4). Among those randomized to 2000 IU/d, 25-OH vitamin D ≥ 60 ng/mL was achieved in 80%, with no adverse effects. Dr Taback is proposing a larger randomized control study following HLA screening of 60 000 newborns over 2 yr, intervening

with 2000 IU/d vs. placebo in addition to usual care (i.e., 2000–2600 IU/d vs. 0–600 IU/d).

Data linking vitamin D deficiency and type 2 diabetes are more limited. Potential mechanisms influence both insulin resistance and β-cell function (33). A newer area of interest is using vitamin D receptor activation to prevent diabetic nephropathy (34).

Dr Linda DiMeglio (USA) highlighted the importance of bone health in children and young adults with both type 1 and type 2 diabetes. Both diabetes types are associated with low bone formation, decreased bone strength, increased fracture risk, and reduced fracture healing (35). Lower bone mineral density (BMD) is seen in adults with type 1 diabetes, perhaps particularly in males (36). In type 2 diabetes, BMD may be low, normal, or high. Several mechanisms contribute to adverse bone health in pediatric diabetes, including standard risk factors (poor calcium intake and low levels of exercise) as well as diabetes-specific factors such as advanced glycation end-product interference with bone matrix collagen crosslinking, reduced osteocalcin, inflammatory cytokine effects, osmotic effects of hyperglycemia on bone formation, reduced bone blood flow due to diabetic microangiopathy, increased urinary calcium losses with glycosuria and gut and urinary calcium losses due to phosphoric acid-containing diet colas instead of milk ingestion. The delayed puberty sometimes seen in children with type 1 diabetes may also adversely affect bone accrual. The lower level of physical activity seen in children with type 1 diabetes may be explained in part by avoidance of hypoglycemia (37).

Optimizing bone health in childhood diabetes is likely to be important in long-term skeletal health. Ensuring adequate calcium intake, optimizing vitamin D, encouraging regular weight-bearing physical activity, actively screening for coeliac disease and assessing BMD in those with low trauma fractures is recommended. Unanswered questions remain including understanding the role of leptin, serotonin, and osteocalcin in human skeletal health, and to what degree improved glycemic control can affect bone mass and quality.

Friday, 21 October 2011

Plenary Session IV:

Dr Bruce Buckingham (USA) opened the ISPAD/JDRF Session on 'New Technologies in Diabetes Management' with a clear outline of the requirements and limitations faced in the vision to create a complete closed-loop system. Existing pump technologies and advances in glucose sensing, potential for multihormone systems, improved algorithms, and faster insulin absorption strategies will help to perfect the closed-loop system. These factors are currently being examined in JDRF multi-center collaborations.

Dr Roman Hovorka (UK) outlined the study results from Cambridge; now in its fourth year of clinical trials on the closed-loop system. Overnight closed-loop systems can reduce the risk of nocturnal hypoglycemia; addressing the time of day of most concern to families (38). Day and night randomized cross over study results have further highlighted the benefit of the overnight closed system in increasing time in target range, as well as the challenges of unannounced exercise and postprandial glucose control.

Dr Revital Nimri (Israel) presented the multi-centre clinical trials using the MD-Logic Artificial Pancreas; a closed-loop system based on an alternative algorithm for automated insulin delivery with a learning capacity of the patient's profile. Most recently, the closed-loop system was studied in an out-of-hospital setting at a diabetes camp.

Dr O. Rubio-Cabezas (Spain) was awarded the ISPAD Young Investigator Award for his work on neonatal diabetes while earning his PhD degree at the Peninsula Medical School, Exeter, UK. Genetic testing is now recommended in all infants presenting with diabetes within the first 6–9 months of life (39). Despite recent advances, around 40% cases of neonatal diabetes remain undiagnosed. Mutations in knockout mice have helped identify candidate genes causing similar phenotypes in man. Rare mutations in two transcription factor genes involved in pancreatic islet development causing neonatal diabetes and extra pancreatic features were described; NEUROD1 (permanent neonatal diabetes, cerebellar hypoplasia, and sensorineural abnormalities) and NEUROG3 (severe congenital malabsorptive diarrhea and neonatal or childhood-onset diabetes) (40, 41). The presence of extrapancreatic features should guide genetic testing in neonatal diabetes when common causes (mutations in the genes *KCNJ11*, *ABCC8*, and *INS*) have been excluded.

Symposium VII: management of type 2 diabetes in youth

Dr Elizabeth Sellers (Canada) highlighted that the natural history and true burden of youth onset type 2 diabetes are largely unknown. The emerging data is concerning; with higher rates of microvascular complications and earlier progression compared to type 1 diabetes (42, 43), as well as worse arterial stiffness on surrogate measures of macrovascular disease (44). Non-classic complications such as risk into the next generation through intra-uterine exposure are additional areas to consider.

Dr Sonia Caprio (USA) described the important background and metabolic profile of US youth with type 2 diabetes enrolled in the Treatment Options for type 2 Diabetes in Adolescents and Youth (TODAY) study, a multi-centre randomized controlled trial examining three therapeutic arms. Dr Caprio also challenged the audience to consider the underlying metabolic defects preceding type 2 diabetes; including insulin sensitivity, reduced β-cell sensitivity to glucose stimuli, and higher fatty liver content in the pharmacological targets for the treatment of type 2 diabetes.

Dr Denise Wilfey (USA) presented baseline psychosocial data from the TODAY study, highlighting the importance of evaluating for the presence of binge eating and depression in youth with type 2 diabetes (45). The TODAY Lifestyle Program provides a model for an intensive family-based weight-management program for type 2 diabetes, taking into account family functioning around diabetes care and psychosocial intervention to initiate and maintain lifestyle change over a longitudinal period (46).

Oral Session IV: epidemiology

Dr Geir Joner (Norway) reported that type 1 diabetes remained the predominant form of diabetes in young people during the last decade, with nine cases of monogenic diabetes (0.7%) and nine of type 2 diabetes, while Dr Jetske Kraan (Australia) reported evidence for a plateau in type1 diabetes incidence in recent years. Dr M Nakhala (Canada) also demonstrated a plateau in diabetes incidence from 2000. Prof. Stephen Greene (Scotland) presented data on the economic burden of type1 diabetes; with an incremental rise in direct medical costs from childhood through adolescence to adulthood. The largest component cost was for hospitalization. Dr Terri Lipman (Philadelphia) reported significant racial disparity in insulin pump use among youth with type1 diabetes, after controlling for socioeconomic status. Dr Wood (USA), on behalf of the T1D exchange network, reported data on more than 4000 participants, with only 28% achieving the ISPAD guidelines target HbA1c $< 7.5\%$.

Oral Session V: pumps and sensors

Dr Olga Kordonouri (Germany) reported 24-month data on a randomized trial of sensor-augmented pump therapy in children, with no difference in HbA1c (7.6%) vs. those on conventional CSII (7.7%). There was some evidence for a smaller decline in c-peptide in the sensor-augmented pump users; these interesting findings should be explored in other populations. Dr Nelly Mauras (USA), on behalf of DirecNet, reported data from a randomized controlled trial of real time continuous glucose monitoring (CGM) in 4–9 yr olds (64% on pumps). While there was a high degree of parental satisfaction with CGM and low rates of hypoglycemia overall, CGM was not associated with a significant benefit on glycemic control. Dr Mary White (Australia) explored potential reasons for failure to achieve target glycemic control in young people on pumps, and found that higher HbA1c before initiation of pump therapy predicted adherence to therapy.

Oral Session VI: obesity/puberty/new agents/MODY

Dr Rachel Besser (UK) presented data on use of a single postmeal urinary C-peptide to creatinine ratio (UCPCR) as a non-invasive measure of endogenous insulin secretion, demonstrating that it can discriminate monogenic from type 1 diabetes in children. Testing for monogenic diabetes should only be performed if the UCPCR is >0.7 nmol/mmol. Dr Clements, on behalf of the T1D Exchange Clinic Network, provided interesting data demonstrating significantly higher levels of Perfluorooctane sulfonate, an endocrine disrupter, in children at onset of type 1 diabetes compared with controls, suggesting this may be a novel biomarker.

Saturday, 22 October 2011

Plenary Session V: type 1 diabetes prevention and modulation of disease progression

Prof. Jay Skyler (USA) led the last session of the meeting with a historical overview of the vision to prevent and modulate progression to type 1 diabetes. There are ongoing antigen based and immunomodulatory new onset studies. Where single agents (such as cyclosporin, Anti-CD3, Anti-CD20, CTLA4-Ig) have only provided a transient effect in maintaining insulin-free status, combination therapy may provide the avenue for achieving sustained effect in future studies. Prof. Skyler emphasized the importance of massive screening efforts and collaborative approach to conduct adequately powered well-designed studies. TrialNet, INIT II, and TRIGR are examples of current multi-center studies that have provided important cohorts for prevention and intervention trials, while population screening at birth may be a future direction (4).

Dr Chantal Mathieu (Belgium) gave an outline of the European collaboration in NAIMIT, which brings together a consortium of researchers and small to medium enterprises in the common goal of finding novel interventional therapies in type 1 diabetes (www.naimit.eu). The underlying concept in NAIMIT

studies is the central role of the immune system in type 1 diabetes, with the β-cell as an active partner in the autoimmune damage and pro-inflammatory dialog. Current studies examining the communication between the β-cell and vitamin D as a natural immune modulator have shown promising results in NOD mice.

Prof. Desmond Schatz (USA) concluded the session with a prevailing sense of optimism resulting from the advances in diabetes research and clinical care. Dr Schatz also reflected on the lessons learnt from the research journey in intervention and prevention studies, as well as the future challenges in addressing long-term benefits, minimizing risk, and re-examining the current understanding of the mechanisms leading to type 1 diabetes.

Oral Session VII: complications; psychosocial issues

Dr Elizabeth Sellers (Canada) presented data on the feasibility of corneal confocal microscopy, a non-invasive technique, to quantify corneal nerve morphology in youth with type2 diabetes. Dr Butwicka (Poland) reported that HbA1c > 8.7% demonstrated good sensitivity and specificity for detecting children who met DSM-IV criteria for mood disorders, and thus may be used as a screening tool.

The 37th Annual Meeting of ISPAD was launched with Opening Addresses from **Prof. Alan Delamater (USA)** as the Conference President and **Dr Lynda Fisher (USA)** as ISPAD President. There were ample opportunities to network with colleagues from countries around the world, among the enthusiastic 'vibe' of people committed to the field of pediatric and adolescent diabetes. The ISPAD flag will now move onto Istanbul, Turkey, where the 38th Annual Meeting will be held in 2012.

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